

# Hardener FX Medium & Fast (2K)

## DNA Custom Paints

Chemwatch: 4780-45  
Version No: 5.1.1.1  
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

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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	Hardener FX Medium & Fast (2K)
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Other means of identification	Part No.: HFXM- & HXF-

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

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

Registered company name	DNA Custom Paints
Address	5-7 Keith Campbell Court Scoresby 3179 VIC Australia
Telephone	+61 3 9764 2088
Fax	+61 3 9764 1244
Website	www.dna-paints.com
Email	Not Available

### Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	+61 3 9573 3112
Other emergency telephone numbers	Not Available

## 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

	Min	Max	
Flammability	2	2	0 = Minimum 1 = Low 2 = Moderate 3 = High 4 = Extreme
Toxicity	2	2	
Body Contact	2	2	
Reactivity	1	1	
Chronic	2	2	

Poisons Schedule	S6
GHS Classification [1]	Flammable Liquid Category 3, Acute Toxicity (Inhalation) Category 4, Respiratory Sensitizer Category 1, Skin Sensitizer Category 1, STOT - SE (Narcosis) Category 3, Aspiration Hazard Category 1
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	<b>DANGER</b>
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### Hazard statement(s)

H226	Flammable liquid and vapour
H332	Harmful if inhaled

Continued...

H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled
H317	May cause an allergic skin reaction
H336	May cause drowsiness or dizziness
H304	May be fatal if swallowed and enters airways
AUH066	Repeated exposure may cause skin dryness and cracking

**Precautionary statement(s) Prevention**

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P261	Avoid breathing 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.
P285	In case of inadequate ventilation wear respiratory protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P272	Contaminated work clothing should not be allowed out of the workplace.

**Precautionary statement(s) Response**

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P331	Do NOT induce vomiting.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER or doctor/physician.
P363	Wash contaminated clothing before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.

**Precautionary statement(s) Storage**

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

**Precautionary statement(s) Disposal**

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
28182-81-2	30-60	<u>hexamethylene diisocyanate polymer</u>
108-65-6	10-30	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u>
123-86-4	10-30	<u>n-butyl acetate</u>

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

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Wash out immediately with fresh running water.</li> <li>▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.</li> <li>▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> </ul>

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	<ul style="list-style-type: none"> <li>▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>▶ Transport to hospital, or doctor.</li> </ul> <p>Following uptake by inhalation, move person to an area free from risk of further exposure. Oxygen or artificial respiration should be administered as needed. Asthmatic-type symptoms may develop and may be immediate or delayed up to several hours. Treatment is essentially symptomatic. A physician should be consulted.</p>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> <li>▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.</li> </ul>

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

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

Treat symptomatically.

for simple esters:

#### BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ Monitor and treat, where necessary, for pulmonary oedema .
- ▶ Monitor and treat, where necessary, for shock.
- ▶ **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- ▶ Give activated charcoal.

#### ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

#### EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Consult a toxicologist as necessary.

*BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994*

For sub-chronic and chronic exposures to isocyanates:

- ▶ This material may be a potent pulmonary sensitiser which causes bronchospasm even in patients without prior airway hyperreactivity.
- ▶ Clinical symptoms of exposure involve mucosal irritation of respiratory and gastrointestinal tracts.
- ▶ Conjunctival irritation, skin inflammation (erythema, pain vesiculation) and gastrointestinal disturbances occur soon after exposure.
- ▶ Pulmonary symptoms include cough, burning, substernal pain and dyspnoea.
- ▶ Some cross-sensitivity occurs between different isocyanates.
- ▶ Noncardiogenic pulmonary oedema and bronchospasm are the most serious consequences of exposure. Markedly symptomatic patients should receive oxygen, ventilatory support and an intravenous line.
- ▶ Treatment for asthma includes inhaled sympathomimetics (epinephrine [adrenalin], terbutaline) and steroids.
- ▶ Activated charcoal (1 g/kg) and a cathartic (sorbitol, magnesium citrate) may be useful for ingestion.
- ▶ Mydriatics, systemic analgesics and topical antibiotics (Sulamyd) may be used for corneal abrasions.
- ▶ There is no effective therapy for sensitised workers.

[Ellenhorn and Barceloux; Medical Toxicology]

**NOTE:** Isocyanates cause airway restriction in naive individuals with the degree of response dependant on the concentration and duration of exposure. They induce smooth muscle contraction which leads to bronchoconstrictive episodes. Acute changes in lung function, such as decreased FEV1, may not represent sensitivity.

[Karol & Jin, Frontiers in Molecular Toxicology, pp 56-61, 1992]

Personnel who work with isocyanates, isocyanate prepolymers or polyisocyanates should have a pre-placement medical examination and periodic examinations thereafter, including a pulmonary function test. Anyone with a medical history of chronic respiratory disease, asthmatic or bronchial attacks, indications of allergic responses, recurrent eczema or sensitisation conditions of the skin should not handle or work with isocyanates. Anyone who develops chronic respiratory distress when working with isocyanates should be removed from exposure and examined by a physician. Further exposure must be avoided if a sensitivity to isocyanates or polyisocyanates has developed.

## SECTION 5 FIREFIGHTING MEASURES

### Extinguishing media

- ▶ Small quantities of water in contact with hot liquid may react violently with generation of a large volume of rapidly expanding hot sticky semi-solid foam.
- ▶ Presents additional hazard when fire fighting in a confined space.
- ▶ Cooling with flooding quantities of water reduces this risk.
- ▶ Water spray or fog may cause frothing and should be used in large quantities.
- ▶ Alcohol stable foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.
- ▶ Water spray or fog - Large fires only.

**Special hazards arising from the substrate or mixture**

<b>Fire Incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result</li> </ul>
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**Advice for firefighters**

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Liquid and vapour are flammable.</li> <li>▶ Moderate fire hazard when exposed to heat or flame.</li> <li>▶ Vapour forms an explosive mixture with air.</li> <li>▶ Moderate explosion hazard when exposed to heat or flame.</li> <li>▶ Vapour may travel a considerable distance to source of ignition.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> </ul> <p>Combustion products include; carbon dioxide (CO<sub>2</sub>) carbon monoxide (CO) isocyanates and minor amounts of hydrogen cyanide nitrogen oxides (NO<sub>x</sub>) other pyrolysis products typical of burning organic material. When heated at high temperatures many isocyanates decompose rapidly generating a vapour which pressurises containers, possibly to the point of rupture. Release of toxic and/or flammable isocyanate vapours may then occur</p> <ul style="list-style-type: none"> <li>▶ Burns with acrid black smoke.</li> </ul>

**SECTION 6 ACCIDENTAL RELEASE MEASURES****Personal precautions, protective equipment and emergency procedures**

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▶ Remove all ignition sources.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb small quantities with vermiculite or other absorbent material.</li> <li>▶ Wipe up.</li> <li>▶ Collect residues in a flammable waste container.</li> </ul>																		
<b>Major Spills</b>	<ul style="list-style-type: none"> <li>▶ Liquid Isocyanates and high isocyanate vapour concentrations will penetrate seals on self contained breathing apparatus - SCBA should be used inside encapsulating suit where this exposure may occur.</li> </ul> <p>For isocyanate spills of less than 40 litres (2 m<sup>2</sup>):</p> <ul style="list-style-type: none"> <li>▶ Evacuate area from everybody not dealing with the emergency, keep them upwind and prevent further access, remove ignition sources and, if inside building, ventilate area as well as possible.</li> <li>▶ Notify supervision and others as necessary.</li> <li>▶ Put on personal protective equipment (suitable respiratory protection, face and eye protection, protective suit, gloves and impermeable boots).</li> <li>▶ Control source of leakage (where applicable).</li> <li>▶ Dike the spill to prevent spreading and to contain additions of decontaminating solution.</li> <li>▶ Prevent the material from entering drains.</li> <li>▶ Estimate spill pool volume or area.</li> <li>▶ Absorb and decontaminate. - Completely cover the spill with wet sand, wet earth, vermiculite or other similar absorbent. - Add neutraliser (for suitable formulations: see below) to the adsorbent materials (equal to that of estimated spill pool volume). Intensify contact between spill, absorbent and neutraliser by carefully mixing with a rake and allow to react for 15 minutes</li> <li>▶ Shovel absorbent/decontaminant solution mixture into a steel drum.</li> <li>▶ Decontaminate surface. - Pour an equal amount of neutraliser solution over contaminated surface. - Scrub area with a stiff bristle brush, using moderate pressure. - Completely cover decontaminant with vermiculite or other similar absorbent. - After 5 minutes, shovel absorbent/decontamination solution mixture into the same steel drum used above.</li> <li>▶ Monitor for residual isocyanate. If surface is decontaminated, proceed to next step. If contamination persists, repeat decontaminate procedure immediately above</li> <li>▶ Place loosely covered drum (release of carbon dioxide) outside for at least 72 hours. Label waste-containing drum appropriately. Remove waste materials for incineration.</li> <li>▶ Decontaminate and remove personal protective equipment.</li> <li>▶ Return to normal operation.</li> <li>▶ Conduct accident investigation and consider measures to prevent reoccurrence.</li> </ul> <p><b>Decontamination:</b> Treat isocyanate spills with sufficient amounts of isocyanate decontaminant preparation ("neutralising fluid"). Isocyanates and polyisocyanates are generally not miscible with water. Liquid surfactants are necessary to allow better dispersion of isocyanate and neutralising fluids/ preparations. Alkaline neutralisers react faster than water/surfactant mixtures alone. Typically, such a preparation may consist of: Sawdust: 20 parts by weight Kieselguhr 40 parts by weight plus a mixture of {ammonia (s.g. 0.880) 8% v/v non-ionic surfactant 2% v/v water 90% v/v}. Let stand for 24 hours Three commonly used neutralising fluids each exhibit advantages in different situations.</p> <p><b>Formulation A :</b></p> <table> <tr> <td>liquid surfactant</td> <td>0.2-2%</td> </tr> <tr> <td>sodium carbonate</td> <td>5-10%</td> </tr> <tr> <td>water to</td> <td>100%</td> </tr> </table> <p><b>Formulation B</b></p> <table> <tr> <td>liquid surfactant</td> <td>0.2-2%</td> </tr> <tr> <td>concentrated ammonia</td> <td>3-8%</td> </tr> <tr> <td>water to</td> <td>100%</td> </tr> </table> <p><b>Formulation C</b></p> <table> <tr> <td>ethanol, isopropanol or butanol</td> <td>50%</td> </tr> <tr> <td>concentrated ammonia</td> <td>5%</td> </tr> <tr> <td>water to</td> <td>100%</td> </tr> </table>	liquid surfactant	0.2-2%	sodium carbonate	5-10%	water to	100%	liquid surfactant	0.2-2%	concentrated ammonia	3-8%	water to	100%	ethanol, isopropanol or butanol	50%	concentrated ammonia	5%	water to	100%
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After application of any of these formulae, let stand for 24 hours.

