

Kit Revision Date: 19/07/2021

8329TFF THERMALLY CONDUCTIVE EPOXY ADHESIVE KIT

MG Chemicals Multipart Product Kit

This product is a kit made up of multiple parts. Each part is an independently packaged chemical component and has independent hazard assessments.

Kit Content

Part	Product Name	Product Use
Α	8329TFF-A	Thermally conductive adhesive resin
В	8329TFF-B	Thermally conductive adhesive hardener

Safety Data Sheets for each part listed above follow this cover sheet.

Transportation Instruction

Before offering this product kit for transport, read Section 14 for <u>all</u> parts listed above.



8329TFF-A Thermally Conductive Epoxy Adhesive MG Chemicals UK Limited

Version No: A-2.00

Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: 18/07/2021 Revision Date: 18/07/2021 L.REACH.GB.EN

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

1.1. Product Identifier

Product name 8329TFF-A		
Synonyms SDS Code: 8329TFF-A; 8329TFF-25ML, 8329TFF-50ML UFI:25F0-D0HN-S00R-JFM2		
Other means of identification Thermally Conductive Epoxy Adhesive		

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

Relevant identified uses	Thermally conductive adhesive resin
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 Not Available		www.mgchemicals.com	
Email	sales@mgchemicals.com	Info@mgchemicals.com	

1.4. Emergency telephone number

Association / Organisation	Verisk 3E (Access code: 335388)	
Emergency telephone numbers	+(44) 20 35147487	
Other emergency telephone numbers	+(0) 800 680 0425	

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classified according to
GB-CLP Regulation, UK SI
2019/720 and UK SI 2020/1567
[1]

H411 - Chronic Aquatic Hazard Category 2, H400 - Acute Aquatic Hazard Category 1, H315 - Skin Corrosion/Irritation Category 2, H319 - Eye Irritation Category 2, H361 - Reproductive Toxicity Category 2, H317 - Skin Sensitizer Category 1

Legend:

1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567

2.2. Label elements

Hazard pictogram(s)







Signal word

Warning

Hazard statement(s)

H411	Toxic to aquatic life with long lasting effects.			
H400	Very toxic to aquatic life.			
H315	Causes skin irritation.			
H319	Causes serious eye irritation.			
H361	Suspected of damaging fertility or the unborn child.			
H317	May cause an allergic skin reaction.			

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.	
P280 Wear protective gloves, protective clothing, eye protection and face protection.		
P261 Avoid breathing mist/vapours/spray.		
P273 Avoid release to the environment.		
P264	Wash all exposed external body areas thoroughly after handling.	
P272	Contaminated work clothing should not be allowed out of the workplace.	

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.		
P302+P352	IF ON SKIN: Wash with plenty of water and soap.		
P305+P351+P338	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	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

Store locked up

Precautionary statement(s) Disposal

P501 Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

2.3. Other hazards

Inhalation and/or ingestion may produce health damage*.

Cumulative effects may result following exposure*.

May produce discomfort of the respiratory system*.

Limited evidence of a carcinogenic effect*.

Possible respiratory sensitizer*.

bisphenol F diglycidyl ether copolymer Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors

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	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
1.21645-51-2 2.244-492-7 3.Not Available 4.Not Available	54	alumina hydrate	EUH210 ^[1]	Not Available
1.9003-36-5 2.500-006-8 3.Not Available 4.Not Available	36	bisphenol F diglycidyl ether copolymer [e]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Germ cell mutagenicity Category 1B, Reproductive Toxicity Category 2, Chronic Aquatic Hazard Category 2; H315, H319, H317, H340, H361fd, H411, EUH205 [1]	Not Available
1.12767-90-7 2.235-804-2 3.Not Available 4.Not Available	7	zinc borate	Eye Irritation Category 2, Reproductive Toxicity Category 1B, Chronic Aquatic Hazard Category 1; H319, H360, H410 [1]	Not Available
1.17557-23-2 2.241-536-7 3.603-094-00-7 4.Not Available	3	neopentyl glycol diglycidyl ether	Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1; H315, H317 [2]	Not Available
Legend		•	assification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. [e] Substance identified as having endocrine disrupting properties	Classification drawn

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye 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. 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, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
Ingestion	 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. Seek medical advice.

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

Copper, magnesium, aluminium, antimony, iron, manganese, nickel, zinc (and their compounds) in welding, brazing, galvanising or smelting operations all give rise to thermally produced particulates of smaller dimension than may be produced if the metals are divided mechanically. Where insufficient ventilation or respiratory protection is available these particulates may produce 'metal fume fever' in workers from an acute or long term exposure.

- Onset occurs in 4-6 hours generally on the evening following exposure. Tolerance develops in workers but may be lost over the weekend. (Monday Morning Fever)
- Pulmonary function tests may indicate reduced lung volumes, small airway obstruction and decreased carbon monoxide diffusing capacity but these abnormalities resolve after
- ▶ Although mildly elevated urinary levels of heavy metal may occur they do not correlate with clinical effects.
- The general approach to treatment is recognition of the disease, supportive care and prevention of exposure.
- Seriously symptomatic patients should receive chest x-rays, have arterial blood gases determined and be observed for the development of tracheobronchitis and pulmonary

[Ellenhorn and Barceloux: Medical Toxicology]

Treat symptomatically.

- Manifestation of aluminium toxicity include hypercalcaemia, anaemia, Vitamin D refractory osteodystrophy and a progressive encephalopathy (mixed dysarthria-apraxia of speech, asterixis, tremulousness, myoclonus, dementia, focal seizures). Bone pain, pathological fractures and proximal myopathy can occur.
- > Symptoms usually develop insidiously over months to years (in chronic renal failure patients) unless dietary aluminium loads are excessive.
- Serum aluminium levels above 60 ug/ml indicate increased absorption. Potential toxicity occurs above 100 ug/ml and clinical symptoms are present when levels exceed 200 ug/ml.
- ▶ Deferoxamine has been used to treat dialysis encephalopathy and osteomalacia. CaNa2EDTA is less effective in chelating aluminium.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

5.1. Extinguishing media

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

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5.2. Special hazards arising fro	m 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	
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear full body protective clothing with breathing apparatus. Prevent, by any means available, spillage from entering drains or water course. Use 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.

