

9200 Structural Epoxy Adhesive (Part A)

MG Chemicals UK Limited

Version No: 1.1 Safety Data Sheet (Conforms to Regulations (EC) No 2015/830) Chemwatch Hazard Alert Code: 2

Issue Date: 01/04/2016 Print Date: 04/04/2016 Initial Date: 01/04/2016 L.REACH.GBR.EN

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

1.1.Product Identifier

Product name	9200 Structural Epoxy Adhesive (Part A)
Synonyms	SDS Code: 9200-Part A; 9200-25ML, 9200-50ML
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol a diglycidyl ether resin, solid)
Other means of identification	Not Available

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

Relevant identified uses	Epoxy adhesive resin for use with hardeners	
Uses advised against	Not Applicable	

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	+(44) 1663 362888	+(1) 800-201-8822
Fax	Not Available	+(1) 800-708-9888
Website	Website Not Available www.mgchemicals.com	
Email	sales@mgchemicals.com	Info@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	CHEMTREC	Not Available
Emergency telephone numbers	+(44) 870-8200418	Not Available
Other emergency telephone numbers	+(1) 703-527-3887	Not Available

SECTION 2 HAZARDS IDENTIFICATION

2.1.Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

2.2. Label elements

CLP label elements





SIGNAL WORD WARNING

Hazard statement(s)

H315	Causes skin irritation.	
H319	Causes serious eye irritation.	
H317	May cause an allergic skin reaction.	
H411	Toxic to aquatic life with long lasting effects.	

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Supplementary statement(s)

Not Applicable.

Precautionary statement(s) Prevention

P280	P280 Wear protective gloves/protective clothing/eye protection/face protection.	
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.	
P273	Avoid release to the environment.	
P272	Contaminated work clothing should not be allowed out of the workplace.	

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

P501

Dispose of contents/container in accordance with local regulations.

2.3. Other hazards

Cumulative effects may result following exposure*.

Limited evidence of a carcinogenic effect*.

Possible respiratory sensitizer*.

REACh - Art.57-59: The mixture does not contain Substances of Very High Concern (SVHC) at the SDS print date.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.28064-14-4 2.Not Available 3.Not Available 4.Not Available	46	bisphenol F glycidyl ether/ formaldehyde copolymer	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2; H315, H319, H317, H411, EUH019 [1]
1.25068-38-6 2.500-033-5 3.603-074-00-8 4.01-2119456619-26-XXXX	32	bisphenol A diglycidyl ether resin, solid	Eye Irritation Category 2, Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2; H319, H315, H317, H411 [3]
1.14807-96-6 2.238-877-9 3.Not Available 4.Not Available	17	<u>talc</u>	Acute Toxicity (Inhalation) Category 4, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation); H332, H335 [1]
1.60506-81-2 2.262-270-8 3.Not Available 4.Not Available	2	dipentaerythritol pentaacrylate	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation); H315, H319, H317, H335 [1]
Legend:	<u> </u>	by Chemwatch; 2. Classification drav	vn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

General

If skin contact occurs:

- ► Immediately remove all contaminated clothing, including footwear.
- Flush skin and hair with running water (and soap if available).
- ▶ Seek medical attention in event of irritation.

If this product comes in contact with the eyes:

Wash out immediately with fresh running water.

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	 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. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. If fumes or combustion products are inhaled remove from contaminated area. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor, without delay. Immediately give a glass of water. First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. 		
Eye Contact	If this product comes in contact with the eyes: • Wash out immediately with fresh running water. • Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. • Seek medical attention without delay; if pain persists or recurs seek medical attention. • Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.		
Skin Contact	If skin contact occurs: Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation.		
Inhalation	 If fumes or combustion products are inhaled remove from contaminated area. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital. or doctor, without delay. 		
Ingestion	► Immediately give a glass of water.		

▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

4.2 Most important symptoms and effects, both acute and delayed

4.3. Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

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

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result	
5.3. Advice for firefighters		
	Alert Fire Brigade and tell them location and nature of hazard.	

Fire Fighting

- ▶ Wear full body protective clothing with breathing apparatus.
- ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area.
- Avoid spraying water onto liquid pools.
- DO NOT approach containers suspected to be hot.
- Cool fire exposed containers with water spray from a protected location.
- ▶ If safe to do so, remove containers from path of fire.

Fire/Explosion Hazard

- ▶ Combustible. Slight fire hazard when exposed to heat or flame.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO).
- May emit acrid smoke.
- ▶ Mists containing combustible materials may be explosive.

Combustion products include; carbon dioxide (CO2) aldehydes silicon dioxide (SiO2) other pyrolysis products typical of burning organic material

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills

Environmental hazard - contain spillage.

Clean up all spills immediately.

Avoid breathing vapours and contact with skin and eyes.

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- ► Control personal contact with the substance, by using protective equipment.
- ▶ Contain and absorb spill with sand, earth, inert material or vermiculite.
- ▶ Wipe up.
- ▶ Place in a suitable, labelled container for waste disposal.

Environmental hazard - contain spillage

Chemical Class: phenols and cresols

For release onto land: recommended sorbents listed in order of priority.

LAND SPILL - SMALL

cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
wood fiber - pillow	1	throw	pitchfork	R, P, DGC, RT
foamed glass - pillow	2	shovel	shovel	R, W, P, DGC
sorbent clay - particulate	2	shovel	shovel	R, I, P
wood fibre - particulate	3	shovel	shovel	R, W, P, DGC

LAND SPILL - MEDIUM

Major Spills

cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	R, SS, DGC
wood fiber - particulate	4	blower	skiploader	R, W, P, DGC
expanded moneral - particulate	4	blower	skiploader	R, I, W, P, DGC

Leaend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT:Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

Moderate hazard.

- ► Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ► No smoking, naked lights or ignition sources.
- ► Increase ventilation.
- Stop leak if safe to do so.
- Contain spill with sand, earth or vermiculite.
- ▶ 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.

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE

7.1. Precautions for safe handling

Safe handling

- Avoid all personal contact, including inhalation.
 - ▶ Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- ► Prevent concentration in hollows and sumps.
- ▶ DO NOT enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights or ignition sources.
- 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.
 - Work clothes should be laundered sep
 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.
 - ▶ DO NOT allow clothing wet with material to stay in contact with skin

Fire and explosion protection

See section 5

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Other information

- Store in original containers.
- Keep containers securely sealed
- ▶ Store in a cool, dry, well-ventilated area.
- Store away from incompatible materials and foodstuff containers.
- Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

7.2. Conditions for safe storage, including any incompatibilities

Metal can or drum Suitable container Packaging as recommended by manufacturer ▶ Check all containers are clearly labelled and free from leaks Avoid reaction with amines, mercaptans, strong acids and oxidising agents Phenols are incompatible with strong reducing substances such as hydrides, nitrides, alkali metals, and sulfides. Avoid use of aluminium, copper and brass alloys in storage and process equipment. Heat is generated by the acid-base reaction between phenols and bases Phenols are sulfonated very readily (for example, by concentrated sulfuric acid at room temperature), these reactions generate heat. Phenols are nitrated very rapidly, even by dilute nitric acid. Nitrated phenols often explode when heated. Many of them form metal salts that tend toward detonation by rather mild shock. Storage incompatibility Glycidyl ethers: real form unstable peroxides on storage in air ,light, sunlight, UV light or other ionising radiation, trace metals - inhibitor should be maintained at adequate levels $\blacksquare \ \ \, \text{may polymerise in contact with heat, organic and inorganic free radical producing initiators} \\$ ▶ may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide attack some forms of plastics, coatings, and rubber

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	talc	Talc, respirable dust	1 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
bisphenol F glycidyl ether/ formaldehyde copolymer	Phenol, polymer with formaldehyde, oxiranylmethyl ether	12 mg/m3	130 mg/m3	790 mg/m3
bisphenol A diglycidyl ether resin, solid	Epoxy resin (EPON 1001)	90 mg/m3	990 mg/m3	5900 mg/m3
bisphenol A diglycidyl ether resin, solid	Epoxy resin (EPON 1007)	90 mg/m3	990 mg/m3	5900 mg/m3
bisphenol A diglycidyl ether resin, solid	Epoxy resin (EPON 820)	41 mg/m3	450 mg/m3	2700 mg/m3
bisphenol A diglycidyl ether resin, solid	Epoxy resin ERL-2795	32 mg/m3	350 mg/m3	2100 mg/m3
bisphenol A diglycidyl ether resin, solid	Polypropylene glycol, (chloromethyl) oxirane polymer	6 mg/m3	66 mg/m3	400 mg/m3
talc	Talc	2 mg/m3	2 mg/m3	2.6 mg/m3

Ingredient	Original IDLH	Revised IDLH
bisphenol F glycidyl ether/ formaldehyde copolymer	Not Available	Not Available
bisphenol A diglycidyl ether resin, solid	Not Available	Not Available
talc	N.E. mg/m3 / N.E. ppm	1,000 mg/m3
dipentaerythritol pentaacrylate	Not Available	Not Available

MATERIAL DATA

For talc (a form of magnesium silicate):

Most health problems associated with occupational exposure to talcs appear to evolve mostly from the nonplatiform content of the talc being mined or milled (being the asbestos-like amphiboles, serpentines (asbestiformes) and other minerals in the form of acicular, prismatic and fibrous crystals including, possibly, asbestos).

Because of severe health effects associated with exposures to asbestos, regulatory agencies tend to regard all elongate mineral crystal particles, whether prismatic, acicular, fibrous, as asbestos

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Consideration is also given to their respirability, their width being less than or equal to 3 um. Only limited data, however, exists on the health effects of elongate mineral particles having prismatic, acicular or fibrous (non-asbestos) forms. Experimental evidence indicates that the carcinogen potential of mineral fibres is related to the size class with diameter of 8 um with shorter, thicker particles having little biological activity.

Dust of nonfibrous talc, consisting entirely of platiform talc crystals and containing no asbestos poses a relatively small respiratory hazard.

Difficulties exist, however, in the determination of asbestos as cleavage fragments of prismatic or acicular crystals, nonasbestos fibres and asbestos fibres are very similar.

Subject to an accurate determination of asbestos and crystalline silica, exposure at or below the recommended TLV-TWA, is thought to protect workers from the significant risk of nonmalignant respiratory effects associated with talc dusts.

8.2. Exposure controls

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

The basic types of engineering controls are:

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

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

Employers may need to use multiple types of controls to prevent employee overexposure.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.

An approved self contained breathing apparatus (SCBA) may be required in some situations.

Provide adequate ventilation in warehouse or closed storage area. 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.

8.2.1. Appropriate engineering controls

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

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

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

8.2.2. Personal protection











o.z.z. Personal protection

► Safety glasses with side shields

Chemical goggles

Eye and face protection

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

Skin protection

See Hand protection below

NOTE:

- ► The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- ► Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Hands/feet protection

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: • frequency and duration of contact,

- chemical resistance of glove material,
- ► glove thickness and
- dexterity

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

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- ► When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

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	 ▶ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. ▶ Contaminated gloves should be replaced. 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. ▶ When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butatoluene rubber), boots and aprons. ▶ DO NOT use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber or polyethylene gloves (which absorb the resin). ▶ DO NOT use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use.
Body protection	See Other protection below
Other protection	Overalls. P.V.C. apron. Barrier cream. Skin cleansing cream. Eye wash unit.
Thermal hazards	Not Available

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the 'Exposure Standard' (or ES), respiratoryprotection is required. Degree of protection varies with both face-piece and Class offilter; 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 P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^{^ -} Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling pointorganic compounds(below 65 degC)

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

9.1. Information on basic physical and chemical properties

Appearance	light grey		
Physical state	Liquid	Relative density (Water = 1)	1.25
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	>150	Molecular weight (g/mol)	Not Available
Flash point (°C)	>113	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Not Available	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2.Chemical stability	 Unstable in the presence of incompatible materials. Product is considered stable. Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2

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10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 TOXICOLOGICAL INFORMATION

11.1. Information on toxicological effects

Inhaled

Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.

The material has NOT been classified by EC Directives or other classification systems as 'harmful by inhalation'. This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols.

Ingestion

The material has NOT been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.

Skin Contact

Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant 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. 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.

The material may accentuate any pre-existing dermatitis condition

Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.

Open cuts, abraded or irritated skin should not be exposed to this material

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.

Eve

Evidence exists, or practical experience predicts, that the material may cause 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. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis);

temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

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. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.

Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.

All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.

Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether.