Formulation B reacts faster than Formulation A. However, ammonia-based neutralisers should be used only under well-ventilated conditions to avoid overexposure to ammonia or if members of the emergency team wear suitable respiratory protection. Formulation C is especially suitable for cleaning of equipment from unreacted isocyanate and neutralizing under freezing conditions. Regard has to be taken to the flammability of the alcoholic solution.

- ▶ Avoid contamination with water, alkalis and detergent solutions.
- ▶ Material reacts with water and generates gas, pressurises containers with even drum rupture resulting.
- ▶ **DO NOT reseal container if contamination is suspected.**
- ▶ Open all containers with care.
- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ May be violently or explosively reactive.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Consider evacuation (or protect in place).
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse /absorb vapour.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Use only spark-free shovels and explosion proof equipment.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Absorb remaining product with sand, earth or vermiculite.
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

## SECTION 7 HANDLING AND STORAGE

### Precautions for safe handling

#### Safe handling

- ▶ Containers, even those that have been emptied, may contain explosive vapours.
  - ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.
  - ▶ **DO NOT allow clothing wet with material to stay in contact with skin**
- The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe
- ▶ **DO NOT concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential.**
  - ▶ Any static discharge is also a source of hazard.
  - ▶ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina.
  - ▶ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage.
  - ▶ Add inhibitor to any distillate as required.
  - ▶ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely.
- 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.
- Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.
- ▶ 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 overexposure occurs.
  - ▶ Use in a well-ventilated area.
  - ▶ Prevent concentration in hollows and sumps.
  - ▶ **DO NOT enter confined spaces until atmosphere has been checked.**
  - ▶ Avoid smoking, naked lights or ignition sources.
  - ▶ Avoid generation of static electricity.
  - ▶ **DO NOT use plastic buckets.**
  - ▶ Earth all lines and equipment.
  - ▶ Use spark-free tools when handling.
  - ▶ Avoid contact with incompatible materials.
  - ▶ **When handling, DO NOT eat, drink or smoke.**
  - ▶ Keep containers securely sealed when not in use.
  - ▶ Avoid physical damage to containers.
  - ▶ Always wash hands with soap and water after handling.
  - ▶ Work clothes should be laundered separately.
  - ▶ Use good occupational work practice.
  - ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
  - ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.

#### Other information

- ▶ Store in original containers in approved flammable liquid storage area.
- ▶ Store away from incompatible materials in a cool, dry, well-ventilated area.
- ▶ **DO NOT store in pits, depressions, basements or areas where vapours may be trapped.**
- ▶ No smoking, naked lights, heat or ignition sources.
- ▶ Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access.
- ▶ Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.
- ▶ Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
- ▶ Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors.
- ▶ Keep adsorbents for leaks and spills readily available.
- ▶ Protect containers against physical damage and check regularly for leaks.

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- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- In addition, for tank storages (where appropriate):
- ▶ Store in grounded, properly designed and approved vessels and away from incompatible materials.
  - ▶ For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.
  - ▶ Storage tanks should be above ground and diked to hold entire contents.
- for commercial quantities of isocyanates:
- ▶ Isocyanates should be stored in adequately bunded areas. Nothing else should be kept within the same bunding. Pre-polymers need not be segregated. Drums of isocyanates should be stored under cover, out of direct sunlight, protected from rain, protected from physical damage and well away from moisture, acids and alkalis.
  - ▶ Where isocyanates are stored at elevated temperatures to prevent solidifying, adequate controls should be installed to prevent the high temperatures and precautions against fire should be taken.
  - ▶ Where stored in tanks, the more reactive isocyanates should be blanketed with a non-reactive gas such as nitrogen and equipped with absorptive type breather valve (to prevent vapour emissions)..
  - ▶ Transfer systems for isocyanates in bulk storage should be fully enclosed and use pump or vacuum systems. Warning signs, in appropriate languages, should be posted where necessary.
  - ▶ Areas in which polyurethane foam products are stored should be supplied with good general ventilation. Residual amounts of unreacted isocyanate may be present in the finished foam, resulting in hazardous atmospheric concentrations.

## Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Packing as supplied by manufacturer.</li> <li>▶ Plastic containers may only be used if approved for flammable liquid.</li> <li>▶ Check that containers are clearly labelled and free from leaks.</li> <li>▶ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure.</li> <li>▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C)</li> <li>▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)</li> <li>▶ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used.</li> <li>▶ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages</li> <li>▶ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Esters react with acids to liberate heat along with alcohols and acids.</li> <li>▶ Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products.</li> <li>▶ Heat is also generated by the interaction of esters with caustic solutions.</li> <li>▶ Flammable hydrogen is generated by mixing esters with alkali metals and hydrides.</li> <li>▶ Esters may be incompatible with aliphatic amines and nitrates.</li> <li>▶ Avoid reaction with water, alcohols and detergent solutions.</li> <li>▶ Isocyanates and thioisocyanates are incompatible with many classes of compounds, reacting exothermically to release toxic gases. Reactions with amines, strong bases, aldehydes, alcohols, alkali metals, ketones, mercaptans, strong oxidisers, hydrides, phenols, and peroxides can cause vigorous releases of heat. Acids and bases initiate polymerisation reactions in these materials.</li> <li>▶ Isocyanates easily form adducts with carbodiimides, isothiocyanates, ketenes, or with substrates containing activated CC or CN bonds.</li> <li>▶ Some isocyanates react with water to form amines and liberate carbon dioxide. This reaction may also generate large volumes of foam and heat. Foaming in confined spaces may produce pressure in confined spaces or containers. Gas generation may pressurise drums to the point of rupture.</li> <li>▶ Do NOT reseal container if contamination is expected</li> <li>▶ Open all containers with care</li> <li>▶ Base-catalysed reactions of isocyanates with alcohols should be carried out in inert solvents. Such reactions in the absence of solvents often occur with explosive violence,</li> <li>▶ Isocyanates will attack and embrittle some plastics and rubbers.</li> <li>▶ Avoid strong acids, bases.</li> <li>▶ A range of exothermic decomposition energies for isocyanates is given as 20-30 kJ/mol.</li> <li>▶ The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment.</li> <li>▶ For example, in "open vessel processes" (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below 500 J/g are unlikely to present a danger, whilst those in "closed vessel processes" (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g.</li> </ul> <p>BREThERICK: Handbook of Reactive Chemical Hazards, 4th Edition</p>



+ X X X + X +

- X** — Must not be stored together  
**O** — May be stored together with specific preventions  
**+** — May be stored together

## SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

## Control parameters

## OCCUPATIONAL EXPOSURE LIMITS (OEL)

## INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	hexamethylene diisocyanate polymer	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Sen
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	274 mg/m3 / 50 ppm	548 mg/m3 / 100 ppm	Not Available	Sk
Australia Exposure Standards	n-butyl acetate	n-Butyl acetate	713 mg/m3 / 150 ppm	950 mg/m3 / 200 ppm	Not Available	Not Available