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)
- Fire/Explosion Hazard May emit acrid smoke.
 - Mists containing combustible materials may be explosive.

Combustion products include:

carbon dioxide (CO2) aldehydes

metal oxides

other pyrolysis products typical of burning organic material.

When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.

Aluminium hydroxide is a flame retardant. At around 200 C, aluminium hydroxide (aluminium trihydrate) is decomposed to aluminium oxide (which forms a protective, non-flammable layer on the material surface) and water. The water (as steam) forms a layer of non-flammable gas near the material's surface, inhibiting flames. The reaction is endothermic (absorbs heat energy), thus cooling the material and slowing burning.

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

· In the event of a spill of a reactive diluent, the focus is on containing the spill to prevent contamination of soil and surface or ground water.

If irritating vapors are present, an approved air-purifying respirator with organic vapor canister is recommended for cleaning up spills and leaks.

· For small spills, reactive diluents should be absorbed with sand.

Minor Spills

Environmental hazard - contain spillage.

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.

Environmental hazard - contain spillage.

Chemical Class: phenols and cresols

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		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

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

Major Spills

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

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

Industrial spills or releases of reactive diluents are infrequent and generally contained. If a large spill does occur, the material should be captured, collected, and reprocessed or disposed of according to applicable governmental requirements.

An approved air-purifying respirator with organic-vapor canister is recommended for emergency work.

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

- 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.
- ▶ 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

Other information

Safe handling

- 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

Suitable container

- Metal can or drum
- Packaging as recommended by manufacturer.
- Check all containers are clearly labelled and free from leaks.

For aluminas (aluminium oxide):

Incompatible with hot chlorinated rubber.

In the presence of chlorine trifluoride may react violently and ignite.

-May initiate explosive polymerisation of olefin oxides including ethylene oxide.

-Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.

- -Produces exothermic reaction with oxygen difluoride.
- -May form explosive mixture with oxygen difluoride.
- -Forms explosive mixtures with sodium nitrate.
- -Reacts vigorously with vinyl acetate.

Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.

The substance may be or contains a 'metalloid'

The following elements are considered to be metalloids; boron, silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium. The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine.

Unlike most metalls, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases.

Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids

Storage incompatibility

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.

react like non-metals when they react with metals and act like metals when they react with non-metals.

- 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.
- Avoid strong acids, bases.

Slycidyl ethers:

- may form unstable peroxides on storage in air ,light, sunlight, UV light or other ionising radiation, trace metals inhibitor should be maintained at adequate levels
- 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

Reactive diluents are stable under recommended storage conditions, but can decompose at elevated temperatures. In some cases, decomposition can cause pressure build-up in closed systems.

- Avoid cross contamination between the two liquid parts of product (kit).
- If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur.
- ► This excess heat may generate toxic vapour
- ▶ Avoid reaction with amines, mercaptans, strong acids and oxidising agents

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
alumina hydrate	Inhalation 10.76 mg/m³ (Systemic, Chronic) Inhalation 10.76 mg/m³ (Local, Chronic) Oral 4.74 mg/kg bw/day (Systemic, Chronic) *	Not Available
zinc borate	Dermal 1 585 mg/kg bw/day (Systemic, Chronic) Inhalation 22.4 mg/m³ (Systemic, Chronic) Dermal 1 205 mg/kg bw/day (Systemic, Chronic) * Inhalation 8.3 mg/m³ (Systemic, Chronic) * Oral 2.4 mg/kg bw/day (Systemic, Chronic) *	2.9 mg/L (Water (Fresh)) 2.9 mg/L (Water - Intermittent release) 13.7 mg/L (Water (Marine)) 117.8 mg/kg sediment dw (Sediment (Fresh Water)) 56.5 mg/kg sediment dw (Sediment (Marine)) 5.7 mg/kg soil dw (Soil) 10 mg/L (STP)

^{*} Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Not Available						

Not Applicable

Emergency Limits

Ingredient	TEEL-1 TEEL-2			TEEL-3	
alumina hydrate	8.7 mg/m3	73 mg/m3		440 mg/m3	
Ingredient	Original IDLH		Revised IDLH		
alumina hydrate	Not Available		Not Available		
bisphenol F diglycidyl ether copolymer	Not Available		Not Available		
zinc borate	Not Available		Not Available Not Available		
neopentyl glycol diglycidyl ether	Not Available		Not Available		

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit	
bisphenol F diglycidyl ether copolymer	E	≤ 0.1 ppm	
zinc borate	E	≤ 0.01 mg/m³	
neopentyl glycol diglycidyl ether	Е	≤ 0.1 ppm	
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.		

MATERIAL DATA

For aluminium oxide:

The experimental and clinical data indicate that aluminium oxide acts as an 'inert' material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.

[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For epichlorohydrin

Odour Threshold Value: 0.08 ppm

NOTE: Detector tubes for epichlorohydrin, measuring in excess of 5 ppm, are commercially available.

Exposure at or below the recommended TLV-TWA is thought to minimise the potential for adverse respiratory, liver, kidney effects. Epichlorohydrin has been implicated as a human skin sensitiser, hence individuals who are hypersusceptible or otherwise unusually responsive to certain chemicals may NOT be adequately protected from adverse health effects. Odour Safety Factor (OSF)

OSF=0.54 (EPICHLOROHYDRIN)

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. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed 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.

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.)