Chronic

A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not n-butyl glycidyl ether, induced morphological transformation in mammalian cells in vitro. n-Butyl glycidyl ether induced micronuclei in mice in vivo following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations in vivo or chromosomal aberrations in animal cells in vitro. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in *Drosophila*. The glycidyl ethers were generally mutagenic to bacteria

Bisphenol F, bisphenol A, fluorine-containing bisphenol A (bisphenol AF), and other diphenylalkanes were found to be oestrogenic in a bioassay with MCF7 human breast cancer cells in culture Bisphenol F (4,4'-dihydroxydiphenylmethane) has been reported to exhibit oestrogen agonistic properties in the uterotrophic assay. Bisphenol F (BPF) is present in the environment and as a contaminant of food. Humans may, therefore, be exposed to BP. BPF has been shown to have genotoxic and endocrine-disruptor properties in a human hepatoma cell line (HepG2), which is a model system for studies of xenobiotic toxicity. BPF was largely metabolised into the corresponding sulfate by the HepG2 cell line. BPF was metabolised into both sulfate and glucuronide by human hepatocytes, but with differences between individuals. The metabolism of BPF in both HepG2 cells and human hepatocytes suggests the existence of a

Bisphenol F was orally administered at doses 0, 20, 100 and 500 mg/kg per day for at least 28 days, but no clear endocrine-mediated changes were detected, and it was concluded to have no endocrine-mediated effects in young adult rats. On the other hand, the main effect of bisphenol F was concluded to be liver toxicity based on clinical biochemical parameters and liver weight, but without histopathological changes. The no-observed-effect level for bisphenol F is concluded to be under 20 mg/kg per day since decreased body weight accompanied by decreased serum total cholesterol, glucose, and albumin values were observed in the female rats given 20 mg/kg per day or higher doses of bisphenol F.

Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone.. Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review.

A 2009 study on Chinese workers in bisphenol A factories found that workers were four times more likely to report erectile dysfunction, reduced sexual desire and overall dissatisfaction with their sex life than workers with no heightened bisphenol A exposure. Bisphenol A workers were also seven times more likely to have ejaculation difficulties. They were also more likely to report reduced sexual function within one year of beginning employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties.

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Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades'

One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials'

One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood.

A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells [whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes.

Bisphenol A is the isopropyl adduct of 4,4'-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called 'cytostatic hormones'. Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown to be oestrogenic in vitro; such sealants may represent an additional source of xenoestrogens in humans and may be the cause of additional concerns in children.

Concerns have been raised about the possible developmental effects on the foetus/embryo or neonate resulting from the leaching of bisphenol A from epoxy linings in metal cans which come in contact with food-stuffs.

Many drugs, including naproxen, salicylic acid, carbamazepine and mefenamic acid can, in vitro, significantly inhibit bisphenol A glucuronidation (detoxification).

9200 Structural Epoxy	TOXICITY	1	RRITATION	
Adhesive (Part A)	Not Available	1	Not Available	
	TOXICITY		IRRITATION	
	dermal (rat) LD50: 4000 mg/kg*e ^[2]	nal (rat) LD50: 4000 mg/kg*e ^[2] * [Ciba-Geigy]		
bisphenol F glycidyl ether/	Oral (rat) LD50: 4000 mg/kg*t ^[2] Effects transient		Effects transient	
formaldehyde copolymer	Eyes * (-) (-) Slight irri		ritant	
			May cause allergic response	
			Skin * (-) (-) Slight irr	itant
	TOXICITY			IRRITATION
bisphenol A diglycidyl ether				Nilusasantani

bisphenol A diglycidyl ether	
resin, solid	

TOXICITY	IRRITATION
dermal (rat) LD50: >800 mg/kg ^[1]	Nil reported
Oral (rat) LD50: 13447 mg/kg ^[1]	

talc

TOXICITY	IRRITATION
Not Available	Skin (human): 0.3 mg/3d-l mild

dipentaerythritol pentaacrylate

TOXICITY	IRRITATION
Not Available	Not Available

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

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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 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.

The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics

Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in

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activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl mojety markedly influence the activities.

Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.

Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane: data presented here may be taken as representative.

for 1,2-butylene oxide (ethyloxirane):

Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic

BISPHENOL F GLYCIDYL ETHER/ FORMALDEHYDE COPOLYMER

BISPHENOL A DIGLYCIDYL

ETHER RESIN, SOLID

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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

No significant acute toxicological data identified in literature search.

CAUTION: Epoxy resin products may contain sensitising glycidyl ethers, even when these are not mentioned in the information given for the product. The likely occurrence of these is greatly reduced in solid grades of the resin.

TALC

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. No significant acute toxicological data identified in literature search.

For talc (a form of magnesium silicate)

The overuse of talc in nursing infants has resulted in pulmonary oedema, pneumonia and death within hours of inhaling talcum powder. The powder dries the mucous membranes of the bronchioles, disrupts pulmonary clearance, clogs smaller airways. Victims display wheezing, rapid or difficult breathing, increased pulse, cyanosis, fever. Mild exposure may cause relatively minor inflammatory lung disease.

Long term exposure may show wheezing, weakness, productive cough, limited chest expansion, scattered rales, cyanosis.

The substance is classified by IARC as Group 3:

NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.

DIPENTAERYTHRITOL **PENTAACRYLATE**

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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.

UV (ultraviolet)/ EB (electron beam) acrylates are generally of low toxicity

UV/EB acrylates are divided into two groups; 'stenomeric' and 'eurymeric' acrylates.

The first group consists of well-defined acrylates which can be described by a simple idealised chemical; they are low molecular weight species with a very narrow weight distribution profile.

The eurymeric acrylates cannot be described by an idealised structure and may differ fundamentally between various suppliers; they are of relatively high molecular weigh and possess a wide weight distribution.

Stenomeric acrylates are usually more hazardous than the eurymeric substances. Stenomeric acrylates are also well defined which allows comparison and exchange of toxicity data - this allows more accurate classification.

The stenomerics cannot be classified as a group; they exhibit substantial variation.

Where no 'official' classification for acrylates and methacrylates exists, there has been cautious attempts to create classifications in the absence of contrary evidence. For example

Monalkyl or monoarylesters of acrylic acids should be classified as R36/37/38 and R51/53

Monoalkyl or monoaryl esters of methacrylic acid should be classified as R36/37/38

No significant acute toxicological data identified in literature search.

Based on the available oncogenicity data and without a better understanding of the carcinogenic mechanism the Health and Environmental Review Division (HERD), Office of Toxic Substances (OTS), of the US EPA previously concluded that all chemicals that contain the acrylate or methacrylate moiety (CH2=CHCOO or CH2=C(CH3)COO) should be considered to be a carcinogenic hazard unless shown otherwise by adequate testing. This position has now been revised and acrylates and methacrylates are no longer *de facto* carcinogens.

Acute Toxicity	0	Carcinogenicity	0
Skin Irritation/Corrosion	✓	Reproductivity	0
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	0
Respiratory or Skin sensitisation	~	STOT - Repeated Exposure	0
Mutagenicity	0	Aspiration Hazard	0

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

12.1. Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
bisphenol A diglycidyl ether resin, solid	LC50	96	Fish	1.2mg/L	2
bisphenol A diglycidyl ether resin, solid	EC50	48	Crustacea	1.1mg/L	2
bisphenol A diglycidyl ether resin, solid	EC50	48	Crustacea	1.7mg/L	2
bisphenol A diglycidyl ether resin, solid	NOEC	504	Crustacea	0.3mg/L	2
bisphenol A diglycidyl ether resin, solid	EC50	72	Algae or other aquatic plants	9.4mg/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				

 $\label{total constraints} \mbox{Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.}$

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

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

For bisphenol A and related bisphenols:

In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms. Among freshwater organisms, fish appear to be the most sensitive species. Evidence of endocrine-related effects in fish, aquatic invertebrates, amphibians and reptiles has been reported at environmentally relevant exposure levels lower than those required for acute toxicity. There is a widespread variation in reported values for endocrine-related effects, but many fall in the range of 1 ug/L to 1 mg/L

Bisphenol A. its derivatives and analogues, can be released from polymers, resins and certain substances by metabolic products

As an environmental contaminant, bisphenol A interferes with nitrogen fixation at the roots of leguminous plants associated with the bacterial symbiont Sinorhizobium meliloti. Despite a half-life in the soil of only 1-10 days, its ubiquity makes it an important pollutant. According to Environment Canada, "initial assessment shows that at low levels, bisphenol A can harm fish and organisms over time. Studies also indicate that it can currently be found in municipal wastewater." However, a study conducted in the United States found that 91-98% of bisphenol A may be removed from water during treatment at municipal water treatment plants.

A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations. A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.

Although abundant data are available on the toxicity of bisphenol-A (2,2-bis (4-hydroxydiphenyl)propane; (BPA) A variety of BPs were examined for their acute toxicity against Daphnia magna, mutagenicity, and oestrogenic activity using the Daphtoxkit (Creasel Ltd.), the umu test system, and the yeast two-hybrid system, respectively, in comparison with BPA. BPA was moderately toxic to D. magna (48-h EC50 was 10 mg/l) according to the current U.S. EPA acute toxicity evaluation standard, and it was weakly oestrogenic with 5 orders of magnitude lower activity than that of the natural estrogen 17 beta-oestradiol in the yeast screen, while no mutagenicity was observed. All seven BPs tested here showed moderate to slight acute toxicity, no mutagenicity, and weak

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oestrogenic activity as well as BPA. Some of the BPs showed considerably higher oestrogenic activity than BPA, and others exhibited much lower activity. Bisphenol S (bis(4hydroxydiphenyl)sulfone) and bis(4-hydroxyphenyl)sulfide) showed oestrogenic activity.

Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F ([bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem,

Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe3+ ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.

Significant environmental findings are limited. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit common characteristics with respect to environmental fate and ecotoxicology. One such oxirane is ethyloxirane and data presented here may be taken as representative

for 1.2-butylene oxide (ethyloxirane):

Environmental fate: Ethyloxirane is highly soluble in water and has a very low soil-adsorption coefficient, which suggests that if released to water, adsorption of ethyloxirane to sediment and suspended solids is not expected. Volatilisation of ethyloxirane from water surfaces would be expected based on the moderate estimated Henry's Law constant. If ethyloxirane is released to soil, it is expected to have low adsorption and thus very high mobility. Volatilisation from moist soil and dry soil surfaces is expected, based on its vapour pressure. It is expected that ethyloxirane exists solely as a vapour in ambient atmosphere, based on its very high vapour pressure. Ethyloxirane may also be removed from the atmosphere by wet deposition processes, considering its relatively high water solubility.

Persistence: The half-life in air is about 5.6 days from the reaction of ethyloxirane with photochemically produced hydroxyl radicals which indicates that this chemical meets the persistence criterion in air (half-life of = 2 days)*.

Ethyloxirane is hydrolysable, with a half-life of 6.5 days, and biodegradable up to 100% degradation and is not expected to persist in water. A further model-predicted biodegradation half-life of 15 days in water was obtained and used to predict the half-life of this chemical in soil and sediment by applying Boethling's extrapolation factors (t1/2water:t1/2 soil:t1/2sediment = 1:1:4) (Boethling 1995). According to these values, it can be concluded that ethyloxirane does not meet the persistence criteria in water and soil (half-lives = 182 days) and sediments (half-life = 365 days).

Experimental and modelled log Kow values of 0.68 and 0.86, respectively, indicate that the potential for bioaccumulation of ethyloxirane in organisms is likely to be low. Modelled bioaccumulation -factor (BAF) and bioconcentration -factor (BCF) values of 1 to 17 L/kg indicate that ethyloxirane does not meet the bioaccumulation criteria (BCF/BAF = 5000)*

Ecotoxicity:

Experimental ecotoxicological data for ethyloxirane (OECD 2001) indicate low to moderate toxicity to aquatic organisms. For fish and water flea, acute LC50/EC50 values vary within a narrow range of 70-215 mg/L; for algae, toxicity values exceed 500 mg/L, while for bacteria they are close to 5000 mg/L

* Persistence and Bioaccumulation Regulations (Canada 2000).

Environmental toxicity is a function of the n-octanol/ water partition coefficient (log Pow, log Kow). Phenols with log Pow > 7.4 are expected to exhibit low toxicity to aquatic organisms. However the toxicity of phenols with a lower log Pow is variable, ranging from low toxicity (LC50 values >100 mg/l) to highly toxic (LC50 values <1 mg/l) dependent on log Pow, molecular weight and substitutions on the aromatic ring. Dinitrophenols are more toxic than predicted from QSAR estimates. Hazard information for these groups is not generally available. **DO NOT** discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
bisphenol A diglycidyl ether resin, solid	HIGH	HIGH

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
bisphenol A diglycidyl ether resin, solid	LOW (LogKOW = 2.6835)

12.4. Mobility in soil

Ingredient	Mobility
bisphenol A diglycidyl ether resin, solid	LOW (KOC = 51.43)

12.5.Results of PBT and vPvB assessment

	P	В	Т
Relevant available data	Not Available	Not Available	Not Available
PBT Criteria fulfilled?	Not Available	Not Available	Not Available

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging

disposa

- ► Containers may still present a chemical hazard/ danger when empty.
- ▶ Return to supplier for reuse/ recycling if possible.