**EMERGENCY LIMITS**

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
hexamethylene diisocyanate polymer	Hexamethylene diisocyanate polymer	7.8 mg/m <sup>3</sup>	86 mg/m <sup>3</sup>	510 mg/m <sup>3</sup>
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
n-butyl acetate	Butyl acetate, n-	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
hexamethylene diisocyanate polymer	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
n-butyl acetate	10,000 ppm	1,700 [LEL] ppm

**MATERIAL DATA**

gt;

**Exposure controls**

<b>Appropriate engineering controls</b>	<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>For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.</p> <p>Spraying of material or material in admixture with other components must be carried out in conditions conforming to local state regulations. Local exhaust ventilation with full face air supplied breathing apparatus (hood or helmet type) is normally required. Unprotected personnel must vacate spraying area.</p> <p><b>NOTE:</b> Isocyanate vapours will not be adequately absorbed by organic vapour respirators. 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"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <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> </tbody> </table>	Type of Contaminant:	Air Speed:	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)					
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<p>Within each range the appropriate value depends on:</p> <table border="1"> <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 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 4-10 m/s (800-2000 f/min.) for extraction of solvents generated by spraying at a point 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> <ul style="list-style-type: none"> <li>All processes in which isocyanates are used should be enclosed wherever possible.</li> <li>Total enclosure, accompanied by good general ventilation, should be used to keep atmospheric concentrations below the relevant exposure standards.</li> <li>If total enclosure of the process is not feasible, local exhaust ventilation may be necessary. Local exhaust ventilation is essential where lower molecular weight isocyanates (such as TDI or HDI) is used or where isocyanate or polyurethane is sprayed.</li> <li>Where other isocyanates or pre-polymers are used and aerosol formation cannot occur, local exhaust ventilation may not be necessary if the atmospheric concentration can be kept below the relevant exposure standards.</li> <li>Where local exhaust ventilation is installed, exhaust vapours should not be vented to the exterior in such a manner as to create a hazard.</li> </ul>	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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<b>Personal protection</b>										
<b>Eye and face protection</b>	<ul style="list-style-type: none"> <li>Safety glasses with side shields.</li> <li>Chemical goggles.</li> <li>Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of 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]</li> </ul>									
<b>Skin protection</b>	See Hand protection below									
<b>Hands/feet protection</b>	<p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>For esters:</p>									

## Hardener FX Medium &amp; Fast (2K)

	<ul style="list-style-type: none"> <li>▶ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>▶ frequency and duration of contact,</li> <li>▶ chemical resistance of glove material,</li> <li>▶ glove thickness and</li> <li>▶ dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>▶ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>▶ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>▶ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>▶ Contaminated gloves should be replaced.</li> </ul> <p>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"> <li>▶ Do NOT wear natural rubber (latex gloves).</li> <li>▶ Isocyanate resistant materials include Teflon, Viton, nitrile rubber and some PVA gloves.</li> <li>▶ Protective gloves and overalls should be worn as specified in the appropriate national standard.</li> <li>▶ Contaminated garments should be removed promptly and should not be re-used until they have been decontaminated.</li> <li>▶ NOTE: Natural rubber, neoprene, PVC can be affected by isocyanates</li> <li>▶ <b>DO NOT use skin cream unless necessary and then use only minimum amount.</b></li> <li>▶ Isocyanate vapour may be absorbed into skin cream and this increases hazard.</li> </ul>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<p>All employees working with isocyanates must be informed of the hazards from exposure to the contaminant and the precautions necessary to prevent damage to their health. They should be made aware of the need to carry out their work so that as little contamination as possible is produced, and of the importance of the proper use of all safeguards against exposure to themselves and their fellow workers. Adequate training, both in the proper execution of the task and in the use of all associated engineering controls, as well as of any personal protective equipment, is essential.</p> <p>Employees exposed to contamination hazards should be educated in the need for, and proper use of, facilities, clothing and equipment and thereby maintain a high standard of personal cleanliness. Special attention should be given to ensuring that all personnel understand instructions, especially newly recruited employees and those with local-language difficulties, where they are known.</p> <ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ PVC Apron.</li> <li>▶ PVC protective suit may be required if exposure severe.</li> <li>▶ Eyewash unit.</li> <li>▶ Ensure there is ready access to a safety shower.</li> </ul> <p>Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.</p> <p>For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).</p> <p>Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.</p>
<b>Thermal hazards</b>	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:

Hardener FX Medium & Fast (2K)

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NATURAL RUBBER	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
TEFLON	C
VITON/BUTYL	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

**NOTE:** As a series of factors will influence the actual performance of the glove, a final

## Respiratory protection

Type A 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	A-AUS / Class 1	-	A-PAPR-AUS / Class 1
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	A-3	-
100+ x ES	-	Air-line**	-

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

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

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.

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### Information on basic physical and chemical properties

<b>Appearance</b>	Flammable liquid; does not mix with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	Not Available
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Available	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	50 (propylene glycol)	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Flammable.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water (g/L)</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available

## SECTION 10 STABILITY AND REACTIVITY

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

## SECTION 11 TOXICOLOGICAL INFORMATION

### Information on toxicological effects

<b>Inhaled</b>	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression, headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures.</p> <p>Prolonged exposure may cause headache, nausea and ultimately loss of consciousness.</p>
<b>Ingestion</b>	<p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <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>
<b>Skin Contact</b>	<p>The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either:</p> <ul style="list-style-type: none"> <li>▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or</li> <li>▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.</li> </ul> <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At</p>

	<p>the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>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>Limited evidence or practical experience suggests, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate.</p> <p>Repeated or prolonged exposure may cause severe inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>												
Chronic	<p>Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Respiratory sensitisation may result in allergic/asthma like responses; from coughing and minor breathing difficulties to bronchitis with wheezing, gasping.</p>												
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n-butyl acetate	<table border="1"> <thead> <tr> <th>TOXICITY</th> <th>IRRITATION</th> </tr> </thead> <tbody> <tr> <td>Dermal (rabbit) LD50: &gt;14080 mg/kg<sup>[1]</sup></td> <td>* [PPG]</td> </tr> <tr> <td>Inhalation (rat) LC50: 2000 ppm/4H<sup>[2]</sup></td> <td>Eye (human): 300 mg</td> </tr> <tr> <td>Inhalation (rat) LC50: 390 ppm/4h<sup>[2]</sup></td> <td>Eye (rabbit): 20 mg (open)-SEVERE</td> </tr> <tr> <td>Oral (rat) LD50: 10736 mg/kg<sup>[1]</sup></td> <td>Eye (rabbit): 20 mg/24h - moderate</td> </tr> <tr> <td></td> <td>Skin (rabbit): 500 mg/24h-moderate</td> </tr> </tbody> </table>	TOXICITY	IRRITATION	Dermal (rabbit) LD50: >14080 mg/kg <sup>[1]</sup>	* [PPG]	Inhalation (rat) LC50: 2000 ppm/4H <sup>[2]</sup>	Eye (human): 300 mg	Inhalation (rat) LC50: 390 ppm/4h <sup>[2]</sup>	Eye (rabbit): 20 mg (open)-SEVERE	Oral (rat) LD50: 10736 mg/kg <sup>[1]</sup>	Eye (rabbit): 20 mg/24h - moderate		Skin (rabbit): 500 mg/24h-moderate
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	Skin (rabbit): 500 mg/24h-moderate												
Legend:	<p>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</p>												

HEXAMETHYLENE DIISOCYANATE POLYMER	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).</p> <p>Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.</p> <p>Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.</p> <p>Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities.</p> <p>Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages.</p> <p>Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>* Bayer SDS ** Ardex SDS</p>
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Hardener FX Medium & Fast (2K)

**PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER**

for propylene glycol ethers (PGEs):  
 Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).  
 Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).  
 This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.  
 Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.  
 As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.  
 As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating  
 None are skin sensitizers.  
 In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).  
 Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.  
 One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.  
 In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.  
 The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*.  
 In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.  
 A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.  
 The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.]  
  
 A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.] \*Shin-Etsu SDS

**N-BUTYL ACETATE**

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

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
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## Hardener FX Medium &amp; Fast (2K)

hexamethylene diisocyanate polymer	LC50	96	Fish	0.015mg/L	3
hexamethylene diisocyanate polymer	EC50	72	Algae or other aquatic plants	>1000mg/L	2
hexamethylene diisocyanate polymer	EC50	24	Crustacea	>=100mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	LC50	96	Fish	100mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	48	Crustacea	373mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	96	Algae or other aquatic plants	9.337mg/L	3
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	504	Crustacea	>100mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	NOEC	336	Fish	47.5mg/L	2
n-butyl acetate	LC50	96	Fish	18mg/L	2
n-butyl acetate	EC50	48	Crustacea	=32mg/L	1
n-butyl acetate	EC50	96	Algae or other aquatic plants	1.675mg/L	3
n-butyl acetate	EC50	96	Fish	18mg/L	2
n-butyl acetate	NOEC	504	Crustacea	23mg/L	2

**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

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

**Persistence and degradability**

Ingredient	Persistence: Water/Soil	Persistence: Air
hexamethylene diisocyanate polymer	HIGH	HIGH
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
n-butyl acetate	LOW	LOW

**Bioaccumulative potential**

Ingredient	Bioaccumulation
hexamethylene diisocyanate polymer	LOW (LogKOW = 7.5795)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
n-butyl acetate	LOW (BCF = 14)

**Mobility in soil**

Ingredient	Mobility
hexamethylene diisocyanate polymer	LOW (KOC = 18560000)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
n-butyl acetate	LOW (KOC = 20.86)

**SECTION 13 DISPOSAL CONSIDERATIONS****Waste treatment methods**

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Containers may still present a chemical hazard/ danger when empty.</li> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> </ul> <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>▶ Reduction</li> <li>▶ Reuse</li> </ul>
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Continued...