8.2.1. Appropriate engineering controls

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











Eye and face protection

- Safety glasses with side shields.
- ► Chemical goggles

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

Skin protection

Hands/feet 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.

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

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

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

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

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

Continued...

use

Contaminated gloves should be replaced.

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

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

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

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

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

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

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

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

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

The performance, based on breakthrough times ,of:

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

As defined in ASTM F-739-96

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

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

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

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

Body protection

See Other protection below

Other protection

- Overalls.P.V.C apron.
- Barrier cream.
- ► Skin cleansing cream.
- ► Eye wash unit.

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1	-
up to 50	1000	-	A-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	A-2
up to 100	10000	-	A-3
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand

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

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

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

Beige

Physical state	Liquid	Relative density (Water = 1)	1.71
Odour	Slight	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)	>150	Molecular weight (g/mol)	Not Available
Flash point (°C)	150	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	Immiscible	pH as a solution (%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	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

The material is not thought to produce either adverse health effects or irritation of the respiratory tract 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 freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.

In animal testing, exposure to aerosols of some reactive diluents (notably o-cresol glycidyl ether, CAS RN: 2210-79-9) has been reported to affect the adrenal gland, central nervous system, kidney, liver, ovaries, spleen, testes, thymus, and respiratory tract.

Ingestion

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

Reactive diluents exhibit a range of ingestion hazards. Small amounts swallowed incidental to normal handling operations are not likely to cause injury. However, swallowing larger amounts may cause injury.

At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss

of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes)

At sufficiently high doses the material may be nephrotoxic (i.e. poisonous to the kidney).

Acute toxic responses to aluminium are confined to the more soluble forms.

Symptoms of borate poisoning include nausea, vomiting, diarrhoea, epigastric pain. These may be accompanied headache, weakness and a distinctive red skin rash. In severe cases there may be shock, increased heart rate and the skin may appear blue. Vomiting (which may be violent) is often persistent and vomitus and faeces may contain blood. Weakness, lethargy, headache, restlessness, tremors and intermittent convulsions may also occur. Poisoning produces central nervous system stimulation followed by depression, gastrointestinal disturbance (haemorrhagic gastro-enteritis), erythematous skin eruptions (giving rise to a boiled lobster appearance) and may also involve kidneys (producing oliguria, albuminuria, anuria) and, rarely, liver (hepatomegaly, jaundice). Toxic symptoms may be delayed for several hours.

Ingested borates are readily absorbed and do not appear to be metabolised via the liver. Excretion occurs mainly through the kidneys in the urine with about half excreted in the first 12 hours and the remainder over 5-12 days. Borates are excreted primarily in the urine regardless of the route of administration.

The borates (tetra-, di-, meta, or ortho- salts, in contrast to perborates) once solubilised in the acid of gastric juices, cannot be distinguished from each other on chemical or toxicological grounds. In humans acute gastroenteric (or percutaneous absorption of as little as 1 gm of sodium borate can result in severe gastrointestinal irritation, kidney damage. In adults the mean lethal dose of sodium borate or boric acid probably exceeds 30 gms (Gosselin) and death occurs due to vascular collapse in the early stages or to central nervous system depression in later stages. Children are thought to be more susceptible to the effects of borate intoxication.

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.

Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.

Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Skin contact with reactive diluents may cause slight to moderate irritation with local redness. Repeated or prolonged skin contact may cause burns.

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.

The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either

- roduces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or
- produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.

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.

Eye

When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury.

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.

Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.

Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers

Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.

Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health

surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.

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

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

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.

Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity. Testicular abnormalities (including testicular atrophy with decreased spermatogenic activity) following exposure to glycidyl ethers have been reported. Haemopoietic abnormalities following exposure to glycidyl ethers, including alteration of the leukocyte count, atrophy of lymphoid tissue, and bone marrow cytotoxicity have also been reported. These abnormalities were usually observed along with pneumonia and/or toxemia, and therefore may be secondary effects. However, especially in light of the generalized reduction in leukocytes and the atrophy of lymphoid tissues, the observed haemopoietic abnormalities may have been predisposing factors to pneumonia. While none of the individual research reports are conclusive with respect to the ability of glycidyl ethers to produce permanent changes to the testes or haemopoietic system in laboratory animals, the pattern of displayed effects is reason for concern

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. A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not not nebutyl 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. For some reactive diluents, prolonged or repeated skin contact may result in absorption of potentially harmful amounts or allergic skin reactions Exposure to some reactive diluents (notably neopentylglycol diglycidyl ether, CAS RN:17557-23-2) has caused cancer in some animal testing. Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.

Skin Contact

Chronic

mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.1 smg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of 'tau' a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995] Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.

Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.

When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.

The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.

Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.

There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.

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.

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 detoxification pathway

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. The presence of the p-hydroxy group on the benzene rings is though to be responsible for the oestradiol mimicry.

. 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.

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).

BPA belongs to the list of compounds having this property as the rodent models have shown that BPA exposure is linked with increased body weigh (obesogens)t. Several mechanisms can help explain the effect of BPA on body weight increase. A possible mechanism leading to triglyceride accumulation is the decreased production of the hormone adiponectin from all human adipose tissue tested when exposed to very low levels (below nanomolar range) of BPA in cell or explant culture settings. The expression of leptin as well as several enzymes and transcription factors is also affected by BPA exposure in vivo as well as in vitro. Together, the altered expression and activity of these important mediators of fat metabolism could explain the increase in weight following BPA exposure in rodent models. These results also suggest that, together with other obesogens, low, environmentally relevant levels of BPA may contribute to the human obesity phenomenon.