Otherwise:

- ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. 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. A Hierarchy of Controls seems to be common - the user should investigate:

▶ Reduction

- Reuse
- 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

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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 or consult manufacturer for recycling options. ► Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Waste treatment options

Sewage disposal options

Labels Required



Not Available

Not Available

Limited Quantity (For 9200-25ML, 9250-50ML kits ship as per Part B)

Land transport (ADR)			
14.1.UN number	3082		
14.2.Packing group			
14.3.UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol a diglycidyl ether resin, solid)		
14.4.Environmental hazard	Not Applicable		
14.5. Transport hazard class(es)	Class 9 Subrisk Not Applicable		
14.6. Special precautions for user	Hazard identification (Kemler) 90 Classification code M6 Hazard Label 9 Special provisions 274 335 375 601 Limited quantity 5 L		

Air transport (ICAO-IATA / DGR)

The transport (roze izaze)	,			
14.1. UN number	3082	3082		
14.2. Packing group	III			
14.3. UN proper shipping name	Environmentally hazard	dous substance, li	quid, n.o.s. * (contains	s bisphenol a digly
14.4. Environmental hazard	Not Applicable			
14.5. Transport hazard	ICAO/IATA Class	9		
class(es)	ICAO / IATA Subrisk	Not Applicable		
` ,	ERG Code	9L		
	Special provisions			A97 A158 A197
	Cargo Only Packing I	nstructions		964
	Cargo Only Maximum	Qty / Pack		450 L
14.6. Special precautions for user	Passenger and Cargo Packing Instructions		964	
usui	Passenger and Cargo	Passenger and Cargo Maximum Qty / Pack		450 L
	Passenger and Cargo	Limited Quantity	Packing Instructions	Y964
	Passenger and Cargo	Limited Maximur	n Qty / Pack	30 kg G

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9200 Structural Epoxy Adhesive (Part A)

14.1. UN number 3082 14.2. Packing group 14.3. UN proper shipping ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol a diglycidyl ether resin, solid) 14.4. Environmental hazard Marine Pollutant IMDG Class 14.5. Transport hazard class(es) IMDG Subrisk Not Applicable **EMS Number** F-A. S-F 14.6. Special precautions for Special provisions 274 335 969 usei Limited Quantities 5 L

Inland waterways transport (ADN)

14.1. UN number	3082		
14.2. Packing group	III		
14.3. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol a diglycidyl ether resin, solid)		
14.4. Environmental hazard	Not Applicable		
14.5. Transport hazard class(es)	9 Not Applicable		
14.6. Special precautions for user	Classification code M6 Special provisions 274; 335; 375; 601 Limited quantity 5 L Equipment required PP Fire cones number 0		

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

BISPHENOL F GLYCIDYL ETHER/ FORMALDEHYDE COPOLYMER(28064-14-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

BISPHENOL A DIGLYCIDYL ETHER RESIN, SOLID(25068-38-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and

Packaging of Substances and Mixtures - Annex VI

TALC(14807-96-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)

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

UK Workplace Exposure Limits (WELs)

DIPENTAERYTHRITOL PENTAACRYLATE(60506-81-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable -: 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
ingredient	OAO HUMBEI	IIIdex No	LOTIA DOSSICI

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bisphenol F glycidyl ether/ formaldehyde copolymer	28064-14-4	Not Available		Not Available	
Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)		Pictogran Code(s)	ms Signal Word	Hazard Statement Code(s)
1	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2		GHS07, G	HS09, Wng	H315, H317, H319
2	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Skin Sens. 1B, Not Classified, STOT SE 3		GHS07, G	iHS09, Wng	H315, H317, H319, H335

 $Harmonisation\ Code\ 1 = The\ most\ prevalent\ classification.\ Harmonisation\ Code\ 2 = The\ most\ severe\ classification.$

Ingredient	CAS number	Index No	ECHA Dossier
bisphenol A diglycidyl ether resin, solid	25068-38-6	603-074-00-8	01-2119456619-26-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317, H319
2	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Not Classified, Acute Tox. 4, Aquatic Chronic 3, Aquatic Chronic 4, Skin Corr. 1A, Aquatic Acute 1, Aquatic Chronic 1	GHS09, Wng, GHS08, Dgr, GHS07	H315, H317, H319, H372
1	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317, H319
2	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Skin Sens. 1B, Skin Sens. 1A, Not Classified	GHS07, GHS09, Wng	H315, H317, H319
1	Skin Irrit. 2, Skin Sens. 1A, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317
2	Skin Irrit. 2, Skin Sens. 1A, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
talc	14807-96-6	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Not Classified	Wng, GHS08, Dgr	H319, H332, H372, H335, H350
2	Not Classified, Eye Irrit. 2, Acute Tox. 4, STOT RE 1, STOT SE 3, Carc. 1A, Aquatic Chronic 4	Wng, GHS08, Dgr	H319, H332, H372, H335, H350

 $Harmonisation\ Code\ 1 = The\ most\ prevalent\ classification.\ Harmonisation\ Code\ 2 = The\ most\ severe\ classification.$

Ingredient	CAS number	Index No	ECHA Dossier
dipentaerythritol pentaacrylate	60506-81-2	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Eye Irrit. 2	GHS07, Wng	H319
2	Not Classified, Eye Irrit. 2, Skin Irrit. 2, STOT SE 3, Skin Sens. 1, Aquatic Chronic 4	GHS07, Wng	H319, H315, H335, H317

 $Harmonisation\ Code\ 1 = The\ most\ prevalent\ classification.\ Harmonisation\ Code\ 2 = The\ most\ severe\ classification.$

National Inventory	Status
Australia - AICS	Υ
Canada - DSL	Υ
Canada - NDSL	N (bisphenol A diglycidyl ether resin, solid; talc; dipentaerythritol pentaacrylate; bisphenol F glycidyl ether/ formaldehyde copolymer)
China - IECSC	Υ
Europe - EINEC / ELINCS / NLP	N (bisphenol F glycidyl ether/ formaldehyde copolymer)
Japan - ENCS	N (bisphenol A diglycidyl ether resin, solid)
Korea - KECI	Υ
New Zealand - NZIoC	Υ
Philippines - PICCS	Υ
USA - TSCA	Υ
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

Full text Risk and Hazard codes

H332	Harmful if inhaled.		
H335	y cause respiratory irritation.		
H350	May cause cancer.		
H372	Causes damage to organs.		

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Other information

Ingredients with multiple cas numbers

Name	CAS No		
bisphenol F glycidyl ether/ formaldehyde copolymer	28064-14-4, 42616-71-7, 59029-73-1, 94422-39-6		
bisphenol A diglycidyl ether resin, solid	25068-38-6, 25085-99-8		

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

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

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

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



9200 Structural Epoxy Adhesive (Part B)

MG Chemicals UK Limited

Version No: 1.4

Safety Data Sheet (Conforms to Regulations (EC) No 2015/830)

Chemwatch Hazard Alert Code: 4

Issue Date: 02/04/2016 Print Date: 02/04/2016 Initial Date: 02/04/2016 L.REACH.GBR.EN

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

1.1.Product Identifier

Product name	9200 Structural Epoxy Adhesive (Part B)
Synonyms	SDS Code: 9200-Part B; 9200-25ML, 9200-50ML
Proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains c18 fatty acid dimers/ tetraethylenepentamine polyamides and triethylenetetramine)
Other means of identification	Not Available

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

Relevant identified uses	Epoxy adhesive hardener for use with resins		
Uses advised against	Not for use as a spray coating		

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	+(44) 1663 362888	+(1) 800-201-8822
Fax	Not Available +(1) 800-708-9888	
Website	Website Not Available www.mgchemicals.com	
Email sales@mgchemicals.com Info@mgche		Info@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	CHEMTREC	Not Available
Emergency telephone numbers	+(44) 870-8200418	Not Available
Other emergency telephone numbers	+(1) 703-527-3887	Not Available

SECTION 2 HAZARDS IDENTIFICATION

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] ^[1]	Skin Corrosion/Irritation Category 1B, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2
Legend:	Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

2.2. Label elements

CLP label elements





SIGNAL WORD DANG

Hazard statement(s)

H314	Causes severe skin burns and eye damage.		
H317	May cause an allergic skin reaction.		
H411	Toxic to aquatic life with long lasting effects.		

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Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P260	Do not breathe dust/fume/gas/mist/vapours/spray.		
P280	Wear protective gloves/protective clothing/eye protection/face protection.		
P273	Avoid release to the environment.		
P272	P272 Contaminated work clothing should not be allowed out of the workplace.		

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.			
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.			
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.			
P310	nmediately call a POISON CENTER/doctor/physician/first aider.			
P302+P352	IF ON SKIN: Wash with plenty of water and soap.			
P363	Wash contaminated clothing before reuse.			
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.			
P362+P364	Take off contaminated clothing and wash it before reuse.			
P391	Collect spillage.			
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.			

Precautionary statement(s) Storage

D405	01
P405	Store locked up.

Precautionary statement(s) Disposal

P501 Dispose of contents/container in accordance with local regulations.

2.3. Other hazards

Ingestion may produce health damage*.

Cumulative effects may result following exposure*.

Possible respiratory sensitizer*.

REACh - Art.57-59: The mixture does not contain Substances of Very High Concern (SVHC) at the SDS print date.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.68683-29-4 2.Not Available 3.Not Available 4.Not Available	32	acrylonitrile/ butadiene copolymer amine terminated	Not Applicable
1.7727-43-7 2.231-784-4 3.Not Available 4.01-2119491274-35-XXXX	30	barium sulfate	Not Applicable
1.68410-23-1 2.Not Available 3.Not Available 4.Not Available	24	C18 fatty acid dimers/ tetraethylenepentamine polyamides	Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation); H315, H318, H335 [1]
1.68082-29-1 2.500-191-5 3.Not Available 4.Not Available	7	tall oil/ triethylenetetramine polyamides	Not Applicable
1.112-24-3 2.203-950-6 3.612-059-00-5 4.Not Available	3	<u>triethylenetetramine</u>	Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1B, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 3; H312, H314, H317, H412 [3]
1.140-31-8 2.205-411-0 3.612-105-00-4	2	N-aminoethylpiperazine	Acute Toxicity (Dermal) Category 4, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1B, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 3; H312, H302, H314, H317, H412 [3]

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4.01-2119471486-30-XXXX

Legend:

1. Classified by Chemwatch: 2. Classification drawn from EC Directive 67/548/EEC - Annex I: 3. Classification drawn from EC Directive 1272/2008 - Annex I VI 4 Classification drawn from C&I

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

If skin or hair contact occurs:

- Immediately flush body and clothes with large amounts of water, using safety shower if available.
- Quickly remove all contaminated clothing, including footwear.
- Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.
- ► Transport to hospital, or doctor.

For amines:

- In case of major exposure to liquid amine, promptly remove any contaminated clothing, including rings, watches, and shoe, preferably under a safety shower.
- ▶ Wash skin for 15 to 30 minutes with plenty of water and soap. Call a physician immediately.
- ▶ Remove and dry-clean or launder clothing soaked or soiled with this material before reuse. Dry cleaning of contaminated clothing may be more effective than normal laundering.
- ▶ Inform individuals responsible for cleaning of potential hazards associated with handling contaminated clothing.
- Discard contaminated leather articles such as shoes, belts, and watchbands.
- Note to Physician: Treat any skin burns as thermal burns. After decontamination, consider the use of cold packs and topical antibiotics.

If this product comes in contact with the eyes:

- ► Immediately hold eyelids apart and flush the eye continuously with running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.
- Transport to hospital or doctor without delay.
- Famoval of contact lenses after an eye injury should only be undertaken by skilled personnel.

For amines:

General

- Fill fliquid amines come in contact with the eyes, irrigate immediately and continuously with low pressure flowing water, preferably from an eye wash fountain, for 15 to 30 minutes
- For more effective flushing of the eyes, use the fingers to spread apart and hold open the eyelids. The eyes should then be "rolled" or moved in all directions.
- Seek immediate medical attention, preferably from an ophthalmologist.
- ▶ If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested
- Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if
- Transport to hospital, or doctor.
 - ▶ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema.
 - Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).
 - As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested.
 - ▶ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered.

This must definitely be left to a doctor or person authorised by him/her

(ICSC13719)

For amines:

- ▶ All employees working in areas where contact with amine catalysts is possible should be thoroughly trained in the administration of appropriate first aid
- ▶ Experience has demonstrated that prompt administration of such aid can minimize the effects of accidental exposure.
- ▶ Promptly move the affected person away from the contaminated area to an area of fresh air.
- ▶ Keep the affected person calm and warm, but not hot.
- ▶ If breathing is difficult, oxygen may be administered by a qualified person.
- If breathing stops, give artificial respiration. Call a physician at once.
- ▶ For advice, contact a Poisons Information Centre or a doctor at once.
- Urgent hospital treatment is likely to be needed.
- If swallowed do **NOT** induce vom
- ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- ▶ Observe the patient carefully.
- ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- Transport to hospital or doctor without delay.

For amines:

- ▶ If liquid amine are ingested, have the affected person drink several glasses of water or milk.
- Do not induce vomiting.
- ▶ Immediately transport to a medical facility and inform medical personnel about the nature of the exposure. The decision of whether to induce vomiting should be made by an attending physician.