## Hardener FX Medium &amp; Fast (2K)

- ▶ Recycling
  - ▶ Disposal (if all else fails)
- 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.
- ▶ **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.
  - ▶ Dispose of by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material).
  - ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

## SECTION 14 TRANSPORT INFORMATION

## Labels Required

	
Marine Pollutant	NO
HAZCHEM	•3Y

## Land transport (ADG)

UN number	1263
Packing group	III
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Environmental hazard	No relevant data
Transport hazard class(es)	Class : 3 Subrisk : Not Applicable
Special precautions for user	Special provisions : 163 223 * Limited quantity : 5 L

## Air transport (ICAO-IATA / DGR)

UN number	1263
Packing group	III
UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class : 3 ICAO / IATA Subrisk : Not Applicable ERG Code : 3L
Special precautions for user	Special provisions : A3 A72 A192 Cargo Only Packing Instructions : 366 Cargo Only Maximum Qty / Pack : 220 L Passenger and Cargo Packing Instructions : 355 Passenger and Cargo Maximum Qty / Pack : 60 L Passenger and Cargo Limited Quantity Packing Instructions : Y344 Passenger and Cargo Limited Maximum Qty / Pack : 10 L

## Sea transport (IMDG-Code / GGVSee)

UN number	1263
Packing group	III
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac solutions, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Environmental hazard	Not Applicable
Transport hazard class(es)	IMDG Class : 3 IMDG Subrisk : Not Applicable

<b>Special precautions for user</b>	EMS Number	F-E, S-E
	Special provisions	163 223 367 955
	Limited Quantities	5 L

**Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code**

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	propylene glycol monomethyl ether acetate, alpha-isomer	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	n-butyl acetate	Y

**SECTION 15 REGULATORY INFORMATION****Safety, health and environmental regulations / legislation specific for the substance or mixture****HEXAMETHYLENE DIISOCYANATE POLYMER(28182-81-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

**PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER(108-65-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

**N-BUTYL ACETATE(123-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (propylene glycol monomethyl ether acetate, alpha-isomer; n-butyl acetate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (hexamethylene diisocyanate polymer)
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****Ingredients with multiple cas numbers**

Name	CAS No
hexamethylene diisocyanate polymer	1192214-73-5, 28182-81-2, 53200-31-0
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 142300-82-1, 84540-57-8

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

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

**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.

# Hardener FX Slow (2K)

## DNA Custom Paints

Chemwatch: 16-4203  
Version No: 4.1.1.1  
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 18/09/2015  
Print Date: 26/11/2015  
Initial Date: **Not Available**  
L.GHS.AUS.EN

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

### Product Identifier

Product name	Hardener FX Slow (2K)
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Other means of identification	Part No.: HFXS-

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

Relevant identified uses	<b>CONTAINS</b> free organic isocyanate. Mixing and application requires special precautions and use of personal protective gear [APMF] Use according to manufacturer's directions.
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### Details of the supplier of the safety data sheet

Registered company name	DNA Custom Paints
Address	5-7 Keith Campbell Court Scoresby 3179 VIC Australia
Telephone	+61 3 9764 2088
Fax	+61 3 9764 1244
Website	www.dna-paints.com
Email	Not Available

### Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	+61 3 9573 3112
Other emergency telephone numbers	Not Available

## 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

	Min	Max
Flammability	3	4
Toxicity	2	3
Body Contact	2	3
Reactivity	1	2
Chronic	2	3

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

Poisons Schedule	S5
GHS Classification [1]	Flammable Liquid Category 3, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Respiratory Sensitizer Category 1, Skin Sensitizer Category 1, STOT - RE Category 2, Acute 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	  
SIGNAL WORD	<b>DANGER</b>
Hazard statement(s)	H226 Flammable liquid and vapour

Continued...

## Hardener FX Slow (2K)

H312	Harmful in contact with skin
H332	Harmful if inhaled
H315	Causes skin irritation
H319	Causes serious eye irritation
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled
H317	May cause an allergic skin reaction
H373	May cause damage to organs through prolonged or repeated exposure
H402	Harmful to aquatic life

### Precautionary statement(s) Prevention

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P233	Keep container tightly closed.
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.
P285	In case of inadequate ventilation wear respiratory protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

### Precautionary statement(s) Response

P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P363	Wash contaminated clothing before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.

### Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
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### 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
28182-81-2	30-60	<u>hexamethylene diisocyanate polymer</u>
1330-20-7	10-30	<u>xylene</u>
123-86-4	1-10	<u>n-butyl acetate</u>
112-07-2	1-10	<u>ethylene glycol monobutyl ether acetate</u>
108-65-6	1-10	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u>
		may contain monomeric isocyanate, as
822-06-0	<1	<u>hexamethylene diisocyanate</u>

## SECTION 4 FIRST AID MEASURES

### Description of first aid measures

Eye Contact	If this product comes in contact with the eyes:
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Continued...

## Hardener FX Slow (2K)

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

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

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

Followed acute or short term repeated exposures to ethylene glycol monoalkyl ethers and their acetates:

- ▶ Hepatic metabolism produces ethylene glycol as a metabolite.
- ▶ Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.
- ▶ Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure.

[Ellenhorn and Barceloux: Medical Toxicology]

For acute or short term repeated exposures to ethylene glycol:

- ▶ Early treatment of ingestion is important. Ensure emesis is satisfactory.
- ▶ Test and correct for metabolic acidosis and hypocalcaemia.
- ▶ Apply sustained diuresis when possible with hypertonic mannitol.
- ▶ Evaluate renal status and begin haemodialysis if indicated. [I.L.O.]
- ▶ Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- ▶ Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- ▶ Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- ▶ Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- ▶ Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.

Laitinen J., et al: *Occupational & Environmental Medicine* 1996; 53, 595-600

for simple esters:

**BASIC TREATMENT**

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ Monitor and treat, where necessary, for pulmonary oedema.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- ▶ Give activated charcoal.

**ADVANCED TREATMENT**

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

**EMERGENCY DEPARTMENT**

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. *EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994*

for lower alkyl ethers:

**BASIC TREATMENT**

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.

## Hardener FX Slow (2K)

- ▶ A low-stimulus environment must be maintained.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ Anticipate and treat, where necessary, for seizures.
- ▶ **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

### ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension without signs of hypovolaemia may require vasopressors.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

### EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Ethers may produce anion gap acidosis. Hyperventilation and bicarbonate therapy might be indicated.
- ▶ Haemodialysis might be considered in patients with impaired renal function.
- ▶ Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For sub-chronic and chronic exposures to isocyanates:

- ▶ This material may be a potent pulmonary sensitiser which causes bronchospasm even in patients without prior airway hyperreactivity.
- ▶ Clinical symptoms of exposure involve mucosal irritation of respiratory and gastrointestinal tracts.
- ▶ Conjunctival irritation, skin inflammation (erythema, pain vesiculation) and gastrointestinal disturbances occur soon after exposure.
- ▶ Pulmonary symptoms include cough, burning, substernal pain and dyspnoea.
- ▶ Some cross-sensitivity occurs between different isocyanates.
- ▶ Noncardiogenic pulmonary oedema and bronchospasm are the most serious consequences of exposure. Markedly symptomatic patients should receive oxygen, ventilatory support and an intravenous line.
- ▶ Treatment for asthma includes inhaled sympathomimetics (epinephrine [adrenalin], terbutaline) and steroids.
- ▶ Activated charcoal (1 g/kg) and a cathartic (sorbitol, magnesium citrate) may be useful for ingestion.
- ▶ Mydriatics, systemic analgesics and topical antibiotics (Sulamyd) may be used for corneal abrasions.
- ▶ There is no effective therapy for sensitised workers.

[Ellenhorn and Barceloux; Medical Toxicology]

**NOTE:** Isocyanates cause airway restriction in naive individuals with the degree of response dependant on the concentration and duration of exposure. They induce smooth muscle contraction which leads to bronchoconstrictive episodes. Acute changes in lung function, such as decreased FEV<sub>1</sub>, may not represent sensitivity.

[Karol & Jin, Frontiers in Molecular Toxicology, pp 56-61, 1992]

Personnel who work with isocyanates, isocyanate prepolymers or polyisocyanates should have a pre-placement medical examination and periodic examinations thereafter, including a pulmonary function test. Anyone with a medical history of chronic respiratory disease, asthmatic or bronchial attacks, indications of allergic responses, recurrent eczema or sensitisation conditions of the skin should not handle or work with isocyanates. Anyone who develops chronic respiratory distress when working with isocyanates should be removed from exposure and examined by a physician.