8329TFF-A Thermally
Conductive Epoxy Adhesive

TOXICITY	IRRITATION
Not Available	Not Available

alumina hydrate

TOXICITY	IRRITATION
Inhalation(Rat) LC50; >2.3 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
Oral(Rat) LD50; >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]

bisphenol F diglycidyl ether copolymer

TOXICITY	IRRITATION		
dermal (rat) LD50: >400 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]		
Oral(Rat) LD50; >2000 mg/kg ^[2]	Skin: adverse effect observed (irritating) ^[1]		

zinc borate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye (rabbit): mild *
Inhalation(Rat) LC50; 4.95 mg/l4h ^[1]	Eye: adverse effect observed (irritating) ^[1]
Oral(Rat) LD50; >5000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Skin: non-irritant *

neopentyl glycol diglycidyl

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 2150 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
Oral(Rat) LD50; 4500 mg/kg ^[2]	Skin (human): Sensitiser [Shell]
	Skin: adverse effect observed (irritating) ^[1]

Legend:

 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

8329TFF-A Thermally Conductive Epoxy Adhesive

For aluminium compounds:

Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex Ligands in food can have

a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate). Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability.

For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe. Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium.

The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account

Systemic toxicity after repeated exposure

No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium.

When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose.

The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent

Reproductive and developmental toxicity:

Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity

High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI.

Genotoxicity

Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.

Carcinogenicity

The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons.

Neurodegenerative diseases.

Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia, some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.

It has been suggested that the body burden of aluminium may be linked to different iseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome.

Aluminium acts not only as an adjuvant, stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitisers causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response. Once inside the skin, the metal ions must bind to proteins to become immunologically reactive. The most important

routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminium

Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste. The various members of the bisphenol family produce hormone like effects, seemingly as a result of binding to estrogen receptor-related receptors (ERRs; not to be confused with estrogen receptors)

A suspected estrogen-related receptors (ERR) binding agent:

Estrogen-related receptors (ERR, oestrogen-related receptors) are so named because of sequence homology with estrogen receptors but do not appear to bind estrogens or other tested steroid hormones. The ERR family have been demonstrated to control energy homeostasis, oxidative metabolism and mitochondrial biogenesis ,while effecting mammalian physiology in the heart, brown adipose tissue, white adipose tissue, placenta, macrophages, and demonstrated additional roles in diabetes and cancer.

ERRs bind enhancers throughout the genome where they exert effects on gene regulation

Although their overall functions remain uncertain, they also share DNA-binding sites, co-regulators, and target genes with the conventional estrogen receptors ERalpha and ERbeta and may function to modulate estrogen signaling pathways.

- ERR-alpha has wide tissue distribution but it is most highly expressed in tissues that preferentially use fatty acids as energy sources such as kidney, heart, brown adipose tissue, cerebellum, intestine, and skeletal muscle. ERRalpha has been detected in normal adrenal cortex tissues, in which its expression is possibly related to adrenal development, with a possible role in fetal adrenal function, in dehydroepiandrosterone (DHEAS) production in adrenarche, and also in steroid production of post-adrenarche/adult life. DHEA and other adrenal androgens such as androstenedione, although relatively weak androgens, are responsible for the androgenic effects of adrenarche, such as early pubic and axillary hair growth, adult-type body odor, increased oiliness of hair and skin, and mild acne.
- ERR-beta is a nuclear receptor . Its function is unknown; however, a similar protein in mouse plays an essential role in placental development

ERR-gamma is a nuclear receptor that behaves as a constitutive activator of transcription. There is evidence that bisphenol A functions as an endocrine disruptor by binding strongly to ERRgamma BPA as well as its nitrated and chlorinated metabolites seems to binds strongly to ERR-gamma (dissociation constant = 5.5 nM), but not to the estrogen receptor (ER). BPA binding to ERR-gamma preserves its basal constitutive activity. Different expression of ERR-gamma in different parts of the body may account for variations in bisphenol A effects. For instance, ERR-gamma has been found in high concentration in the placenta, explaining reports of high bisphenol A accumulation there

BISPHENOL F DIGLYCIDYL ETHER COPOLYMER

Data for liquid polymer, ie for molecular weights generally less than 700 CAUTION: Epoxy resin products may contain sensitising glycidyl ethers, even when these are not mentioned in the information given for the product. Limited animal studies have indicated that bisphenol A diglycidyl ethers may be potential carcinogens. [CISDOC Patty]

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

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

NEOPENTYL GLYCOL DIGLYCIDYL ETHER

* Anchor SDS] 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

8329TFF-A Thermally Conductive Epoxy Adhesive & BISPHENOL F DIGLYCIDYL ETHER COPOLYMER & NEOPENTYL GLYCOL DIGLYCIDYL ETHER

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.

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.

8329TFF-A Thermally Conductive Epoxy Adhesive & BISPHENOL F DIGLYCIDYL ETHER COPOLYMER

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 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 moiety 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.

In vitro cell models were used to evaluate the ability of 22 bisphenols (BPs) to induce or inhibit estrogenic and androgenic activity. BPA, Bisphenol AF (BPAF), bisphenol Z (BPZ), bisphenol C (BPC), tetramethyl bisphenol A (TMBPA), bisphenol S (BPS), bisphenol E (BPE), 4,4-bisphenol F (4,4-BPF), bisphenol AP (BPAP), bisphenol B (BPB), tetrachlorobisphenol A (TCBPA), and benzylparaben (PHBB) induced estrogen receptor (ER)alpha and/or ERbeta-mediated activity. With the exception of BPS, TCBPA, and PHBB, these same BPs were also androgen receptor (AR) antagonists. Only 3 BPs were found to be ER antagonists. Bisphenol P (BPP) selectively inhibited ERbeta-mediated activity and 4-(4-phenylmethoxyphenyl)sulfonylphenol (BPS-MPE) and 2,4-bisphenol S (2,4-BPS) selectively inhibited ERalpha-mediated activity. None of the BPs induced AR-mediated activity.