Skin Contact

If this product comes in contact with the eyes:

- ▶ Immediately hold eyelids apart and flush the eye continuously with running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.
- Transport to hospital or doctor without delay. **Eye Contact**
 - ► Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

For amines:

- ▶ If liquid amines come in contact with the eyes, irrigate immediately and continuously with low pressure flowing water, preferably from an eye wash fountain, for 15 to 30 minutes
- For more effective flushing of the eyes, use the fingers to spread apart and hold open the eyelids. The eyes should then be "rolled" or moved in all directions.
- Seek immediate medical attention, preferably from an ophthalmologist.

If skin or hair contact occurs:

- ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available.
- Quickly remove all contaminated clothing, including footwear.
 - Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.
 - ▶ Transport to hospital, or doctor.

For amines:

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- ▶ In case of major exposure to liquid amine, promptly remove any contaminated clothing, including rings, watches, and shoe, preferably under a safety shower.
 - Wash skin for 15 to 30 minutes with plenty of water and soap. Call a physician immediately.
- Remove and dry-clean or launder clothing soaked or soiled with this material before reuse. Dry cleaning of contaminated clothing may be more effective than normal laundering.
- Inform individuals responsible for cleaning of potential hazards associated with handling contaminated clothing.
- Discard contaminated leather articles such as shoes, belts, and watchbands.
- ▶ Note to Physician: Treat any skin burns as thermal burns. After decontamination, consider the use of cold packs and topical antibiotics.
- If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested.
- ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- ▶ Transport to hospital, or doctor.
- Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema.
- ▶ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).
- As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested.
- ▶ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered.

This must definitely be left to a doctor or person authorised by him/her.

(ICSC13719)

Inhalation

Ingestion

For amines:

- All employees working in areas where contact with amine catalysts is possible should be thoroughly trained in the administration of appropriate first aid procedures.
- ▶ Experience has demonstrated that prompt administration of such aid can minimize the effects of accidental exposure.
- ▶ Promptly move the affected person away from the contaminated area to an area of fresh air.
- ▶ Keep the affected person calm and warm, but not hot.
- If breathing is difficult, oxygen may be administered by a qualified person.
- If breathing stops, give artificial respiration. Call a physician at once
- ▶ For advice, contact a Poisons Information Centre or a doctor at once.
- Urgent hospital treatment is likely to be needed.
- If swallowed do NOT induce vomiting
- ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- Observe the patient carefully.
- ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- ► Transport to hospital or doctor without delay.

For amines

- If liquid amine are ingested, have the affected person drink several glasses of water or milk.
- Do not induce vomiting.
- Immediately transport to a medical facility and inform medical personnel about the nature of the exposure. The decision of whether to induce vomiting should be made by an attending physician.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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

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

Alkalis continue to cause damage after exposure.

INGESTION:

Milk and water are the preferred diluents

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

- ▶ Neutralising agents should never be given since exothermic heat reaction may compound injury.
- * Catharsis and emesis are absolutely contra-indicated.
- * Activated charcoal does not absorb alkali.
- * Gastric lavage should not be used.

Supportive care involves the following:

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

SKIN AND EYE:

▶ Injury should be irrigated for 20-30 minutes.

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

For amine

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

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

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

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

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

Medical surveillance programs shouldconsist of a pre-placement evaluation to determine if workers or applicantshave any impairments (e.g., hyperreactive airways or bronchial asthma) thatwould limit their fitness for work in jobs with potential for exposure toamines. A clinical baseline can be established at the time of this evaluation.

Periodic medical evaluations can have significant value in theearly detection of disease and in providing an opportunity for healthcounseling.

Medical personnel conducting medical surveillance of individualspotentially exposed to polyurethane amine catalysts should consider thefollowing:

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

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

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

Broadly speaking, exposure to amines, as characterised by aminecatalysts, may cause effects similar to those caused by exposure to ammonia. Assuch, amines should be considered potentially injurious to any tissue that isdirectly contacted.

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

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

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

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

Alliance for Polyurethanes Industry

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

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

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility

Fire Fighting

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

5.3. Advice for firefighters

- Alert Fire Brigade and tell them location and nature of hazard.
- Wear full body protective clothing with breathing apparatus.
- Prevent, by any means available, spillage from entering drains or water course
- $\,\blacktriangleright\,$ Use fire fighting procedures suitable for surrounding area.
- Do not approach containers suspected to be hot.
 - Cool fire exposed containers with water spray from a protected location.
 - If safe to do so, remove containers from path of fire.
 - ▶ Equipment should be thoroughly decontaminated after use.

For amines:

- For firefighting, cleaning up large spills, and other emergency operations, workers must wear a self-contained breathing apparatus with full face-piece, operated in a pressure-demand mode.
- Airline and air purifying respirators should not be worn for firefighting or other emergency or upset conditions.
- Respirators should be used in conjunction with a respiratory protection program, which would include suitable fit testing and medical evaluation of the user.

Fire/Explosion Hazard

- Slight fire hazard when exposed to heat or flame.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO).
- ► Mists containing combustible materials may be explosive.

Combustion products include; carbon dioxide (CO2) nitrogen oxides (NOx) sulfur oxides (SOx) other pyrolysis products typical of burning organic materialMay emit corrosive fumes.

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

- Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.
- Check regularly for spills and leaks.
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact with the substance, by using protective equipment.
- Contain and absorb spill with sand, earth, inert material or vermiculite.
- ▶ Wipe up.
- Place in a suitable, labelled container for waste disposal

Minor Spills

- ▶ If possible (i.e., without risk of contact or exposure), stop the leak.
 - Contain the spilled material by diking, then neutralize.
- Next, absorb the neutralized product with clay, sawdust, vermiculite, or other inert absorbent and shovel into containers.
- Store the containers outdoors
- ▶ Brooms and mops should be disposed of, along with any remaining absorbent, in accordance with all applicable federal, state, and local regulations and requirements.
- Decontamination of floors and other hard surfaces after the spilled material has been removed may be accomplished by using a 5% solution of acetic acid, followed by very hot water
- Dispose of the material in full accordance with all federal, state, and local laws and regulations governing the disposal of chemical wastes.
- ▶ Waste materials from an amine catalyst spill or leak may be "hazardous wastes" that are regulated under various laws.

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Chemical Class: bases

For release onto land; recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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LAND SPILL - SMALL

cross-linked polymer - particulate	1	shovel	shovel	R,W,SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R, I, P
foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT
expanded minerals - particulate	3	shovel	shovel	R, I, W, P, DGC
foamed glass - particulate	4	shovel	shovel	R, W, P, DGC,

LAND SPILL - MEDIUM

cross-linked polymer -particulate	1	blower	skiploader	R,W, SS
sorbent clay - particulate	2	blower	skiploader	R, I, P
expanded mineral - particulate	3	blower	skiploader	R, I,W, P, DGC
cross-linked polymer - pillow	3	throw	skiploader	R, DGC, RT
foamed glass - particulate	4	blower	skiploader	R, W, P, DGC
foamed glass - pillow	4	throw	skiploader	R, P, DGC., RT

Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT:Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

Major Spills W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

- ▶ Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear full body protective clothing with breathing apparatus.
- Prevent, by any means available, spillage from entering drains or water course.
- Consider evacuation (or protect in place).
- Stop leak if safe to do so.
- Contain spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Neutralise/decontaminate residue (see Section 13 for specific agent).
- Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains
- After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using
- If contamination of drains or waterways occurs, advise emergency services.

- First remove all ignition sources from the spill area
- Have firefighting equipment nearby, and have firefighting personnel fully trained in the proper use of the equipment and in the procedures used in fighting a chemical fire.
- Spills and leaks of polyurethane amine catalysts should be contained by diking, if necessary, and cleaned up only by properly trained and equipped personnel. All others should promptly leave the contaminated area and stay upwind
- Protective equipment for cleanup crews should include appropriate respiratory protective devices and impervious clothing, footwear, and gloves.
- All work areas should be equipped with safety showers and eyewash fountains in good working order.
- Any material spilled or splashed onto the skin should be quickly washed off.
- Figure 5. Spills or releases may need to be reported to federal, state, and local authorities. This reporting contingency should be a part of a site's emergency response
- Protective equipment should be used during emergency situations whenever there is a likelihood of exposure to liquid amines or to excessive concentrations of amine vapor. "Emergency" may be defined as any occurrence, such as, but not limited to, equipment failure, rupture of containers, or failure of control equipment that results in an uncontrolled release of amine liquid or vapor.
- Emergency protective equipment should include:
- Self-contained breathing apparatus, with full face-piece, operated in positive pressure or pressure-demand mode.
- ▶ Long-sleeve coveralls or impervious full body suit
- Head protection, such as a hood, made of material(s) providing protection against amine catalysts
- Firefighting personnel and other on-site Emergency Responders should be fully trained in Chemical Emergency Procedures. However back-up from local authorities should be sought

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE

7.1. Precautions for safe handling

- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Safe handling Use in a well-ventilated area.
 - WARNING: To avoid violent reaction, ALWAYS add material to water and NEVER water to material

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- Avoid smoking, naked lights or ignition sources.
- Avoid contact with incompatible materials
- When handling, DO NOT eat, drink or smoke
- Keep containers securely sealed when not in use.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately. Launder contaminated clothing before re-use.
- Use good occupational work practice.
- Observe manufacturer's storage and handling recommendations contained within this SDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
- DO NOT allow clothing wet with material to stay in contact with skin

Fire and explosion protection

Other information

See section 5

▶ Store in original containers.

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

7.2. Conditions for safe storage, including any incompatibilities

- ► DO NOT use aluminium, galvanised or tin-plated containers
- Lined metal can, lined metal pail/ can.
- Plastic pail.
- Polyliner drum.
- Packing as recommended by manufacturer.
- ▶ Check all containers are clearly labelled and free from leaks

For low viscosity materials

- ▶ Drums and jerricans must be of the non-removable head type.
 - ▶ Where a can is to be used as an inner package, the can must have a screwed enclosure.

For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):

- Removable head packaging;
- ▶ Cans with friction closures and
- ► low pressure tubes and cartridges

may be used.

Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.

Storage incompatibility

Suitable container

Barium sulfate (barytes)

- reacts violently with dimethyl sulfoxide, sodium acetylide, finely divided carbon, aluminium, magnesium, zirconium, and possibly other active metals, especially at elevated temperatures
- ▶ is incompatible with potassium, phosphorus (ignites when primed with nitrate-calcium silicide)
- Avoid contact with copper, aluminium and their alloys.
- ► Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.
- ▶ Avoid reaction with oxidising agents

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	barium sulfate	Barium sulphate inhalable dust / Barium sulphate respirable dust	10 mg/m3 / 4 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
barium sulfate	Barium sulfate	30 mg/m3	330 mg/m3	2000 mg/m3
C18 fatty acid dimers/ tetraethylenepentamine polyamides	C-18 Unsaturated fatty acid, dimers, reaction products with polyethylenepolyamines; (Versamid 140 polyamide resin; Versamid 125)	30 mg/m3	330 mg/m3	2000 mg/m3
triethylenetetramine	Triethylenetetramine	3 ppm	5.7 ppm	83 ppm

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N-aminoethylpiperazine	Aminoethylpiperazine, N-		0.45 mg/m3	4.9 mg/m3	420 mg/m3
Ingredient	Original IDLH	Revised IDLH			
acrylonitrile/ butadiene copolymer amine terminated	Not Available	Not Available			
barium sulfate	Not Available	Not Available			
C18 fatty acid dimers/ tetraethylenepentamine polyamides	Not Available	Not Available			
tall oil/ triethylenetetramine polyamides	Not Available	Not Available			
triethylenetetramine	Not Available	Not Available			
N-aminoethylpiperazine	Not Available	Not Available			

MATERIAL DATA

For 1,3-butadiene:

Odour Threshold Value: 0.45 ppm (detection), 1.1 ppm (recognition)

Exposure at or below the TLV-TWA is thought to provide significant protection for workers against systemic toxicity including cancer.

US rubber workers reached an accord in 1996 to limit exposure to 1 ppm with a 15-minute, short-term limit of 5 ppm. This TLV-TWA is currently under review in light of a report of animal carcinogenicity at 6.25 ppm.

Odour Safety Factor(OSF)

OSF=1.3 ('1,3-BUTADIENE')

Barium sulfate has been identified as a nontoxic dust. However high dust levels have caused benign pneumoconiosis (baritosis). The TLV-TWA is thought to be protective against the risk of eye, nose and upper respiratory tract irritation and perhaps, pneumoconiosis.

Polyamide hardeners have much reduced volatility, toxicity and are much less irritating to the skin and eyes than amine hardeners. However commercial polyamides may contain a percentage of residual unreacted amine and all unnecessary contact should be avoided. for barium compounds:

The recommended TLV-TWA is based on satisfactory results achieved while employing an internal limit for barium nitrate at a national laboratory. It is not known what degree of added safety this limit incorporates.