Further exposure must be avoided if a sensitivity to isocyanates or polyisocyanates has developed.

Following acute or short term repeated exposures to toluene:

- ▶ Toluene is absorbed across the alveolar barrier, the blood/air mixture being 11.2/15.6 (at 37 degrees C.) The concentration of toluene, in expired breath, is of the order of 18 ppm following sustained exposure to 100 ppm. The tissue/blood proportion is 1/3 except in adipose where the proportion is 8/10.
- ▶ Metabolism by microsomal mono-oxygenation, results in the production of hippuric acid. This may be detected in the urine in amounts between 0.5 and 2.5 g/24 hr which represents, on average 0.8 gm/gm of creatinine. The biological half-life of hippuric acid is in the order of 1-2 hours.
- ▶ Primary threat to life from ingestion and/or inhalation is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (eg cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO<sub>2</sub> <50 mm Hg or pCO<sub>2</sub> > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial damage has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenaline) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- ▶ Lavage is indicated in patients who require decontamination; ensure use.

### BIOLOGICAL EXPOSURE INDEX - BEI

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

Determinant	Index	Sampling Time	Comments
o-Cresol in urine	0.5 mg/L	End of shift	B
Hippuric acid in urine	1.6 g/g creatinine	End of shift	B, NS
Toluene in blood	0.05 mg/L	Prior to last shift of workweek	

NS: Non-specific determinant; also observed after exposure to other material

B: Background levels occur in specimens collected from subjects NOT exposed

For acute or short term repeated exposures to xylene:

- ▶ Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- ▶ Pulmonary absorption is rapid with about 60-65% retained at rest.
- ▶ Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO<sub>2</sub> < 50 mm Hg or pCO<sub>2</sub> > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

### BIOLOGICAL EXPOSURE INDEX - BEI

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

## Hardener FX Slow (2K)

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

### SECTION 5 FIREFIGHTING MEASURES

#### Extinguishing media

- ▶ Small quantities of water in contact with hot liquid may react violently with generation of a large volume of rapidly expanding hot sticky semi-solid foam.
  - ▶ Presents additional hazard when fire fighting in a confined space.
  - ▶ Cooling with flooding quantities of water reduces this risk.
  - ▶ Water spray or fog may cause frothing and should be used in large quantities.
  - ▶ Alcohol stable foam.
  - ▶ Dry chemical powder.
  - ▶ BCF (where regulations permit).
  - ▶ Carbon dioxide.
  - ▶ Water spray or fog - Large fires only.
- Do not** use a water jet to fight fire.

#### Special hazards arising from the substrate or mixture

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

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<p>Combustion products include:</p> <ul style="list-style-type: none"> <li>▶ Liquid and vapour are highly flammable.</li> <li>▶ Severe fire hazard when exposed to heat, flame and/or oxidisers.</li> <li>▶ Vapour may travel a considerable distance to source of ignition.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> </ul> <p>, carbon dioxide (CO<sub>2</sub>) carbon monoxide (CO) isocyanates and minor amounts of hydrogen cyanide nitrogen oxides (NO<sub>x</sub>) other pyrolysis products typical of burning organic material/When heated at high temperatures many isocyanates decompose rapidly generating a vapour which pressurises containers, possibly to the point of rupture. Release of toxic and/or flammable isocyanate vapours may then occur</p> <ul style="list-style-type: none"> <li>▶ Burns with acrid black smoke.</li> </ul>

### SECTION 6 ACCIDENTAL RELEASE MEASURES

#### Personal precautions, protective equipment and emergency procedures

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▶ Remove all ignition sources.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb small quantities with vermiculite or other absorbent material.</li> <li>▶ Wipe up.</li> <li>▶ Collect residues in a flammable waste container.</li> </ul>
<b>Major Spills</b>	<ul style="list-style-type: none"> <li>▶ Liquid Isocyanates and high isocyanate vapour concentrations will penetrate seals on self contained breathing apparatus - SCBA should be used inside encapsulating suit where this exposure may occur.</li> <li>For isocyanate spills of less than 40 litres (2 m<sup>2</sup>):</li> <li>▶ Evacuate area from everybody not dealing with the emergency, keep them upwind and prevent further access, remove ignition sources and, if inside building, ventilate area as well as possible.</li> <li>▶ Notify supervision and others as necessary.</li> <li>▶ Put on personal protective equipment (suitable respiratory protection, face and eye protection, protective suit, gloves and impermeable boots).</li> <li>▶ Control source of leakage (where applicable).</li> <li>▶ Dike the spill to prevent spreading and to contain additions of decontaminating solution.</li> <li>▶ Prevent the material from entering drains.</li> <li>▶ Estimate spill pool volume or area.</li> <li>▶ Absorb and decontaminate. - Completely cover the spill with wet sand, wet earth, vermiculite or other similar absorbent. - Add neutraliser (for suitable formulations: see below) to the adsorbent materials (equal to that of estimated spill pool volume). Intensify contact between spill, adsorbent and neutraliser by carefully mixing with a rake and allow to react for 15 minutes</li> <li>▶ Shovel absorbent/decontaminant solution mixture into a steel drum.</li> <li>▶ Decontaminate surface. - Pour an equal amount of neutraliser solution over contaminated surface. - Scrub area with a stiff bristle brush, using moderate pressure. - Completely cover decontaminant with vermiculite or other similar absorbent. - After 5 minutes, shovel absorbent/decontamination solution mixture into the same steel drum used above.</li> <li>▶ Monitor for residual isocyanate. If surface is decontaminated, proceed to next step. If contamination persists, repeat decontaminate procedure immediately above</li> <li>▶ Place loosely covered drum (release of carbon dioxide) outside for at least 72 hours. Label waste-containing drum appropriately. Remove waste materials for incineration.</li> <li>▶ Decontaminate and remove personal protective equipment.</li> <li>▶ Return to normal operation.</li> <li>▶ Conduct accident investigation and consider measures to prevent reoccurrence.</li> </ul> <p><b>Decontamination:</b> Treat isocyanate spills with sufficient amounts of isocyanate decontaminant preparation ("neutralising fluid"). Isocyanates and polyisocyanates are generally not</p>

## Hardener FX Slow (2K)

miscible with water. Liquid surfactants are necessary to allow better dispersion of isocyanate and neutralising fluids/ preparations. Alkaline neutralisers react faster than water/surfactant mixtures alone.

Typically, such a preparation may consist of:

Sawdust: 20 parts by weight Kieselguhr 40 parts by weight plus a mixture of (ammonia (s.g. 0.880) 8% v/v non-ionic surfactant 2% v/v water 90% v/v).

Let stand for 24 hours

Three commonly used neutralising fluids each exhibit advantages in different situations.

**Formulation A :**

liquid surfactant	0.2-2%
sodium carbonate	5-10%
water to	100%

**Formulation B**

liquid surfactant	0.2-2%
concentrated ammonia	3-8%
water to	100%

**Formulation C**

ethanol, isopropanol or butanol	50%
concentrated ammonia	5%
water to	100%

After application of any of these formulae, let stand for 24 hours.

Formulation B reacts faster than Formulation A. However, ammonia-based neutralisers should be used only under well-ventilated conditions to avoid overexposure to ammonia or if members of the emergency team wear suitable respiratory protection. Formulation C is especially suitable for cleaning of equipment from unreacted isocyanate and neutralizing under freezing conditions. Regard has to be taken to the flammability of the alcoholic solution.

- ▶ Avoid contamination with water, alkalies and detergent solutions.
- ▶ Material reacts with water and generates gas, pressurises containers with even drum rupture resulting.
- ▶ **DO NOT reseal container if contamination is suspected.**
- ▶ Open all containers with care.
- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ May be violently or explosively reactive.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Consider evacuation (or protect in place).
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse /absorb vapour.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Use only spark-free shovels and explosion proof equipment.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Absorb remaining product with sand, earth or vermiculite.
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ If contamination of drains or waterways occurs, advise emergency services.
- ▶ Reactions of isocyanates with amines, aldehydes, alcohols, alkali metals, ketones, mercaptans, strong oxidisers, hydrides, phenols, and peroxides can cause vigorous release of heat.
- ▶ Acids and bases initiate polymerisation reactions with isocyanates.
- ▶ Avoid reaction with compounds containing active hydrogen (such as water, ammonia, amines, alcohols, acids, etc.) - the reaction may be violent, the rate being dependent upon the active hydrogen compound and the presence of catalysts.
- ▶ Some isocyanates react with water to form amines and liberate carbon dioxide.
- ▶ Base-catalysed reactions of isocyanates with alcohols should be carried out in inert solvents. Such reactions in the absence of solvents often occur with explosive violence
- ▶ A range of exothermic decomposition energies for isocyanates is given as 20-30 kJ/mol.
- ▶ The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment.
- ▶ For example, in "open vessel processes" (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below 500 J/g are unlikely to present a danger, whilst those in "closed vessel processes" (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g.