ALUMINA HYDRATE & BISPHENOL F DIGLYCIDYL ETHER COPOLYMER

No significant acute toxicological data identified in literature search.

Acute Toxicity

X

Carcinogenicity

5

Skin Irritation/Corrosion	*	Reproductivity	✓
Serious Eye Damage/Irritation	~	STOT - Single Exposure	×
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	×
Mutagenicity	×	Aspiration Hazard	×

Leaend:

X - Data either not available or does not fill the criteria for classification

- Data available to make classification

11.2.1. Endocrine Disruption Properties

Many chemicals may mimic or interfere with the body's hormones, known as the endocrine system. Endocrine disruptors are chemicals that can interfere with endocrine (or hormonal) systems. Endocrine disruptors interfere with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body. Any system in the body controlled by hormones can be derailed by hormone disruptors. Specifically, endocrine disruptors may be associated with the development of learning disabilities, deformations of the body various cancers and sexual development problems. Endocrine disrupting chemicals cause adverse effects in animals. But limited scientific information exists on potential health problems in humans. Because people are typically exposed to multiple endocrine disruptors at the same time, assessing public health effects is difficult.

SECTION 12 Ecological information

12.1. Toxicity

8329TFF-A Thermally	, Endpoint Test Durati		Test Duration (hr) Species		Value	Value		Source	
nductive Epoxy Adhesive	Not Available	Not Available Not Available			Not Available Not Available		ole Not Available		
	Endpoint	Те	est Duration (hr)	Spec	cies		Value		Source
	NOEC(ECx)	72h		Algae or other aquatic plants		>100mg	Ί	1	
alumina hydrate	LC50	96h		Fish		0.57mg/	0.57mg/l		
	EC50	48	ih	Crustacea		>0.065m	>0.065mg/l		
	EC50	96	ih	Alga	e or other aquatic plant	s	0.46mg/		2
sphenol F diglycidyl ether	Endpoint		Test Duration (hr)		Species	Value		Source	,
copolymer	Not Available	Not Available Not Available			Not Available	Not Available		Not Available	
	Endpoint	Те	est Duration (hr)	Sne	cies		Value		Source
	EC50	72		Algae or other aquatic plants		40.2mg	1/1	2	
	LC50	96		Fish		1.793n	,	2	
zinc borate	EC50	48	Bh	Crustacea		1mg/l	<u> </u>	2	
NOEC(ECx) 768h		Fish	Fish		0.009mg/l		2		
	EC50	96	6h	Alga	ae or other aquatic plan	its	15.4mg]/I	2
eopentyl glycol diglycidyl	Endpoint		Test Duration (hr)		Species	Value		Source	
ether	Not Available		Not Available		Not Available	Not Availab	le	Not Ava	ilable
Legend:	Extracted from 1. IU	JCLID T	oxicity Data 2. Europe EC	CHA Regist	ered Substances - Eco	toxicological Infor	mation - Aqua	atic Toxicit	y 3. EPIWIN

Very 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:

Environmental fate:

Biodegradability (28 d) 89% - Easily biodegradable

Bioconcentration factor (BCF) 7.8 mg/l

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

Substance does not meet the criteria for PBT or vPvB according to Regulation (EC) No 1907/2006, Annex XIII

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.

Fish LC50 (96 h): 4.6 mg/l (freshwater fish); 11 mg/l (saltwater fish): NOEC 0.016 mg/l (freshwater fish- 144 d); 0.064 mg/l (saltwater fish 164 d)

Fresh water invertebrates EC50 (48 h): 10.2 mg/l: NOEC 0.025 mg/l - 328 d)

Marine water invertebrate EC50 (96 h): 1.1 mg/l; NOEC 0.17 mg/l (28 d)

Freshwater algae (96 h): 2.73 mg/l

Marine water algae (96 h): 1.1 mg/l

Fresh water plant EC50 (7 d): 20 mg/l: NOEC 7.8 mg/l

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

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 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(4-hydroxydiphenyl)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.

Reactive diluents generally have a low to moderate potential for bioconcentration (tendency to accumulate in the food chain) and a high to very high potential for mobility in soil. Small amounts that escape to the atmosphere will photodegrade.

They would not be expected to persist in the environment.

Most reactive diluents should be considered slightly to moderately toxic to aquatic organisms on an acute basis while some might also be considered harmful to the environment. Environmental toxicity is a function of the n-octanol/water partition coefficient (log Pow, log Kow). Compounds with log Pow >5 act as neutral organics, but at a lower log Pow, the toxicity of epoxide-containing polymers is greater than that predicted for simple narcotics.

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)*

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).

Reactive diluents which are only slightly soluble in water and do not evaporate quickly are expected to sink to the bottom or float to the top, depending on the density, where they would be expected to biodegrade slowly.

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.

Microbial methylation plays important roles in the biogeochemical cycling of the metalloids and possibly in their detoxification. Many microorganisms (bacteria, fungi, and yeasts) and animals are now known to biomethylate arsenic, forming both volatile (e.g., methylarsines) and nonvolatile (e.g., methylarsonic acid and dimethylarsinic acid) compounds. Antimony and bismuth, also undergo biomethylation to some extent. Trimethylstibine formation by microorganisms is now well established, but this process apparently does not occur in animals. Formation of trimethylbismuth by microorganisms has been reported in a few cases.

For boron and borates

Environmental fate:

Boron is generally found in nature bound to oxygen and is never found as the free element. Atmospheric boron may be in the form of particulate matter or aerosols as borides, boron oxides, borates, boranes, organoboron compounds, trihalide boron compounds, or borazines. Borates are relatively soluble in water, and will probably be removed from the atmosphere by precipitation and dry deposition. The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions. Boron readily hydrolyses in water to form the electrically neutral, weak monobasic acid boric acid (H3BO3) and the monovalent ion, B(OH)4-. In concentrated solutions, boron may polymerise, leading to the formation of complex and diverse molecular arrangements. Because most environmentally relevant boron minerals are highly soluble in water, it is unlikely that mineral equilibria will control the fate of boron in water. Boron was found to not be significantly removed during the conventional treatment of waste water. Boron may, however, be co-precipitated with aluminum, silicon, or iron to form hydroxyborate compounds on the surfaces of minerals.