8.2. Exposure controls

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

The basic types of engineering controls are:

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

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

Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed storage areas. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

8.2.1. Appropriate engineering controls

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion)	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

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

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

8.2.2. Personal protection











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where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.

Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient

- Chemical googles, whenever there is a danger of the material coming in contact with the eyes; googles must be properly fitted.
- Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.
- Alternatively a gas mask may replace splash goggles and face shields.
- ► 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]

Eye and face protection

For amines:

- SPECIAL PRECAUTION: ▶ Because amines are alkaline materials that can cause rapid and severe tissue damage, wearing of contact lenses while working with amines is strongly discouraged. Wearing such lenses can prolong contact of the eye tissue with the amine, thereby causing more severe damage
- Appropriate eye protection should be worn whenever amines are handled or whenever there is any possibility of direct contact with liquid products, vapors, or aerosol mists.

CAUTION:

- ▶ Ordinary safety glasses or face-shields will not prevent eye irritation from high concentrations of vapour.
- In operations where positive-pressure, air-supplied breathing apparatus is not required, all persons handling liquid amine catalysts or other polyurethane components in open containers should wear chemical workers safety goggles.
- ► Eyewash fountains should be installed, and kept in good working order, wherever amines are used.

Skin protection

See Hand protection below

- ▶ Elbow length PVC gloves
- When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.

NOTE:

- Fig. The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- ► Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

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

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

Hands/feet protection

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

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

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.

- ▶ When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butatoluene rubber), boots and aprons.
- DO NOT use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber or polyethylene gloves (which absorb the resin).
- ▶ DO NOT use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to

For amines:

- ▶ Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly.
- Application of a non-perfumed moisturiser is recommended
- Where there is a possibility of exposure to liquid amines skin protection should include: rubber gloves, (neoprene, nitrile, or butyl).
- DO NOT USE latex

Body protection

See Other protection below

Other protection

Overalls.

- PVC Apron
- PVC protective suit may be required if exposure severe
- ▶ Ensure there is ready access to a safety shower.

Thermal hazards

Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

Forsberg Clothing Performance Index'.

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

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Material	СРІ
BUTYL	Α
NEOPRENE	С
NITRILE	С
PE/EVAL/PE	С
VITON	С

Respiratory protection

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

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

Degree of protection varies with both face-piece and Class offilter; 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	AEK-AUS P2	-	AEK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AEK-AUS / Class 1 P2	-
up to 100 x ES	-	AEK-2 P2	AEK-PAPR-2 P2 ^

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* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

^ - Full-face

 $A(All\ classes) = Organic\ vapours,\ B\ AUS\ or\ B1 = Acid\ gasses,\ B2 = Acid\ gas\ or\ hydrogen\ cyanide(HCN),\ E = Sulfur\ dioxide(SO2),\ G = Agricultural\ chemicals,\ K = Ammonia(NH3),\ Hg = Mercury,\ NO = Oxides\ of\ nitrogen,\ MB = Methyl\ bromide,\ AX = Low\ boiling\ pointorganic\ compounds(below\ 65\ degC)$

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

9.1. Information on basic physical and chemical properties

Appearance	amber		
Physical state	Liquid	Relative density (Water = 1)	1.18
Odour	AmmoniaLike	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	>20.5
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	>122	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	<0.001	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2.Chemical stability	Unstable in the presence of incompatible materials. Product is considered stable. Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 TOXICOLOGICAL INFORMATION

11.1. Information on toxicological effects

Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales. The material is not thought to produce adverse health effects following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.

Inhaled

Inhalation of amine vapours may cause irritation of the mucous membranes of the nose and throat and lung irritation with respiratory distress and cough. Single exposures to near lethal concentrations and repeated exposures to sublethal concentrations produces tracheitis, bronchitis, pneumonitis and pulmonary oedema. Aliphatic and alicyclic amines are generally well absorbed from the respiratory tract. Systemic effects include headache, nausea, faintness and anxiety. These effects are thought to be transient and are probably related to the pharmacodynamic action of the amines. Histamine release by aliphatic amines may produce bronchoconstriction and wheezing.

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Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation.

Accidental ingestion of the material may be damaging to the health of the individual.

All cases of acute oral barium poisoning in adults exhibit gastrointestinal disturbances as the initial symptoms. These include gastric pain, vomiting, and diarrhea.

Ingestion of soluble barium compounds may result in ulceration of the mucous membranes of the gastrointestinal tract, tightness in the muscles of the face and neck, gastroenteritis, vomiting, diarrhoea, muscular tremors and paralysis, anxiety, weakness, laboured breathing, cardiac irregularity due to contractions of smooth, striated and cardiac muscles (often violent and painful), slow irregular pulse, hypertension, convulsions and respiratory failure.

The predominant musculoskeletal effect observed in cases of barium toxicity in humans is progressive muscle weakness, often leading to partial or total paralysis. In severe cases, the paralysis affects the respiratory system. The likely cause of the muscle weakness was the barium-induced hypokalaemia (low potassium levels) rather than a direct effect on muscles.

Numbness and tingling around the mouth and neck were sometimes among the first symptoms of barium toxicity in humans. Occasionally, these neurological symptoms extended to the extremities. Partial and complete paralysis occurred in severe cases, often accompanied by an absence of deep tendon reflexes Toxic effects on the kidneys have been observed in several adult cases of acute barium

poisoning. Effects include hemoglobin in the urine (which may be indicative of kidney damage), renal insufficiency, degeneration of the kidneys, and acute renal failure.

Studies in animals suggest that the kidney is a critical target of barium toxicity. An increase in relative kidney weight (kidney/brain weight ratio) was observed in male and female rats receiving a single gavage dose of 198 mg barium/kg/day as barium chloride in water.

Ingestion

Acute exposure to presumably high doses of barium carbonate, barium sulfate, or barium chloride can result in serious effects on heart rhythm. Barium adversely affects cardiac automaticity resulting in ventricular tachycardia and other disruptions of rhythm. Hypotension has also been reported in some cases. The likely cause of these effects was barium-induced hypokalaemia.

Several human studies have investigated a possible association between exposure to low levels of barium and alterations in blood pressure and cardiac rhythms. In a small-scale (11 subjects) study of individuals exposed to 0.1 or 0.2 mg barium/kg/day as barium chloride in drinking water for 4 weeks, no significant alterations in blood pressure or ECG readings were found. There was no significant alteration in blood pressure measurements or alterations in hypertension, heart disease, or stroke among residents of two communities with elevated (0.2 mg barium/kg/day) or low (0.003 mg barium/kg/day) levels of barium in drinking water. Significantly higher mortality rates for cardiovascular disease and heart disease (arteriosclerosis) were found in the elevated barium communities (0.06-0.3 mg barium/kg/day) than in the low barium communities (0.006 mg barium/kg/day). The largest difference between the groups was in individuals 65 years of age and older. These results should be interpreted cautiously because the study did not control for a number of potential confounding variables such as the use of water softeners, which would reduce the amount of barium and increase sodium levels, duration of exposure, or actual barium interpreted.

Several animal studies have examined potential cardiovascular end points following acute-, intermediate-, or chronic-duration exposures. Significant increases in systolic blood pressure were observed in rats exposed to 8.6 or 11 mg barium/kg/day for 1 or 4 months, respectively; no effect levels were 1.0 and 1.2 mg barium/kg/day. When the duration of exposure was longer (8-16 months), the LOAEL for increased blood pressure was 0.80 mg barium/kg/day and the NOAEL was 0.17 mg barium/kg/day. Depressed rates of cardiac contraction and cardiac conductivity and decreased cardiac ATP levels were observed in another group of rats exposed to 7.2 mg barium/kg/day. In contrast to the findings in this study, a second study could find no significant alterations in blood pressure were observed in rats exposed to up to 150 mg barium/kg/day in drinking water for 16 weeks; it should be noted that the second was conducted in uninephrectomized rats or Dahl salt-sensitive and salt-resistant rats. NTP (1994) also found no significant alterations in blood pressure, heart rate, or ECG readings in rats exposed to 180 mg barium/kg/day for 45 or 90 days. The low metal diet used in the first study may have influenced the study outcome.

When evaluating the health effects of barium compounds, it is important to keep in mind that different barium compounds have different solubilities in water and body fluids and therefore serve as variable sources of the Ba2+ ion. The Ba2+ ion and the soluble compounds of barium (notably chloride, nitrate, and hydroxide) are generally highly toxic to humans and experimental animals. The insoluble barium compounds (notably sulfate) are inefficient sources of the Ba2+ ion and therefore are generally nontoxic. Although barium carbonate is insoluble in water, barium ions would be released from ingested barium carbonate in the acid milieu of the stomach.

Aliphatic and alicyclic amines are generally well absorbed from the gut. Corrosive action may cause tissue damage throughout the gastrointestinal tract. Detoxification is thought to occur in the liver, kidney and intestinal mucosa with the enzymes, monoamine oxidase and diamine oxidase (histaminase) having a significant role.

The material can produce severe chemical burns following direct contact with the skin.

Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds. lesions or abrasions.

Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep.

Skin Contact

Volatile amine vapours produce primary skin irritation and dermatitis. Direct local contact, with the lower molecular weight liquids, may produce skin burns. Percutaneous absorption of simple aliphatic amines is known to produce lethal effects often the same as that for oral administration. Cutaneous sensitisation has been recorded chiefly due to ethyleneamines. Histamine release following exposure to many aliphatic amines may result in 'triple response' (white vasoconstriction, red flare and wheal) in human skin.

Open cuts, abraded or irritated skin should not be exposed to this material.

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.

Eye

Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight. Vapours of volatile amines cause eye irritation with lachrymation, conjunctivitis and minor transient corneal oedema which results in 'halos' around lights (glaucopsia, 'blue haze', or 'blue-grey haze'). Vision may become misty and halos may appear several hours after workers are exposed to the substance This effect generally disappears spontaneously within a few hours of the end of exposure, and does not produce physiological after-effects. However oedema of the corneal epithelium, which is primarily responsible for vision disturbances, may take more than one or more days to clear, depending on the severity of exposure. Photophobia and discomfort from the roughness of the corneal surface also may occur after greater exposures.

Although no detriment to the eye occurs as such, glaucopsia predisposes an affected individual to physical accidents and reduces the ability to undertake skilled tasks such as driving a vehicle.

Direct local contact with the liquid may produce eye damage which may be permanent in the case of the lower molecular weight species.

Chronic

Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.

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

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.

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Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.

Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.

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.

Workers exposed to barium compounds have been reported to show an increased incidence of hypertension, irritation of the respiratory system, and damage to the spleen, liver and bone marrow. Long term exposure to some barium compounds (especially inorganic species) may produce a condition known as baritosis, a form of benign pneumoconiosis. X-ray may show this when no other abnormal signs are present.

Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. Barium sulfate produces noncollagenous pneumoconiosis identified by minimal stromal reaction, consisting mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible. Miners of ores containing barium sulfate do not show symptoms, abnormal physical signs, an incapacity to work, diminished lung function, an increased likelihood of developing pulmonary or other bronchial infections or other thoracic disease despite the fact that particulate matter may have been retained in the lungs for many years.

No changes in mortality were observed in rats chronically exposed to doses as high as 60 mg barium/kg/day as barium chloride in the drinking water. An increase in mortality, attributable to nephropathy, was observed in mice chronically exposed to 160 mg barium/kg/day as barium chloride in drinking water; the number of deaths was similar to controls in mice exposed to 75 mg barium/kg/day. In male mice exposed to 0.95 mg barium/kg/day as barium acetate in drinking water, a significant decrease in longevity (defined as average lifespan of the last five surviving animals) was observed; however, no significant differences in mean lifespan were observed. Similarly, lifespan was not significantly altered in female mice exposed to 0.95 mg barium/kg/day or male or female rats exposed to 0.7 mg barium/kg/day as barium acetate in drinking water.