BREThERICK: Handbook of Reactive Chemical Hazards, 4th Edition

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

## SECTION 7 HANDLING AND STORAGE

### Precautions for safe handling

#### Safe handling

- ▶ Containers, even those that have been emptied, may contain explosive vapours.
- ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.
- ▶ **DO NOT allow clothing wet with material to stay in contact with skin**
- ▶ Electrostatic discharge may be generated during pumping - this may result in fire.
- ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment.
- ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge ( $\leq 1$  m/sec until fill pipe submerged to twice its diameter, then  $\leq 7$  m/sec).
- ▶ Avoid splash filling.
- ▶ Do NOT use compressed air for filling discharging or handling operations.
- ▶ Avoid all personal contact, including inhalation.
- ▶ Wear protective clothing when risk of overexposure occurs.
- ▶ Use in a well-ventilated area.
- ▶ Prevent concentration in hollows and sumps.
- ▶ **DO NOT enter confined spaces until atmosphere has been checked.**
- ▶ Avoid smoking, naked lights or ignition sources.
- ▶ Avoid generation of static electricity.
- ▶ **DO NOT use plastic buckets.**
- ▶ Earth all lines and equipment.
- ▶ Use spark-free tools when handling.
- ▶ Avoid contact with incompatible materials.

Continued...

## Hardener FX Slow (2K)

	<ul style="list-style-type: none"> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> </ul> <p>The material when ready for use, i.e. with 2 parts mixed together, <b>CONTAINS</b> free organic isocyanate. Application requires control measures, special precautions and use of personal protective gear. [APMF]</p>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers in approved flammable liquid storage area.</li> <li>▶ Store away from incompatible materials in a cool, dry, well-ventilated area.</li> <li>▶ <b>DO NOT store in pits, depressions, basements or areas where vapours may be trapped.</b></li> <li>▶ No smoking, naked lights, heat or ignition sources.</li> <li>▶ Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access.</li> <li>▶ Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.</li> <li>▶ Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.</li> <li>▶ Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors.</li> <li>▶ Keep adsorbents for leaks and spills readily available.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul> <p>In addition, for tank storages (where appropriate):</p> <ul style="list-style-type: none"> <li>▶ Store in grounded, properly designed and approved vessels and away from incompatible materials.</li> <li>▶ For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.</li> <li>▶ Storage tanks should be above ground and diked to hold entire contents.</li> </ul> <p>for commercial quantities of isocyanates:</p> <ul style="list-style-type: none"> <li>▶ Isocyanates should be stored in adequately bunded areas. Nothing else should be kept within the same bunding. Pre-polymers need not be segregated. Drums of isocyanates should be stored under cover, out of direct sunlight, protected from rain, protected from physical damage and well away from moisture, acids and alkalis.</li> <li>▶ Where isocyanates are stored at elevated temperatures to prevent solidifying, adequate controls should be installed to prevent the high temperatures and precautions against fire should be taken.</li> <li>▶ Where stored in tanks, the more reactive isocyanates should be blanketed with a non-reactive gas such as nitrogen and equipped with absorptive type breather valve (to prevent vapour emissions)..</li> <li>▶ Transfer systems for isocyanates in bulk storage should be fully enclosed and use pump or vacuum systems. Warning signs, in appropriate languages, should be posted where necessary.</li> <li>▶ Areas in which polyurethane foam products are stored should be supplied with good general ventilation. Residual amounts of unreacted isocyanate may be present in the finished foam, resulting in hazardous atmospheric concentrations.</li> </ul>

### Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Packing as supplied by manufacturer.</li> <li>▶ Plastic containers may only be used if approved for flammable liquid.</li> <li>▶ Check that containers are clearly labelled and free from leaks.</li> <li>▶ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure.</li> <li>▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C)</li> <li>▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)</li> <li>▶ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used.</li> <li>▶ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages</li> <li>▶ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid cross contamination between the two liquid parts of product (kit).</li> <li>▶ If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur.</li> <li>▶ This excess heat may generate toxic vapour</li> <li>▶ Reactions of isocyanates with amines, aldehydes, alcohols, alkali metals, ketones, mercaptans, strong oxidisers, hydrides, phenols, and peroxides can cause vigorous release of heat.</li> <li>▶ Acids and bases initiate polymerisation reactions with isocyanates.</li> <li>▶ Avoid reaction with compounds containing active hydrogen (such as water, ammonia, amines, alcohols, acids, etc.) - the reaction may be violent, the rate being dependent upon the active hydrogen compound and the presence of catalysts.</li> <li>▶ Some isocyanates react with water to form amines and liberate carbon dioxide.</li> <li>▶ Base-catalysed reactions of isocyanates with alcohols should be carried out in inert solvents. Such reactions in the absence of solvents often occur with explosive violence</li> <li>▶ Avoid reaction with water, alcohols and detergent solutions.</li> <li>▶ Isocyanates and thioisocyanates are incompatible with many classes of compounds, reacting exothermically to release toxic gases. Reactions with amines, strong bases, aldehydes, alcohols, alkali metals, ketones, mercaptans, strong oxidisers, hydrides, phenols, and peroxides can cause vigorous releases of heat. Acids and bases initiate polymerisation reactions in these materials.</li> <li>▶ Isocyanates easily form adducts with carbodiimides, isothiocyanates, ketenes, or with substrates containing activated CC or CN bonds.</li> <li>▶ Some isocyanates react with water to form amines and liberate carbon dioxide. This reaction may also generate large volumes of foam and heat. Foaming in confined spaces may produce pressure in confined spaces or containers. Gas generation may pressurise drums to the point of rupture.</li> <li>▶ Do NOT reseal container if contamination is expected</li> <li>▶ Open all containers with care</li> <li>▶ Base-catalysed reactions of isocyanates with alcohols should be carried out in inert solvents. Such reactions in the absence of solvents often occur with explosive violence,</li> <li>▶ Isocyanates will attack and embrittle some plastics and rubbers.</li> <li>▶ A range of exothermic decomposition energies for isocyanates is given as 20-30 kJ/mol.</li> <li>▶ The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment.</li> <li>▶ For example, in "open vessel processes" (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below</li> </ul>

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- ▶ 500 J/g are unlikely to present a danger, whilst those in "closed vessel processes" (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g.
- BREThERICK: Handbook of Reactive Chemical Hazards, 4th Edition
- ▶ Avoid strong acids, bases.



+                    X                    X                    X                    +                    X                    +

- X** — Must not be stored together  
**0** — May be stored together with specific preventions  
**+** — May be stored together

## SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

## Control parameters

## OCCUPATIONAL EXPOSURE LIMITS (OEL)

## INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	hexamethylene diisocyanate polymer	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Sen
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	350 mg/m3 / 80 ppm	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	n-butyl acetate	n-Butyl acetate	713 mg/m3 / 150 ppm	950 mg/m3 / 200 ppm	Not Available	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether acetate	2-Butoxyethyl acetate	133 mg/m3 / 20 ppm	333 mg/m3 / 50 ppm	Not Available	Sk
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	274 mg/m3 / 50 ppm	548 mg/m3 / 100 ppm	Not Available	Sk
Australia Exposure Standards	hexamethylene diisocyanate	Hexamethylene diisocyanate	Not Available	Not Available	Not Available	Not Available

## EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
hexamethylene diisocyanate polymer	Hexamethylene diisocyanate polymer	7.8 mg/m3	86 mg/m3	510 mg/m3
xylene	Xylenes	Not Available	Not Available	Not Available
n-butyl acetate	Butyl acetate, n-	Not Available	Not Available	Not Available
ethylene glycol monobutyl ether acetate	Butoxyethanol acetate, 2-; (Ethylene glycol monobutyl ether acetate)	20 ppm	20 ppm	73 ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
hexamethylene diisocyanate	Hexamethylene diisocyanate; (1,6-Diisocyanatohexane)	0.005 ppm	0.02 ppm	0.8 ppm

Ingredient	Original IDLH	Revised IDLH
hexamethylene diisocyanate polymer	Not Available	Not Available
xylene	1,000 ppm	900 ppm
n-butyl acetate	10,000 ppm	1,700 [LEL] ppm
ethylene glycol monobutyl ether acetate	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
hexamethylene diisocyanate	Not Available	Not Available

## MATERIAL DATA

for propylene glycol monomethyl ether acetate (PGMEA)

Saturated vapour concentration: 4868 ppm at 20 C.

A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternal ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

for 1,6-hexamethylene diisocyanate (HDI):

The toxicological action of HDI is similar to that of toluene diisocyanate and the TLV-TWA is analogous. In light of reported asthmatic/ respiratory sensitisation-like responses in HDI exposed workers, individuals who may be hypersusceptible or otherwise unusually responsive may not be adequately protected at this limit.

## Exposure controls

Appropriate engineering	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

Continued...