Waterborne boron may be adsorbed by soils and sediments. Adsorption-desorption reactions are expected to be the only significant mechanism that will influence the fate of boron in water. The extent of boron adsorption depends on the pH of the water and the chemical composition of the soil. The greatest adsorption is generally observed at pH 7.5-9.0. the single most important property of soil that will influence the mobility of boron is the abundance of amorphous aluminum oxide. The extent of boron adsorption has also been attributed to the levels of iron oxide, and to a lesser extent, the organic matter present in the soil, although other studies found that the amount of organic matter present was not important. The adsorption of boron may not be reversible in some soils. The lack of reversibility may be the result of solid-phase formation on mineral surfaces and/or the slow release of boron by diffusion from the interior of clay minerals.

It is unlikely that boron is bioconcentrated significantly by organisms from water. A bioconcentration factor (BCF) relates the concentration of a chemical in the tissues of aquatic and terrestrial animals or plants to the concentration of the chemical in water or soil. The BCFs of boron in marine and freshwater plants, fish, and invertebrates were estimated to be <100. Experimentally measured BCFs for fish have ranged from 52 to 198. These BCFs suggest that boron is not significantly bioconcentrated.

As an element, boron itself cannot be degraded in the environment; however, it may undergo various reactions that change the form of boron (e.g., precipitation, polymerization, and acid-base reactions) depending on conditions such as its concentration in water and p.H. In nature, boron in generally found in its oxygenated form. In aqueous solution, boron is normally present as boric acid and borate ions, with the dominant form of inorganic boron in natural aqueous systems as undissociated boric acid. Boric acid acts as an electron acceptor in aqueous solution, accepting an hydroxide ion from water to form (B(OH)4)-ion. In dilute solution, the favored form of boron is B(OH)4. In more concentrated solutions (>0.1 M boric acid) and at neutral to alkaline pH (6–11), polymeric species are formed (e.g., B3O3(OH)4-, B3O3(OH)52-, and B4O5(OH)42-)

Most boron compounds are transformed to borates in soil due to the presence of moisture. Borates themselves are not further degraded in soil. However, borates can exist in a variety of forms in soil. Borates are removed from soils by water leaching and by assimilation by plants.

The most appreciable boron exposure to the general population is likely to be ingestion of food and to a lesser extent in water. As boron is a natural component of the environment, individuals will have some exposure from foods and drinking water

Boron-containing salts (borates) are ubiquitous in the environment. Surface soil, unpolluted waterways and seawater all typically contain significant amounts of boron as borate. Boron is an essential micronutrient for healthy growth of plants, however, it can be harmful to boron sensitive plants in higher quantities. In some areas such as the American Southwest, boron occurs naturally in surface waters in concentrations that have been shown to be toxic to commercially important plants.

Based on the collected information regarding aquatic toxicity, boron is not regarded as dangerous to aquatic organisms. The concentration in treated municipal waste water is a factor

100 lower than the NOEC-value for Daphnia magna.

No quality criteria exist for the concentration of boron in soil and compost. Boron is added to farmland when sewage sludge is applied as a soil improving agent, but there is not sufficient data to evaluate its effect on soil organisms. Being an essential micro-nutrient, no adverse effects of boron are expected at low concentrations.

Ecotoxicity

In aquatic environments low concentrations of borates generally promote the growth of algae, whereas higher concentrations inhibited algal growth. In a growth inhibition test with *Scenedesmus subspicatus*, an EC50 value of 34 mg B/l was determined. Boric acid toxicity in Daphnia 48 h-LC50 (static test) was found to be 95 mg B/l. In a separate study it was concluded that chronic effects of boron to Daphnia may occur at a concentration of > 10 mg/l.

The toxicity of boron in fish is often higher in soft water than in hard water. The acute toxicity of boron towards *Danio rerio* (96 h-LC50) has been determined to 14.2 mg B/l. In a fish early life stage test with rainbow trout NOEC levels of boron have been determined in the range between 0.009 and 0.103 mg B/l, whereas the EC50 ranged from 27 to 100 mg B/l dependent on the water hardness.

For aluminium and its compounds and salts:

Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals. Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.

Environmental fate:

Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.

Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.

As an element, aluminium cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminium in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminium can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminium in the environment will depend on the ligands present and the pH.

The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion, [Al(H2O)6]3+, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H2O)5(OH)]2+, [Al(H2O)4(OH)2]+). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)2+ and Al(OH)2+, while the solid Al(OH)3 is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)4- is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)3, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.

Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.

Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.

The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.

Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.

Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, Abies amabilis, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.

Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues. The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.

Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.

Ecotoxicity:

Freshwater species pH >6.5

Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (Salmo salar) - 106 mg/L; Chronic NOEC (8-28 d): 7 spp,NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for Micropterus sp.

Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L

Freshwater species pH <6.5 (all between pH 4.5 and 6.0)

Fish LC50 (24-96 h): 4 spp, 0.015 (S. trutta) - 4.2 mg/L; chronic data on Salmo trutta, LC50 (21-42 d) 0.015- 0.105 mg/L

Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)

Alga: 1 sp NOEC growth 2.0 mg/L

Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.

The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4.5.4, with a maximum toxicity occurring around pH 5.0.5.2. The inorganic single unit aluminium species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.

Drinking Water Standards: aluminium: 200 ug/l (UK max.)

200 ug/l (WHO guideline)

chloride: 400 mg/l (UK max.)