9200 Structural Epoxy	TOXICITY	IRRI	TATION		
Adhesive (Part B)	Not Available	Not A	Available		
	TOXICITY	CITY IRRITATION			
acrylonitrile/ butadiene copolymer amine	dermal (rat) LD50: >3000 mg/kg**[2]		Eye (ra	abbit): irritant *	
terminated	Inhalation (rat) LC50: 5.61 mg/L/4h *[2]		Skin: ir	irritant, Draize Score 3.6*	
	Oral (rat) LD50: >15380 mg/kg** ^[2]				
	TOXICITY				IRRITATION
barium sulfate	dermal (rat) LD50: >2000 mg/kg ^[1]				Not Available
	Oral (rat) LD50: 307000 mg/kg ^[1]				
C18 fatty acid dimers/	TOXICITY				IRRITATION
tetraethylenepentamine polyamides	dermal (rat) LD50: >5000 mg/kg*d ^[2]			* [Epoxylite]	
polyantides	Oral (rabbit) LD50: 800 mg/kg**] ^[2]			Nil reported	
	TOXICITY			RRITATION	
tall oil/ triethylenetetramine polyamides	Oral (rat) LD50: >5000 mg/kg*d ^[2]			' [Gibson-Homans	s Co.]
polyantides				**[Devoe]	
				Nil reported	
	TOXICITY	IDD	ITATION		
	Dermal (rabbit) LD50: 805 mg/kgE ^[2]		Eye (rabbit):20 mg/24 h - moderate		erate
triethylenetetramine	Oral (rat) LD50: 2500 mg/kgE ^[2]	Eye (rabbit); 49 mg - SEVERE			
		Skin (rabbit): 490 mg open SEVERE			
		Skin	rabbit):	5 mg/24 SEVERE	Ξ
	TOXICITY	IRF	IRRITATION		
N-aminoethylpiperazine	Dermal (rabbit) LD50: 866 mg/kg ^[1]	Еує	e (rabbit): 2	20 mg/24h - mod	
14-aminocutyipiperazitie	Oral (rat) LD50: >1000 mg/kg ^[1]	Skii	n (rabbit):	0.1 mg/24h - mild	d
		Skii	n (rabbit):	5 mg/24h - SEVE	ERE

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

9200 Structural Epoxy Adhesive (Part B) 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

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involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

For Fatty Nitrogen Derived (FND)Amides (including several high molecular weight alkyl amino acid amides)

The chemicals in the FattyNitrogen Derived (FND) Amides of surfactants are similar to the class ingeneral as to physical/chemical properties, environmental fate and toxicity. Humanexposure to these chemicals is substantially documented.

Some typical applications of FNDAmides are:

masonry cement additive; curingagent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives forpolymers.

The safety of the FND Amides tohumans is recognised by the U.S. FDA, which has approved stearamide, oleamideand/or erucamide for adhesives; coatings for articles in food contact; coatingsfor polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of preparedfoods; release agents in manufacture of food packaging materials, food contactsurface of paper and paperboard; cellophane in food packaging; closure sealinggaskets; and release agents in polymeric resins and petroleum wax. The loworder of toxicity indicates that the use of FND Amides does not pose asignificant hazard to human health.

The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, oraddition of an amino group in the chain would not be expected to have an impacton the toxicity profile. This conclusion is supported by a number of studies inthe FND family of chemicals (amines, cationics, and amides as separatecategories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of theselong-chain substituted chemicals

The Fatty nitrogen-derived amides(FND amides) comprise four categories:

- Subcategory I: Substituted Amides
- Subcategory II: Fatty Acid Reaction Products with Amino Compounds(Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicalsas major components)
- Subcategory III: Imidazole Derivatives

developmental effectsfor the FND group as a whole.

Subcategory IV: FND Amphoterics

Acute Toxicity: The lowacute oral toxicity of the FND Amides is well established across allSubcategories by the available data. The limited acute toxicity of thesechemicals is also confirmed by four acute dermal and two acute inhalationstudies

Repeated Dose and ReproductiveToxicity: Two subchronic toxicity studies demonstrating low toxicity areavailable for Subcategory I chemicals. In addition, a 5-day repeated dose studyfor a third chemical confirmed the minimal toxicity of these chemicals. Sincethe Subcategory I chemicals are major components of many Subcategory Ilchemicals, and based on the low repeat-dose toxicity of the amino compounds(e.g. diethanolamine, triethanolamine) used for producing the Subcategory Ilderivatives, the Subcategory I repeat-dose toxicity studies adequately supportSubcategory II.

Two subchronic toxicity studiesin Subcategory III confirmed the low order of repeat dose toxicity for the FNDAmides Imidazole derivatives. For Subcategory IV, two subchronic toxicitystudies for one of the chemicals indicated a low

order of repeat-dose toxicity forthe FND amphoteric salts similar to that seen in the other categories.

Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonellareverse mutation assay exist for all of the subcategories.

Developmental Toxicity: Adevelopmental toxicity study in Subcategory I and in Subcategory IV and a thirdstudy for a chemical in Subcategory III are available. The studies indicatethese chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similarchemicals. As above for repeat-dose toxicity, the data for Subcategory I areadequate to support Subcategory II. In evaluating potential toxicityof the FND Amides chemicals, it is also useful to review the available data forthe related FND Cationic and FND Amines Category chemicals. Acute oral toxicitystudies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparentorgan specific toxicity. Similarly, repeated dose toxicity studies(approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicitystudies (in vitro bacterial and mammalian cells as well as in vivo studies)indicated no mutagenic activity among more than 30 chemicals tested. Forreproductive evaluations, 14 studies evaluated reproductive

endpoints and/orreproductive organs for 11 chemicals, and 15 studies evaluated developmentaltoxicity for 13 chemicals indicating no reproductive or

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.

While it is difficult to generalise about the full range ofpotential health effects posed by exposure to the many different aminecompounds, characterised by those used in the manufacture of polyurethane andpolyisocyanurate foams, it is agreed that overexposure to the majority of thesematerials may cause adverse health effects.

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

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

Inhalation:

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

Products with higher vapour pressures have a greater potential forhigher airborne concentrations. This increases the probability of workerexposure. Higher concentrations of certain amines can produce severe respiratoryirritation, characterised by nasal discharge, coughing, difficulty inbreathing, and chest pains.

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

While most polyurethane amine catalysts are not sensitisers, somecertain individuals may also become sensitized to amines and may experiencerespiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronicor repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronicoverexposure may lead to permanent pulmonary injury, including a reduction inlung function, breathlessness, chronic bronchitis, and immunologic lungdisease.

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

Skin contact with amine catalysts poses a number of concerns.Direct skin contact can cause moderate to severe irritation and injury-i.e.,from simple redness and swelling to painful blistering, ulceration, andchemical burns. Repeated or prolonged exposure may also result in severecumulative dermatitis. Skin contact with some amines may result in allergicsensitisation. Sensitised persons should avoid all contact with aminecatalysts. Systemic effects resulting from the absorption of the amines throughskin exposure may include headaches, nausea, faintness, anxiety, decrease inblood pressure, reddening

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of the skin, hives, and facial swelling. Thesesymptoms may be related to the pharmacological action of the amines, and they are usually transient.

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

Direct contact with the liquid amine may cause severe irritationand tissue injury, and the "burning" may lead to blindness. (Contact with solidproducts may result in mechanical irritation, pain, and corneal injury.)

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

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

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

The oral toxicity of amine catalysts varies from moderately tovery toxic.

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

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

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

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

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

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

ACRYLONITRILE/ BUTADIENE **COPOLYMER AMINE TERMINATED**

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

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

The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

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

BARIUM SULFATE

C18 FATTY ACID DIMERS/

POLYAMIDES

TETRAETHYLENEPENTAMINE

No significant acute toxicological data identified in literature search.

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. For Fatty Nitrogen Derived (FND)Amides (including several high molecular weight alkyl amino acid amides)

The chemicals in the FattyNitrogen Derived (FND) Amides of surfactants are similar to the class ingeneral as to physical/chemical properties, environmental fate and toxicity. Humanexposure to these chemicals is substantially documented. Some typical applications of FNDAmides are:

masonry cement additive; curingagent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives forpolymers.

The safety of the FND Amides tohumans is recognised by the U.S. FDA, which has approved stearamide, oleamideand/or erucamide for adhesives; coatings for articles in food contact; coatingsfor polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of preparedfoods; release agents in manufacture of food packaging materials, food contactsurface of paper and paperboard; cellophane in food packaging; closure sealinggaskets; and release agents in polymeric resins and petroleum wax. The loworder of toxicity indicates that the use of FND Amides does not pose asignificant hazard to human

The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, oraddition of an amino group in the chain would not be expected to have an impacton the toxicity profile. This conclusion is supported by a number of studies inthe FND family of chemicals (amines, cationics, and

toxicity of theselong-chain substituted chemicals The Fatty nitrogen-derived amides(FND amides) comprise four categories:

- Subcategory I: Substituted Amides
 - Subcategory II: Fatty Acid Reaction Products with Amino Compounds(Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicalsas major components)

amides as separatecategories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited

- Subcategory III: Imidazole Derivatives
- Subcategory IV: FND Amphoterics

Acute Toxicity: The lowacute oral toxicity of the FND Amides is well established across allSubcategories by the available data. The limited acute toxicity of thesechemicals is also confirmed by four acute dermal and two acute inhalationstudies

Repeated Dose and ReproductiveToxicity: Two subchronic toxicity studies demonstrating low toxicity areavailable for Subcategory I chemicals. In addition, a 5-day repeated dose studyfor a third chemical confirmed the minimal toxicity of these chemicals. Sincethe Subcategory I chemicals are major components of many Subcategory Ilchemicals, and based on the low repeat-dose toxicity of the amino compounds(e.g. diethanolamine, triethanolamine) used for producing the Subcategory Ilderivatives, the Subcategory I repeat-dose toxicity studies adequately supportSubcategory II.

Two subchronic toxicity studiesin Subcategory III confirmed the low order of repeat dose toxicity for the FNDAmides Imidazole derivatives. For Subcategory IV, two subchronic toxicitystudies for one of the chemicals indicated a low

Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonellareverse mutation assay exist for all of the subcategories.

order of repeat-dose toxicity forthe FND amphoteric salts similar to that seen in the other categories.

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Developmental Toxicity: Adevelopmental toxicity study in Subcategory I and in Subcategory IV and a thirdstudy for a chemical in Subcategory III are available. The studies indicatethese chemicals are not developmental toxicants, as expected based on theirstructures, molecular weights, physical properties and knowledge of similarchemicals. As above for repeat-dose toxicity, the data for Subcategory I areadequate to support Subcategory II. In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data forthe related FND Cationic and FND Amines Category chemicals. Acute oral toxicitystudies (approximately 80 studies for 40 chemicals in the three categories)provide LD50 values from approximately 400 to 10,000 mg/kg with no apparentorgan specific toxicity. Similarly, repeated dose toxicity studies(approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicitystudies (*in vitro* bacterial and mammalian cells as well as *in vivo* studies)indicated no mutagenic activity among more than 30 chemicals tested. Forreproductive evaluations, 14 studies evaluated reproductive endpoints and/orreproductive organs for 11 chemicals, and 15 studies evaluated developmentaltoxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.

The following information refers to contact allergens as a group and may not be specific to this product.

**[Valspar]

Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds.

Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines.

In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper

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

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

For alkyl polyamines:

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

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

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

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

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

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

TETA is of moderate acute toxicity: LD50(oral, rat) > 2000mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure tosaturated vapour via inhalation was tolerated without impairment. Exposure to to aerosol leads to reversible irritations of the mucous membranes inthe respiratory tract. Following repeated oral dosing via drinking water only in mice butnot in rats at concentration of 3000 ppm there were signs of impairment. TheNOAEL is 600 ppm [92 mg/kg bw (oral, 90 days)]. Lifelong dermal application tomice (1.2 mg/mouse) did not result in tumour formation.

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

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

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

Experience with female patients suffering from Wilson's diseasedemonstrated that no miscarriages and no foetal abnormalities occur duringtreatment with TETA...

In rats, there are several studies concerning developmentaltoxicity. The oral treatment of rats with 75, 375 and 750 mg/kg resulted in noeffects on dams and fetuses, except slight increased fetal body weight After oral treatment of rats with 830 or 1670 mg/kg bw only in thehighest dose group increased foetal abnormalities in 27/44 fetus (69,2 %) wererecorded, when simultaneously the copper content of the feed was reduced. Coppersupplementation in the feed reduced significant the fetal abnormalities of thehighest dose group to 3/51 (6,5 % foetus. These findings suggest that thedevelopmental toxicity is produced as a secondary consequence of the chelatingproperties of TETA.

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

N-AMINOETHYLPIPERAZINE

TRIETHYLENETETRAMINE

The following information refers to contact allergens as a group and may not be specific to this product.

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Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor

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exposure to these compounds.

for piperazine:

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Exposure to piperazine and its salts has clearly been demonstrated to cause asthma in occupational settings. No NOAEL can be estimated for respiratory sensitisation (asthma).

Although the LD50 levels indicate a relatively low level of oral acute toxicity (LD50 1-5 g/kg bw), signs of neurotoxicity may appear in humans after exposure to lower doses. Based on exposure levels of up to 3.4 mg/kg/day piperazine base and a LOAEL of 110 mg/kg, there is no concern for acute toxicity In pigs, piperazine is readily absorbed from the gastrointestinal tract, and the major part of the resorbed compound is excreted as unchanged piperazine during the first 48 hours. The principal route of excretion of piperazine and its metabolites is via urine, with a minor fraction recovered from faeces (16%). In humans the kinetics of the uptake and excretion of piperazine and its metabolites with urine appear to be roughly similar to that in the pig, and the nature and extent of conversion to metabolites has not been determined.

Piperazine has demonstrated a low acute toxicity (LD50 = 1-5 g/kg bw) by the oral, dermal, and subcutaneous route of administration to rodents, whereas adequate inhalation toxicity data have not been found. However, there are findings of EEG (electroencephalogram) changes in 37% of 89 children administrated 90-130 mg/kg piperazine (two doses during one day), corroborated by a proposed GABA (gamma-aminobutyric acid) receptor agonism exerted by piperazine. Since clinical symptoms of neurotoxicity may occur after exposure to higher doses, a LOAEL of 110 mg/kg piperazine base for acute neurotoxicity in humans after acute exposure is proposed.