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	<p>effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</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>For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.</p> <p>Spraying of material or material in admixture with other components must be carried out in conditions conforming to local state regulations. Local exhaust ventilation with full face air supplied breathing apparatus (hood or helmet type) is normally required. Unprotected personnel must vacate spraying area.</p> <p><b>NOTE:</b> Isocyanate vapours will not be adequately absorbed by organic vapour respirators. 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="359 519 1487 609"> <tr> <td>Type of Contaminant:</td> <td>Air Speed:</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> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="359 667 1487 833"> <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 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 4-10 m/s (800-2000 f/min.) for extraction of solvents generated by spraying at a point 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> <p>   In confined spaces wear full-face air supplied breathing apparatus.</p>	Type of Contaminant:	Air Speed:	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.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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<p><b>Personal protection</b></p>															
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of 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]</li> </ul>														
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>														
<p><b>Hands/feet protection</b></p>	<p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>▶ frequency and duration of contact,</li> <li>▶ chemical resistance of glove material,</li> <li>▶ glove thickness and</li> <li>▶ dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>▶ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>▶ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>▶ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>▶ Contaminated gloves should be replaced.</li> </ul> <p>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"> <li>▶ Isocyanate resistant materials include Teflon, Viton, nitrile rubber and some PVA gloves.</li> <li>▶ Protective gloves and overalls should be worn as specified in the appropriate national standard.</li> <li>▶ Contaminated garments should be removed promptly and should not be re-used until they have been decontaminated.</li> <li>▶ NOTE: Natural rubber, neoprene, PVC can be affected by isocyanates</li> </ul>														
<p><b>Body protection</b></p>	<p>See Other protection below</p>														
<p><b>Other protection</b></p>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ PVC Apron.</li> <li>▶ PVC protective suit may be required if exposure severe.</li> <li>▶ Eyewash unit.</li> <li>▶ Ensure there is ready access to a safety shower.</li> </ul> <p>Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.</p> <p>For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).</p>														

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	Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.
<b>Thermal hazards</b>	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:

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Material	CPI
##ethylene glycol monobutyl ether	acetate
##n-butyl	acetate
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
TEFLON	C
VITON	C
VITON/BUTYL	C
##hexamethylene	diisocyanate

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

## Respiratory protection

Type A 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	A-AUS / Class 1	-	A-PAPR-AUS / Class 1
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	A-3	-
100+ x ES	-	Air-line**	-

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

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

## Information on basic physical and chemical properties

<b>Appearance</b>	Flammable liquid; does not mix with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	Not Available
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	23	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Flammable.	<b>Oxidising properties</b>	Not Available

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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)	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	>1	VOC g/L	Not Available

### SECTION 10 STABILITY AND REACTIVITY

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

### SECTION 11 TOXICOLOGICAL INFORMATION

#### Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation hazard is increased at higher temperatures.</p> <p>Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue.</p> <p>Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.</p>
Ingestion	<p>Considered an unlikely route of entry in commercial/industrial environments The liquid may produce considerable gastrointestinal discomfort and may be harmful or toxic if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis</p> <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>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> <li>▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.</li> </ul> <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression.</p>
Eye	<p>The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.</p> <p>Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>Eye contact with alkyl ethers (vapours or liquid) may produce irritation, redness and lachrymation.</p>
Chronic	<p>Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>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> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</p>

## Hardener FX Slow (2K)

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

Persons with a history of asthma or other respiratory problems or are known to be sensitised, should not be engaged in any work involving the handling of isocyanates. [CCTRADE-Bayer, APMF]

A 90-day inhalation study in rats with polymeric MDI (6 hours/day, 5 days/week) produced moderate to severe hyperplastic inflammatory lesions in the nasal cavities and lungs at levels of 8 mg/m<sup>3</sup> or greater.

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

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

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

Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.

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

Respiratory sensitisation may result in allergic/asthma like responses; from coughing and minor breathing difficulties to bronchitis with wheezing, gasping.

Sensitisation may give severe responses to very low levels of exposure, in situations where exposure may occur.

Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities.

Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages.

Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates.

Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.

Hardener FX Slow (2K)	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
hexamethylene diisocyanate polymer	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >5000 mg/kg <sup>[2]</sup>	Skin (rabbit): 500 mg - moderate
	Inhalation (rat) LC50: 18.5 mg/L/1he <sup>[2]</sup>	
	Oral (rat) LD50: >10000 mg/kg <sup>[2]</sup>	
xylene	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1700 mg/kg <sup>[2]</sup>	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 5000 ppm/4h <sup>[2]</sup>	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kg <sup>[2]</sup>	Eye (rabbit): 87 mg mild
		Skin (rabbit):500 mg/24h moderate
n-butyl acetate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >14080 mg/kg <sup>[1]</sup>	* [PPG]
	Inhalation (rat) LC50: 2000 ppm/4Hg <sup>[2]</sup>	Eye ( human): 300 mg
	Inhalation (rat) LC50: 390 ppm/4h <sup>[2]</sup>	Eye (rabbit): 20 mg (open)-SEVERE
	Oral (rat) LD50: 10736 mg/kg <sup>[1]</sup>	Eye (rabbit): 20 mg/24h - moderate
		Skin (rabbit): 500 mg/24h-moderate
ethylene glycol monobutyl ether acetate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 1500 mg/kg <sup>[2]</sup>	Eye (rabbit): 500 mg/24hr - mild
	Oral (rat) LD50: 7012.4 mg/kg <sup>[1]</sup>	Skin (rabbit): 500 mg - mild
propylene glycol monomethyl ether acetate, alpha-isomer	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	* [CCINFO]
	Inhalation (rat) LC50: 4345 ppm/6h <sup>[2]</sup>	Nil reported
	Oral (rat) LD50: >14.1 ml <sup>[1]</sup>	
hexamethylene diisocyanate	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >7000 mg/kg <sup>[1]</sup>	Not Available
	Inhalation (rat) LC50: 0.06 mg/L/4h <sup>[2]</sup>	
	Inhalation (rat) LC50: 0.124 mg/L/4H <sup>[2]</sup>	

## Hardener FX Slow (2K)

Inhalation (rat) LC50: 0.462 mg/L/4H<sup>[2]</sup>

Oral (rat) LD50: 710 mg/kg<sup>[1]</sup>

**Legend:**

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

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.

Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).

Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.

Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO<sub>2</sub>, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO<sub>2</sub>, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

**Respiratory Effects.** Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

**Cardiovascular Effects.** Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12-24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

**Gastrointestinal Effects.** Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

**Musculoskeletal Effects.** Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

**Hepatic Effects.** Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

**Renal Effects.** Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

**Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

**Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

**Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

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**Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

**Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

**Genotoxic Effects:** Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m<sup>3</sup> for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m<sup>3</sup>. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m<sup>3</sup>), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating

None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m<sup>3</sup> (600 ppm) for PnB and 2,010 mg/m<sup>3</sup> (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m<sup>3</sup> (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m<sup>3</sup> (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m<sup>3</sup>) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m<sup>3</sup>). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m<sup>3</sup>), with decreased body weights occurring at 3000 ppm (11058 mg/m<sup>3</sup>). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.

The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.]

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

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.

Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens

**HEXAMETHYLENE  
DIISOCYANATE POLYMER**

## Hardener FX Slow (2K)

	<p>in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.</p> <p>Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.</p> <p>Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities.</p> <p>Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages.</p> <p>Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>* Bayer SDS ** Ardex SDS</p>
<p style="text-align: center;"><b>XYLENE</b></p>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>The substance is classified by IARC as Group 3: <b>NOT</b> classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. Reproductive effector in rats</p>
<p style="text-align: center;"><b>N-BUTYL ACETATE</b></p>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<p style="text-align: center;"><b>ETHYLENE GLYCOL MONOBUTYL ETHER ACETATE</b></p>	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>For ethylene glycol:</p> <p>Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO<sub>2</sub>, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO<sub>2</sub>, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.</p> <p><b>Respiratory Effects.</b> Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).</p> <p><b>Cardiovascular Effects.</b> Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.</p> <p>Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.</p> <p><b>Gastrointestinal Effects.</b> Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.</p> <p><b>Musculoskeletal Effects.</b> Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.</p> <p><b>Hepatic Effects.</b> Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.</p> <p><b>Renal Effects.</b> Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.</p> <p><b>Metabolic Effects.</b> One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).</p> <p><b>Neurological Effects:</b> Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to</p>

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generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

**Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

**Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

**Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

**Genotoxic Effects:** Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

**Acute Toxicity:** Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m<sup>3</sup>) for EGHE, LC50 > 400ppm (2620 mg/m<sup>3</sup>) for EGBEA to LC50 > 2132 ppm (9061 mg/m<sup>3</sup>) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitizers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

**Repeat dose toxicity:** The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.

**Mutagenicity:** In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

**Carcinogenicity:** In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity.

**Reproductive and developmental toxicity.** The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m<sup>3</sup> and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m<sup>3</sup>), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m<sup>3</sup>), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m<sup>3</sup>) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m<sup>3</sup> (rabbit-EGPE), 100 ppm or 425 mg/m<sup>3</sup> (rat-EGPE), 50 ppm or 241 mg/m<sup>3</sup> (rat EGBE) and 100 ppm or 483 mg/m<sup>3</sup> (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m<sup>3</sup> (rat and rabbit-EGHE).

mae

for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m<sup>3</sup> for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m<sup>3</sup>. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m<sup>3</sup>), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating.