250 mg/l (WHO guideline)

fluoride: 1.5 mg/l (UK max.)

1.5 mg/l (WHO guideline)

nitrate: 50 mg/l (UK max.) 50 mg/l (WHO guideline)

sulfate: 250 mg/l (UK max.)

Soil Guideline: none available.

Air Quality Standards: none available.

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
neopentyl glycol diglycidyl ether	HIGH	HIGH

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
neopentyl glycol diglycidyl ether	LOW (LogKOW = 0.2342)

12.4. Mobility in soil

Ingredient	Mobility
neopentyl glycol diglycidyl ether	LOW (KOC = 10)

12.5. Results of PBT and vPvB assessment

	P	В	Т
Relevant available data	Not Available	Not Available	Not Available
PBT	X	×	×
vPvB	×	X	×
PBT Criteria fulfilled?			
vPvB			No

12.6. Endocrine Disruption Properties

The evidence linking adverse effects to endocrine disruptors is more compelling in the environment than it is in humans. Endocrine distruptors profoundly alter reproductive physiology of ecosystems and ultimately impact entire populations. Some endocrine-disrupting chemicals are slow to break-down in the environment. That characteristic makes them potentially hazardous over long periods of time. Some well established adverse effects of endocrine disruptors in various wildlife species include; eggshell-thinning, displayed of characteristics of the opposite sex and impaired reproductive development. Other adverse changes in wildlife species that have been suggested, but not proven include; reproductive abnormalities, immune dysfunction and skeletal deformaties.

12.7. Other adverse effects

Not 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
- 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.

Product / Packaging disposal

- ▶ 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.

Removal of bisphenol A (BPA) from aqueous solutions was accomplished by adsorption of enzymatically generated quinone derivatives on chitosan beads. The use of chitosan in the form of beads was found to be more effective because heterogeneous removal of BPA with chitosan beads was much faster than homogeneous removal of BPA with chitosan solutions, and the removal efficiency was enhanced by increasing the amount of chitosan beads dispersed in the BPA solutions and BPA was completely removed by quinone adsorption in the presence of chitosan beads more than 0.10 cm3/cm3. In addition, a variety of bisphenol derivatives were completely or effectively removed by the procedure constructed in this study, although the enzyme dose or the amount of chitosan beads was further increased as necessary for some of the bisphenol derivatives used.

M. Suzuki, and E Musashi J Appl Polym Sci, 118(2):721 - 732; October 2010

- ▶ 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.

Waste treatment options Not Available
Sewage disposal options Not Available

SECTION 14 Transport information

Labels Required

NOT REGULATED by Ground ADR Special Provision 375
NOT REGULATED by Air IATA Special Provision A197
NOT REGULATED by Sea IMDG per 2.10.2.7
NOT REGULATED by ADN Special Provision 274 (The provision of 3.1.2.8 apply)

Land transport (ADR-RID)

14.1. UN number	3082			
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc borate and bisphenol F diglycidyl ether copolymer)			
14.3. Transport hazard class(es)	Class 9			
	Subrisk Not Applicable			
14.4. Packing group				
14.5. Environmental hazard	Environmentally hazardous			
	Hazard identification (Kemler)	90		
	Classification code	M6		
14.6. Special precautions for user	Hazard Label	9		
	Special provisions	274 335 375 601		
	Limited quantity	5 L		
	Tunnel Restriction Code	3 (-)		

Air transport (ICAO-IATA / DGR)

14.1. UN number	3082			
14.2. UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains zinc borate and bisphenol F diglycidyl ether copolymer)			
14.3. Transport hazard class(es)	ICAO/IATA Class ICAO / IATA Subrisk	9 Not Applicable		
ciass(es)	ERG Code 9L			
14.4. Packing group				
14.5. Environmental hazard	Environmentally hazardous			
	Special provisions		A97 A158 A197 A215	
	Cargo Only Packing Instructions		964	
	Cargo Only Maximum Qty / Pack		450 L	
14.6. Special precautions for user	Passenger and Cargo Packing Instructions		964	
4001	Passenger and Cargo Maximum Qty / Pack		450 L	
	Passenger and Cargo Limited Quantity Packing Instructions		Y964	
	Passenger and Cargo	Limited Maximum Qty / Pack	30 kg G	

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3082		
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc borate and bisphenol F diglycidyl ether copolymer)		
14.3. Transport hazard class(es)	IMDG Class 9 IMDG Subrisk Not Applicable		
14.4. Packing group			
14.5. Environmental hazard	Marine Pollutant		
14.6. Special precautions for user	EMS Number F-A , S-F Special provisions 274 335 969 Limited Quantities 5 L		

Inland waterways transport (ADN)

14.1. UN number	3082
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc borate and bisphenol F diglycidyl ether copolymer)
14.3. Transport hazard class(es)	9 Not Applicable

14.4. Packing group	III		
14.5. Environmental hazard	Environmentally hazardous		
	Classification code	M6	
	Special provisions	274; 335; 375; 601	
14.6. Special precautions for user	Limited quantity	5 L	
user	Equipment required	PP	
	Fire cones number	0	

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

Not Applicable

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

Product name	Group
alumina hydrate	Not Available
bisphenol F diglycidyl ether copolymer	Not Available
zinc borate	Not Available
neopentyl glycol diglycidyl ether	Not Available

14.9. Transport in bulk in accordance with the ICG Code

•	
Product name	Ship Type
alumina hydrate	Not Available
bisphenol F diglycidyl ether copolymer	Not Available
zinc borate	Not Available
neopentyl glycol diglycidyl ether	Not Available