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

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

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

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

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

Acute Toxicity	0	Carcinogenicity	0
Skin Irritation/Corrosion	✓	Reproductivity	0
Serious Eye Damage/Irritation	0	STOT - Single Exposure	0
Respiratory or Skin sensitisation	~	STOT - Repeated Exposure	0
Mutagenicity	0	Aspiration Hazard	0

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

12.1. Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
barium sulfate	LC50	96	Fish	>3.5mg/L	2
barium sulfate	EC50	48	Crustacea	32mg/L	2
barium sulfate	EC50	72	Algae or other aquatic plants	>1.15mg/L	2
barium sulfate	EC50	72	Algae or other aquatic plants	>30.07mg/L	2
barium sulfate	NOEC	72	Algae or other aquatic plants	>=1.15mg/L	2

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C18 fatty acid dimers/ tetraethylenepentamine polyamides	LC50	96	Fish	7.07mg/L	2
C18 fatty acid dimers/ tetraethylenepentamine polyamides	EC50	24	Crustacea	7.07mg/L	2
C18 fatty acid dimers/ tetraethylenepentamine polyamides	EC50	48	Crustacea	5.18mg/L	2
C18 fatty acid dimers/ tetraethylenepentamine polyamides	EC50	72	Algae or other aquatic plants	4.11mg/L	2
C18 fatty acid dimers/ tetraethylenepentamine polyamides	NOEC	72	Algae or other aquatic plants	1.25mg/L	2
tall oil/ triethylenetetramine polyamides	LC50	96	Fish	7.07mg/L	2
tall oil/ triethylenetetramine polyamides	EC50	24	Crustacea	9.72mg/L	2
tall oil/ triethylenetetramine polyamides	EC50	48	Crustacea	7.07mg/L	2
tall oil/ triethylenetetramine polyamides	EC50	72	Algae or other aquatic plants	4.34mg/L	2
tall oil/ triethylenetetramine polyamides	NOEC	72	Algae or other aquatic plants	0.5mg/L	2
triethylenetetramine	EC50	48	Crustacea	31.1mg/L	1
triethylenetetramine	EC10	72	Algae or other aquatic plants	0.67mg/L	1
triethylenetetramine	EC50	72	Algae or other aquatic plants	2.5mg/L	1
triethylenetetramine	NOEC	72	Algae or other aquatic plants	<2.5mg/L	1
triethylenetetramine	LC50	96	Fish	180mg/L	1
N-aminoethylpiperazine	EC50	48	Crustacea	=32mg/L	1
N-aminoethylpiperazine	EC50	96	Algae or other aquatic plants	175.657mg/L	3
N-aminoethylpiperazine	LC50	96	Fish	>100mg/L	2
N-aminoethylpiperazine	EC50	48	Crustacea	32mg/L	2
N-aminoethylpiperazine	NOEC	48	Crustacea	10mg/L	2

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

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

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

For barium and its compounds::

Environmental fate:

Under natural conditions, barium is stable in the +2 valence stateand is found primarily in the form of inorganic complexes. Conditions such aspH, Eh (oxidation-reduction potential), cation exchange capacity, and thepresence of sulfate, carbonate, and metal oxides (e.g., oxides of aluminum,manganese, silicon, and titanium) will affect the partitioning of barium andits compounds in the environment. The major features of the biogeochemicalcycle of barium include wet and dry deposition to land and surface water,leaching from geological formations to groundwater, adsorption to soil andsediment particulates, and biomagnification in terrestrial and aquatic foodchains.

Barium is a highly reactive metal that occurs naturally only in acombined state. The element is released to environmental media by both natural processes and anthropogenic sources. The general population is exposed to barium through consumption of drinking water and foods, usually at low levels. Most barium released to the environment from industrial sources is in forms that do not become widely dispersed. In the atmosphere, barium is likely to be present in particulate form. Although chemical reactions may cause changes in speciation of barium in air, the mainmechanisms for the removal of barium compounds from the atmosphere are likely to be wet and dry deposition.

In aquatic media, barium is likely to precipitate out of solutionas an insoluble salt (i.e., as BaSO4 or BaCO3). Waterborne barium may alsoadsorb to suspended particulate matter through the formation of ion pairs withnatural anions such as bicarbonate or sulfate in the matter.

Precipitation of barium sulfate salts is accelerated when riversenter the ocean because of the high sulfate content (905 mg/L) in the ocean. It is estimated that only 0.006% of the total barium input into oceans fromfreshwater sources remains in solution. Sedimentation of suspended solidsremoves a large portion of the barium content from surface waters. There is evidence to suggest that the precipitation of barium from the surface of freshand marine waters occurs, in part, as the result of the barite crystalformation in microorganisms.

Barium in sediments is found largely in the form of barium sulfate(barite). Coarse silt sediment in a turbulent environment will often grind andcleave the barium sulfate from the sediment particles leaving a buildup ofdense barites. Estimated soil:water distribution coefficients (Kd) (i.e., theratio of the quantity of barium sorbed per gram of sorbent to the concentrationof barium remaining in solution at equilibrium) range from 200 to 2,800 forsediments and sandy loam soils. The uptake of barium by fish and marineorganisms is also an important removal mechanism. Barium levels in sea waterrange from 2 to 63 µg/L with a mean concentration of about 13 µg/L. Barium wasfound to bioconcentrate in marine plants by a factor of 400-4,000 times thelevel present in the water. Bioconcentration factors in marine animals, plankton, and brown algae of 100, 120, and 260, respectively, have been reported. In freshwater, a bioconcentration factor of 129 was estimated in fishwhere the barium in water was 0.07 mg/L.

Barium added to soils (e.g., from the land farming of wastedrilling muds) may either be taken up by vegetation or transported through soilwith precipitation. Relative to the amount of barium found in soils, little istypically bioconcentrated by plants. For example, a bioconcentration factor of 0.4 has been estimated for plants in a Virginia floodplain with a barium soilconcentration of 104.2 mg/kg. However, there are some plants, such as legumes, forage plants, Brazil nuts, and mushrooms that accumulate barium. Bioconcentration factors from 2 to 20 have been reported for tomatoes andsoybeans.

Barium is not very mobile in most soil systems, due to theformation of water-insoluble salts and an inability of the barium in to formsoluble complexes with fulvic and humic acids. The rate of transportation ofbarium in soil is dependent on the characteristics of the soil material. Soilproperties that influence the transportation of barium to groundwater arecation exchange capacity, calcium carbonate (CaCO3) content and pH. In soilwith a high cation exchange capacity (e.g., fine textured mineral soils orsoils with high organic matter content), barium mobility will be limited byadsorption. High CaCO3 content limits mobility by precipitation of the elementas BaCO3. Barium will also precipitate as barium sulfate in the presence ofsulfate ions. Barium is more mobile and is more likely to be leached from soilsin the presence of chloride due to the high solubility of barium chloride ascompared to other chemical forms of barium. Barium may become more mobile insoils under acid conditions as barium in water-insoluble salts, such as bariumsulfate and carbonate, becomes more soluble. Barium complexes with fatty acids(e.g., in acidic landfill leachate) will be much more mobile in the soil due to the lower charge of these complexes and subsequent reduction in adsorptioncapacity.

The alkali metal cyanides (and other metal cyanides) are verysoluble in water. As a result, they readily dissociate into their respectiveanions and cations when released into water. Depending on the pH of the water, the resulting cyanide ion may then form hydrogen cyanide or react with variousmetals in natural water. The proportion of hydrogen cyanide formed from solublecyanides increases as the water pH decreases. At pH <7, >99% of thecyanide ions in water are converted to hydrogen cyanide. As the pH increases, cyanide ions in the water may form complex metallocyanides in the

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presence of excess cyanides; however, if metals are prevalent, simple metal cyanides are formed. Volatilization is the dominant mechanism for the removal of freecvanide. At pH >9.2, most of the free cyanide should exist as HCN, avolatile form of cyanide. Wide variations in the rate of volatilization are expected since this process is affected by a number of parameters such astemperature, pH wind speed, and cyanide concentration. Volatilization of freecyanide from concentrated solutions is most effective under conditions of hightemperatures, high dissolved oxygen levels, and at increased concentrations of atmospheric carbon dioxide

Unlike water-soluble alkali metal cyanides, insoluble metalcyanides are not expected to degrade to hydrogen cyanide. Cyanide occurs mostcommonly as hydrogen cyanide in water, although it can also occur as thecyanide ion, alkali and alkaline earth metal cyanides (potassium cyanide, sodium cyanide, calcium cyanide), relatively stable metallocyanide complexes(ferricyanide complex [Fe(CN)6]-3), moderately stable metallocyanide complexes (complex nickel and copper cyanide), or easily decomposable metallocyanidecomplexes (zinc cyanide [Zan(CN)2], cadmium cyanide [Cd(CN)2]). Oxidation, hydrolysis, and photolysis (photodegradation) are the threepredominant chemical processes that may cause loss of simple cyanides inaquatic media.

Certain cyanides are oxidised to isocyanates by strong oxidisingagents; the isocyanates may be further hydrolysed to ammonia and carbondioxide. However, it has not yet been determined whether such oxidation and subsequent hydrolysis of isocyanate is a significant fate process in natural waters known to contain peroxy radicals. In water, hydrogen cyanide and cyanide ion exist in equilibrium with their relative concentrations primarilydependent on pH and temperature. At pH <8, >93% of the free cyanide inwater will exist as undissociated hydrogen cyanide. Hydrogen cyanide can behydrolysed to formamide, which is subsequently hydrolysed to ammonium andformate ions. However, the relatively slow rates of hydrolysis reported for hydrogen cyanide in acidic solution and of cyanides under alkalineconditions indicate that hydrolysis is not competitive with volatilisation andbiodegradation for removal of free cyanide from ambient waters. At pH<9.2, most of the free cyanide in solution should exist as hydrogen cyanide, a volatile cyanide form. On the basis of Henry's law constant and thevolatility characteristics associated with various ranges of Henry's lawconstant, volatilization is a significant and probably dominant fate processfor hydrogen cyanide in surface water. The most common alkali metal cyanides(e.g., sodium and potassium cyanide) may also be lost from surface waterprimarily through volatilization; whereas, the sparingly soluble metal cyanidessuch as copper (I) cyanide are removed from water predominantly bysedimentation and biodegradation. Because volatilisation is not an importantfate process for cyanide in groundwater, cyanide would be expected to persistfor considerably longer periods of time in underground aguifers than in surfacewater

The significance of photolysis in the fate of cyanides in waterhas not been fully investigated. Hydrogen cyanide and cyanide ions in agueoussolution have been found to be very resistant to photolysis by naturalsunlight, except under heterogeneous photocatalytic conditions. Photocatalyticoxidation may not be significant in natural waters, however, because of significant light reduction at increasingly greater depths. In clear water orat water surfaces, some metallocyanides, such as ferrocyanides and ferricyanides, may decompose to the cyanide ion by photodissociation and subsequentlyform hydrogen cyanide.