None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A

**PROPYLENE GLYCOL  
MONOMETHYL ETHER  
ACETATE, ALPHA-ISOMER**

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dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m<sup>3</sup> (600 ppm) for PnB and 2,010 mg/m<sup>3</sup> (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m<sup>3</sup> (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m<sup>3</sup> (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m<sup>3</sup>) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m<sup>3</sup>). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m<sup>3</sup>), with decreased body weights occurring at 3000 ppm (11058 mg/m<sup>3</sup>). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.]

A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.] \*Shin-Etsu SDS

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.

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.

Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).

Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.

Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.

for 1,6-hexamethylene diisocyanate:

Exposures to HDI are often associated with exposures to its prepolymers, especially to a trimeric biuretic prepolymer of HDI (HDI-BT), which is widely used as a hardener in automobile and airplane paints, and which typically contains 0.5-1% unreacted HDI. There is evidence that diisocyanate prepolymers may induce asthma at the same or greater frequency as the monomers; therefore, there is a need to assess the potential for human exposure to prepolymeric HDI as well as monomeric HDI.

1,6-Hexamethylene diisocyanate is corrosive to the skin and the eye.

1,6-Hexamethylene diisocyanate was found to induce dermal and respiratory sensitization in animals and humans. There is no threshold known for this effect.

Inhalation studies with repeated exposures to 1,6-hexamethylene diisocyanate vapor show that the respiratory tract is the target with 1,6-hexamethylene diisocyanate showing primarily upper respiratory tract lesions (nasal cavity). 1,6-Hexamethylene diisocyanate did not show a neurotoxic effect in a combined reproduction/developmental/neurotoxicity study. Life-time inhalation exposure to rats revealed a progression of non-neoplastic respiratory tract lesions, primarily to the nasal cavity, and represented the sequelae of non-specific irritation. Based on the presence of only reversible tissue responses to irritation at the low concentration of 0.005 ppm, this concentration was a NOAEL. No carcinogenic potential in rats was observed after life-time inhalation.

1,6-Hexamethylene diisocyanate showed no mutagenic activity *in vitro* in bacterial and in mammalian cell test systems.

1,6-Hexamethylene diisocyanate showed no clastogenic activity *in vivo*.

1,6-Hexamethylene diisocyanate has no effect on fertility and post-natal viability through post-natal day 4 in the rat after inhalation up to 0.299 ppm. The overall NOEL was 0.005 ppm.

Inhalation of 1,6-hexamethylene diisocyanate during the pregnancy of rats produced maternal effects (nasal turbinate histopathology) at concentrations <sup>3</sup> 0.052 ppm. No developmental toxicity was observed up to 0.308 ppm.

Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities.

Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages.

Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitized people can react to very low levels of airborne isocyanates.

Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.

HEXAMETHYLENE  
DIISOCYANATE

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
hexamethylene diisocyanate polymer	LC50	96	Fish	0.015mg/L	3
hexamethylene diisocyanate polymer	EC50	72	Algae or other aquatic plants	>1000mg/L	2
hexamethylene diisocyanate polymer	EC50	24	Crustacea	>=100mg/L	2
xylene	LC50	96	Fish	0.0013404mg/L	4
xylene	EC50	48	Crustacea	>3.4mg/L	2
xylene	EC50	72	Algae or other aquatic plants	4.6mg/L	2
xylene	EC50	24	Crustacea	0.711mg/L	4
xylene	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
n-butyl acetate	LC50	96	Fish	18mg/L	2
n-butyl acetate	EC50	48	Crustacea	=32mg/L	1
n-butyl acetate	EC50	96	Algae or other aquatic plants	1.675mg/L	3
n-butyl acetate	EC50	96	Fish	18mg/L	2
n-butyl acetate	NOEC	504	Crustacea	23mg/L	2
ethylene glycol monobutyl ether acetate	LC50	96	Fish	>20- <40mg/L	2
ethylene glycol monobutyl ether acetate	EC50	48	Crustacea	=37mg/L	1
ethylene glycol monobutyl ether acetate	EC50	96	Algae or other aquatic plants	3.228mg/L	3
ethylene glycol monobutyl ether acetate	EC0	48	Crustacea	=10mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	LC50	96	Fish	100mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	48	Crustacea	373mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	96	Algae or other aquatic plants	9.337mg/L	3
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	504	Crustacea	>100mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	NOEC	336	Fish	47.5mg/L	2
hexamethylene diisocyanate	LC50	96	Fish	22mg/L	1
hexamethylene diisocyanate	EC50	72	Algae or other aquatic plants	>77.4mg/L	2
hexamethylene diisocyanate	EC0	24	Crustacea	<0.33mg/L	1
hexamethylene diisocyanate	NOEC	72	Algae or other aquatic plants	11.7mg/L	2

#### 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.

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

### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
hexamethylene diisocyanate polymer	HIGH	HIGH
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)

## Hardener FX Slow (2K)

n-butyl acetate	LOW	LOW
ethylene glycol monobutyl ether acetate	LOW	LOW
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
hexamethylene diisocyanate	LOW	LOW

### Bioaccumulative potential

Ingredient	Bioaccumulation
hexamethylene diisocyanate polymer	LOW (LogKOW = 7.5795)
xylene	MEDIUM (BCF = 740)
n-butyl acetate	LOW (BCF = 14)
ethylene glycol monobutyl ether acetate	LOW (BCF = 3.2)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
hexamethylene diisocyanate	LOW (LogKOW = 3.1956)

### Mobility in soil

Ingredient	Mobility
hexamethylene diisocyanate polymer	LOW (KOC = 18560000)
n-butyl acetate	LOW (KOC = 20.86)
ethylene glycol monobutyl ether acetate	LOW (KOC = 10)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
hexamethylene diisocyanate	LOW (KOC = 5864)

## SECTION 13 DISPOSAL CONSIDERATIONS

### Waste treatment methods

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Containers may still present a chemical hazard/ danger when empty.</li> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> </ul> <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>▶ Reduction</li> <li>▶ Reuse</li> <li>▶ Recycling</li> <li>▶ Disposal (if all else fails)</li> </ul> <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Recycle wherever possible.</li> <li>▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.</li> <li>▶ Dispose of by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material).</li> <li>▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.</li> </ul>
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## SECTION 14 TRANSPORT INFORMATION

### Labels Required

	
<b>Marine Pollutant</b>	NO
<b>HAZCHEM</b>	•3Y

**Land transport (ADG)**

<b>UN number</b>	1263
<b>Packing group</b>	III
<b>UN proper shipping name</b>	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
<b>Environmental hazard</b>	No relevant data
<b>Transport hazard class(es)</b>	Class : 3 Subrisk : Not Applicable
<b>Special precautions for user</b>	Special provisions : 163 223 * Limited quantity : 5 L

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

<b>UN number</b>	1263
<b>Packing group</b>	III
<b>UN proper shipping name</b>	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)
<b>Environmental hazard</b>	No relevant data
<b>Transport hazard class(es)</b>	ICAO/IATA Class : 3 ICAO / IATA Subrisk : Not Applicable ERG Code : 3L
<b>Special precautions for user</b>	Special provisions : A3 A72 A192 Cargo Only Packing Instructions : 366 Cargo Only Maximum Qty / Pack : 220 L Passenger and Cargo Packing Instructions : 355 Passenger and Cargo Maximum Qty / Pack : 60 L Passenger and Cargo Limited Quantity Packing Instructions : Y344 Passenger and Cargo Limited Maximum Qty / Pack : 10 L

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

<b>UN number</b>	1263
<b>Packing group</b>	III
<b>UN proper shipping name</b>	PAINT (including paint, lacquer, enamel, stain, shellac solutions, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
<b>Environmental hazard</b>	Not Applicable
<b>Transport hazard class(es)</b>	IMDG Class : 3 IMDG Subrisk : Not Applicable
<b>Special precautions for user</b>	EMS Number : F-E, S-E Special provisions : 163 223 367 955 Limited Quantities : 5 L

**Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code**

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	xylene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	n-butyl acetate	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	ethylene glycol monobutyl ether acetate	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	propylene glycol monomethyl ether acetate, alpha-isomer	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	hexamethylene diisocyanate	Y

**SECTION 15 REGULATORY INFORMATION**

**Safety, health and environmental regulations / legislation specific for the substance or mixture****HEXAMETHYLENE DIISOCYANATE POLYMER(28182-81-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

**XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**N-BUTYL ACETATE(123-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

**ETHYLENE GLYCOL MONOBUTYL ETHER ACETATE(112-07-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

**PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER(108-65-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

**HEXAMETHYLENE DIISOCYANATE(822-06-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (propylene glycol monomethyl ether acetate, alpha-isomer; xylene; n-butyl acetate; ethylene glycol monobutyl ether acetate; hexamethylene diisocyanate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (hexamethylene diisocyanate polymer)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
<b>Legend:</b>	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****Ingredients with multiple cas numbers**

Name	CAS No
hexamethylene diisocyanate polymer	1192214-73-5, 28182-81-2, 53200-31-0
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 142300-82-1, 84540-57-8

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

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

**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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