Biodegradation is an important transformation process for cyanidein natural surface waters, and is dependent on such factors as cyanideconcentrations, pH, temperature, availability of nutrients, and acclimation ofmicrobes. Although the cyanide ion is toxic to microorganisms at concentrations as low as 5-10 mg/L, acclimation increases tolerance to this compound. Mixed microorganisms in sewage sludge or activated sludge acclimated tocyanide also significantly biodegrade concentrations <=100 mg/L of mostsimple and complex cyanides. It is known that there is a naturalattentuation of the cyanide ion and thiocyanide concentrations in waste waters, for example those obtained gold mill tails, that is due the acclimation of indigenous microflora in the tailings. A number of microorganisms have been identified that are capable of uptake, conversion, sorption, and/or precipitation of the cyanide ion, cyanate, and thio cyanate, including species of the genera, Actinomyces, Alcaligenes, Arthrobacter, Bacillus, Micrococcus, Neisseria, Paracoccus, Pseudomonas, and Thiobacillus. Some of these species, forexample Pseudomonas, are capable of using the cyanide ion andthiocyanate as the sole source of carbon and nitrogen and therefore, are particularly effective at cyanide degradation. Infact, Pseudomonas is the basis of commercial applications fordegrading the cyanide ion to ammonia and carbonate in waste waters generated inmining operations that use the cyanide ion to leach gold and other preciousmetals for low-grade ores. Sulfur transferases such as rhodanese areinvolved in substitution reactions that result in the conversion of the cyanideion to the less toxic thiocyanate, whereas pyridoxal phosphate enzymes areinvolved in substitution/addition reactions that result in production ofnitrile derivatives of a-amino acids. These organic nitriles may then beultimately degraded via enzyme catalysed hydrolysis to either the correspondingamino acid and ammonia or the carboxylic acid and ammonia. The cyanidehydratase and cyanidase enzymes catalyse the hydrolysis of the cyanide ion toformamide or formic acid and ammonia, respectively In soil, cyanidepresent at low concentrations would biodegrade under aerobic conditions withthe initial formation of ammonia, which would be converted to nitrite and nitratein the presence of nitrifying bacteria. Under anaerobic conditions, thecyanides ion will denitrify to gaseous nitrogen. Upper limits of 200 and 2 ppm(mg/kg CN-), respectively, have been reported for uninhibited aerobic andanaerobic biodegradation of cyanide in soil; however, these limits have notbeen confirmed in other studies. Cyanide ions in soil are not involved inoxidationreduction reactions but may undergo complexation reactions with metalions in soil

Cyanides are sorbed by various natural media, including clays, biological solids and sediments Hydrogen cyanide and the alkali metalcyanides are not likely to be strongly sorbed onto sediments and suspendedsolids because of their high water solubilities. Soluble metal cyanides may showsomewhat stronger sorption than hydrogen cyanide, with the extent of sorptionincreasing with decreasing pH and increasing iron oxide, clay, and organicmaterial contents of sediment and suspended solids. However, sorption isprobably insignificant even for metal cyanides when compared to volatilisationand biodegradation. Cyanides are fairly mobile in soil. Mobility is lowest insoils with low pH and high concentrations of free iron oxides, positivelycharged particles, and clays (e.g., chlorite, kaolin, gibbsite), and highest insoils with high pH, high concentrations of free CaCO3 and negatively chargedparticles, and low clay content. Although cyanide has a low soil sorptioncapability, it is usually not detected in groundwater, probably because offixation by trace metals through complexation or transformation by soilmicroorganisms. In soils where cyanide levels are high enough to be toxic tomicroorganisms (i.e., landfills, spills), this compound may leach intogroundwater. Leaching of cyanide into a shallow aquifer has been demonstrated. Volatilisation of hydrogen cyanide would be a significant loss mechanism forcyanides from soil surfaces at a pH <9.2.

Most cyanide in the atmosphere exists almost entirely as hydrogencyanide gas, although small amounts of metal cyanides may be present asparticulate matter in the air. Hydrogen cyanide is very resistant to photolysisat wavelengths of normal sunlight. The most important reaction of hydrogencyanide in air is the reaction with photochemically-generated hydroxyl radicalsand subsequent rapid oxidation to carbon monoxide (CO) and nitric oxide (NO); photolysis and reaction with ozone are not important transformation processes, and reaction with singlet oxygen is not a significant transformationprocess except at stratospheric altitudes where singlet oxygen is present insignificant concentrations. The rate of hydroxyl radical reaction with hydrogencyanide in the atmosphere depends on the altitude, and the rate of the reactionis at least an order of magnitude faster at lower tropospheric altitudes (0-8km) than at upper tropospheric altitudes (10-12 km). Based on a reaction rateconstant of 3x10-14 cm3/(molecule-sec) at 25 °C and assuming an averagehydroxyl radical concentration of 5x105 molecules/cm3, the residence time forthe reaction of hydrogen cyanide vapor with hydroxyl radicals in the atmosphereis approximately 2 years

There is some evidence that certain metal cyanide complexes bioaccumulate in aquatic organisms. Fish from water with soluble silver and copper cyanide complexes were found to have metal cyanides in their tissues atconcentrations ranging up to 168 and 304 µg/g, respectively (wet or dry weightnot specified). It is difficult to evaluate the toxicologic significance ofbioaccumulation of metal cvanide complexes because these compounds are muchless toxic than soluble hydrogen cyanide, or potassium cyanide. There is no evidence of biomagnification of cyanides in the food chain. Accumulation of cyanide in food webs is not expected, considering the rapiddetoxification of cyanide by most species and the lethal effects of large dosesof cyanide Prevent, by any means available, spillage from entering drains or water courses.

DO NOT discharge into sewer or waterways

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
triethylenetetramine	LOW	LOW
N-aminoethylpiperazine	HIGH	HIGH

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
triethylenetetramine	LOW (LogKOW = -2.6464)
N-aminoethylpiperazine	LOW (LogKOW = -1.5677)

12.4. Mobility in soil

Ingredient	Mobility
triethylenetetramine	LOW (KOC = 309.9)
N-aminoethylpiperazine	LOW (KOC = 171.7)

12.5.Results of PBT and vPvB assessment

	P	В	Т
Relevant available data	Not Available	Not Available	Not Available
PBT Criteria fulfilled?	Not Available	Not Available	Not Available

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12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

- Containers may still present a chemical hazard/ danger when empty.
- ▶ Return to supplier for reuse/ recycling if possible.

Otherwise:

- ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.

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.

A Hierarchy of Controls seems to be common - the user should investigate:

- Reduction
- ▶ Reuse
- Recycling

Product / Packaging disposal

► 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.
- ► Treat and neutralise at an approved treatment plant.
- ► Treatment should involve: Neutralisation with suitable dilute acid followed by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material).
- ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

Waste treatment options

Not Available

Sewage disposal options

Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required



Limited Quantity: 9200-25ML, 9200-50ML

Land transport (ADR)

14.1.UN number	2735	
14.2.Packing group	II	
14.3.UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.C polyamides and triethylenetetramine)	O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains c18 fatty acid dimers/ tetraethylenepentamine
14.4.Environmental hazard	Not Applicable	
14.5. Transport hazard class(es)	Class 8 Subrisk Not Applicable	
14.6. Special precautions for user	Hazard identification (Kemler) 80 Classification code C7 Hazard Label 8 Special provisions 274 Limited quantity 1 L	

Air transport (ICAO-IATA / DGR)

14.1. UN number

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14.2. Packing group	П	
14.3. UN proper shipping name	Amines, liquid, corrosive, n.o.s. *; Polyamines, liquid, corrosive, triethylenetetramine)	n.o.s. * (contains c18 fatty acid dimers/ tetraethylenepentamine polyamides and
14.4. Environmental hazard	Not Applicable	
14.5. Transport hazard class(es)	ICAO/IATA Class 8 ICAO / IATA Subrisk Not Applicable ERG Code 8L	
	Special provisions Cargo Only Packing Instructions	A3A803 855
440.000.0101.000.0001.000.1000	Cargo Only Maximum Qty / Pack	30 L
14.6. Special precautions for user	Passenger and Cargo Packing Instructions	851
	Passenger and Cargo Maximum Qty / Pack	1L
	Passenger and Cargo Limited Quantity Packing Instructions	Y840
	Passenger and Cargo Limited Maximum Qty / Pack	0.5 L

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	2735
14.2. Packing group	П
14.3. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains c18 fatty acid dimers/ tetraethylenepentamine polyamides and triethylenetetramine)
14.4. Environmental hazard	Marine Pollutant
14.5. Transport hazard class(es)	IMDG Class 8 IMDG Subrisk Not Applicable
14.6. Special precautions for user	EMS Number F-A, S-B Special provisions 274 Limited Quantities 1 L

Inland waterways transport (ADN)

14.1. UN number	2735
14.2. Packing group	
14.3. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains c18 fatty acid dimers/ tetraethylenepentamine polyamides and triethylenetetramine)
4.4. Environmental hazard	Not Applicable
14.5. Transport hazard class(es)	8 Not Applicable
I.6. Special precautions for user	Classification code C7 Special provisions 274 Limited quantity 1 L Equipment required PP, EP Fire cones number 0

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED(68683-29-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

BARIUM SULFATE(7727-43-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Customs Inventory of Chemical Substances ECICS (English)

UK Workplace Exposure Limits (WELs)

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)

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C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES(68410-23-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

TALL OIL/ TRIETHYLENETETRAMINE POLYAMIDES(68082-29-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)

TRIETHYLENETETRAMINE(112-24-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles European Customs Inventory of Chemical Substances ECICS (English) European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

N-AMINOETHYLPIPERAZINE(140-31-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles European Customs Inventory of Chemical Substances ECICS (English) European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 91/689/EE1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

2

ECHA SUMMARY							
Ingredient	CAS number	Index No	Index No		ECHA Doss	ECHA Dossier	
acrylonitrile/ butadiene copolymer amine terminated	68683-29-4	Not Available	Not Available		Not Available	Not Available	
Harmonisation (C&L Inventory) Hazard Class and Category Code(s) Pictograms Signal Word Code(s) Hazard Statement Code(Hazard Statement Code(s)		
1	Aquatic Chronic 4 GHS07,		iHS07, Wng			H317, H315, H332, H319	
2	Aquatic Chronic 4, Skin Sens. 1, Skin Irrit. 2, Acute Tox. 4, Not Classified GHS07, Wng		07, Wng	H317, H315		H317, H315, H332, H319	
Harmonisation Code 1 = The mos	st prevalent classification. Harmonisation Code	2 = The most severe classification.					
In our diant	CAS number	Index No		FOUA Dansi			
Ingredient	CAS number	Index No		ECHA Dossi	ECHA DOSSIEI		
barium sulfate	7727-43-7	Not Available 01-21194912		01-21194912	274-35-XXXX		
Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)		Pictograms Signal Word Code(s)		Word	Hazar	d Statement Code(s)
1	Not Classified		Wng, 0	GHS08		H302, H315	H332, H371, H319, H373, H335,

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Not Classified, Acute Tox. 4, STOT SE 2, Eye Irrit. 2, STOT RE 2, STOT SE

Ingredient	CAS number	Index No	ECHA Dossier
C18 fatty acid dimers/ tetraethylenepentamine polyamides	68410-23-1	Not Available	Not Available

Wng. GHS08

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2, Eye Irrit. 2	GHS07, Wng	H315, H319
2	Skin Irrit. 2, Skin Sens. 1A, Eye Dam. 1, Aquatic Chronic 2, Eye Irrit. 2, Skin Sens. 1, Skin Corr. 1C, Aquatic Chronic 1, Aquatic Acute 1, Not Classified	GHS09, GHS05, Dgr, Wng, GHS06	H317, H318, H314, H335

Harmonisation Code 1 = The most prevalent classification, Harmonisation Code 2 = The most severe classification,

Ingredient	CAS number	Index No	ECHA Dossier	
tall oil/ triethylenetetramine polyamides	68082-29-1	Not Available	Not Available	
Harmonisation (C&L	H101		Pictograms Signal	Hazard Statement
Inventory)	Hazard Class and Category Code(s)		Word Code(s)	Code(s)

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier

H302, H332, H371, H319, H373, H335,

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triethylenetetramine	112-24-3	612-059-00-5		Not Available	
Harmonisation (C&L Inventory)	Hazard Class and Catedory Code(s)		Pictogra Code(s)	ams Signal Word	Hazard Statement Code(s)
1	Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3		GHS07,	GHS05, Dgr	H312, H314, H317
2	Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3, Eye Dam. 1, Acute Tox. 3, Resp. Sens. 1, STOT SE 3, Aquatic Chronic 2, Not Classified		GHS05, GHS08,	Dgr, GHS06, GHS09	H314, H317, H318, H302, H311, H334, H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
N-aminoethylpiperazine	140-31-8	612-105-00-4	01-2119471486-30-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3	GHS07, GHS05, Dgr	H302, H312, H314, H317
2	Acute Tox. 4, Acute Tox. 3, Skin Corr. 1B, Skin Sens. 1, Eye Dam. 1, Aquatic Chronic 3, Aquatic Chronic 1	GHS06, GHS05, Dgr, GHS09	H302, H311, H314, H317, H318, H305, H332

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Υ
Canada - NDSL	N (N-aminoethylpiperazine; tall oil/ triethylenetetramine polyamides; acrylonitrile/ butadiene copolymer amine terminated; C18 fatty acid dimers/ tetraethylenepentamine polyamides; barium sulfate; triethylenetetramine)
China - IECSC	Υ
Europe - EINEC / ELINCS / NLP	N (acrylonitrile/ butadiene copolymer amine terminated; C18 fatty acid dimers/ tetraethylenepentamine polyamides)
Japan - ENCS	N (tall oil/ triethylenetetramine polyamides; acrylonitrile/ butadiene copolymer amine terminated)
Korea - KECI	Υ
New Zealand - NZIoC	Υ
Philippines - PICCS	Υ
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

Full text Risk and Hazard codes

H290	May be corrosive to metals.
H302	Harmful if swallowed.
H305	May be harmful if swallowed and enters airways
H311	Toxic in contact with skin.
H312	Harmful in contact with skin.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H371	May cause damage to organs.
H373	May cause damage to organs.
H412	Harmful to aquatic life with long lasting effects.

Other information

Ingredients with multiple cas numbers

•	•	
Name		CAS No
barium sulfate		13462-86-7, 7727-43-7

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

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

www.chemwatch.net

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

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

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EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

Definitions and abbreviations

 $\begin{array}{ll} {\sf PC-TWA: Permissible Concentration-Time Weighted Average} \\ {\sf PC-STEL: Permissible Concentration-Short Term Exposure Limit} \end{array}$

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