SECTION 15 Regulatory information

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

alumina hydrate is found on the following regulatory lists	
Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
bisphenol F diglycidyl ether copolymer is found on the following regulatory lists	
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances	Europe EC Inventory
zinc borate is found on the following regulatory lists	
Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
neopentyl glycol diglycidyl ether is found on the following regulatory lists	
Chemical Footprint Project - Chemicals of High Concern List Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
Europe 20 inventory	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 - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

National Inventory Status

National inventory Status	
National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (alumina hydrate; bisphenol F diglycidyl ether copolymer; neopentyl glycol diglycidyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (bisphenol F diglycidyl ether copolymer)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes

National Inventory	Status
Mexico - INSQ	No (neopentyl glycol diglycidyl ether)
Vietnam - NCI	Yes
Russia - FBEPH	No (neopentyl glycol diglycidyl ether)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 Other information

Revision Date	18/07/2021
Initial Date	29/03/2019

Full text Risk and Hazard codes

H340	May cause genetic defects.			
H360	May damage fertility or the unborn child.			
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.			
H410	Very toxic to aquatic life with long lasting effects.			

Other information

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

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

ES: Exposure Standard

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

AIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory

NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act

TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

Reason For Change

A-2.00 - Format change to the SDS



8329TFF-B Thermally Conductive Epoxy Adhesive (Part B) MG Chemicals UK Limited

Version No: A-3.00

Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: 19/07/2021 Revision Date: 19/07/2021 L.REACH.GB.EN

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

1.1. Product Identifier

Product name 8329TFF-B		
Synonyms SDS Code: 8329TFF-Part B; 8329TFF-25ML, 8329TFF-50ML UFI:58F0-W072-3007-6T64		
Other means of identification Thermally Conductive Epoxy Adhesive (Part B)		

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

Relevant identified uses	Thermally conductive adhesive hardener		
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		9347 - 193 Street Surrey V4N 4E7 British Columbia Canada	
Telephone +(44) 1663 362888 +(1) 800-201-8822		+(1) 800-201-8822	
Fax Not Available +(1) 800-708-9888		+(1) 800-708-9888	
Website	Not Available	www.mgchemicals.com	
Email sales@mgchemicals.com Info@mgchemicals.com			

1.4. Emergency telephone number

Association / Organisation	Verisk 3E (Access code: 335388)				
Emergency telephone numbers	+(44) 20 35147487				
Other emergency telephone numbers	+(0) 800 680 0425				

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H314 - Skin Corrosion/Irritation Category 1B, H317 - Skin Sensitizer Category 1B, H412 - Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567

2.2. Label elements

Hazard pictogram(s)



	_
Signal word	Dange

Hazard statement(s)

H314	Causes severe skin burns and eye damage.		
H317	May cause an allergic skin reaction.		
H412	Harmful to aquatic life with long lasting effects.		

Supplementary statement(s)

Not Applicable

P260	Do not breathe mist/vapours/spray.			
P264	P264 Wash all exposed external body areas thoroughly after handling.			
P280	Wear protective gloves, protective clothing, eye protection and face protection.			
P273	Avoid release to the environment.			
P272	Contaminated work clothing should not be allowed out of the workplace.			

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.			
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].			
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.			
P310	Immediately call a POISON CENTER/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.			
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.			

Precautionary statement(s) Storage

P405 Store locked up.

Precautionary statement(s) Disposal

P501 Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

2.3. Other hazards

Inhalation 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	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
1.21645-51-2 2.244-492-7 3.Not Available 4.Not Available	50	aluminium hydroxide	Eye Irritation Category 2; H319, EUH066 ^[1]	Not Available
1.72244-98-5 2.Not Available 3.Not Available 4.Not Available	42	trimercaptan ether, propoxylated	Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 3; H317, H412 [1]	Not Available
1.90-72-2 2.202-013-9 3.603-069-00-0 4.Not Available	8	2.4.6- tris[(dimethylamino)methyl]phenol	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2; H302, H315, H319 [2]	Not Available
Legend:	Legend: 1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties			3. Classification drawn

SECTION 4 First aid measures

4.1. Description of first aid measures

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.
Skin Contact	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.
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. 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 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.
Ingestion	 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 induvomiting 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 amines:

- Certain amines may cause injury to the respiratory tract and lungs if aspirated. Also, such products may cause tissue destruction leading to stricture. If lavage is performed, endotracheal and/or 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 airway irritants.

Lung injury may result following a single massive overexposure to high vapour concentrations or multiple exposures to lower concentrations of any pulmonary irritant material. Health effects of amines, such as skin irritation and transient corneal edema ("blue haze," "halo effect," "glaucopsia"), are best prevented by means of formal worker education, industrial hygiene monitoring, and exposure control methods. Persons who are highly sensitive to the triggering effect of non-specific irritants should not be assigned to jobs in which such agents are used, handled, or manufactured.

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

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

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

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

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

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

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

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

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

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

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	Incom	patibility

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.
- 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.
- Fire Fighting 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.

▶ Slight fire

- 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 comb
- Mists containing combustible materials may be explosive. Combustion products include:

Fire/Explosion Hazard

carbon dioxide (CO2) nitrogen oxides (NOx)

sulfur oxides (SOx) other pyrolysis products typical of burning organic material.

May emit corrosive fumes

Aluminium hydroxide is a flame retardant. At around 200 C, aluminium hydroxide (aluminium trihydrate) is decomposed to aluminium oxide (which forms a protective, non-flammable layer on the material surface) and water. The water (as steam) forms a layer of non-flammable gas near the material's surface, inhibiting flames. The reaction is endothermic (absorbs heat energy), thus cooling the material and slowing burning.

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

- Prains 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.
- $\mbox{\ }\mbox{\ }\mbox{\ }$ 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

for amines: **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.
- Foroms 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.

Chemical Class: amines, alkyl

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
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT,
treated wood fibre - pillow	3	throw	pitchfork	DGC, RT
foamed glass - pillow	4	throw	pitchfork	R, P, DGC, RT

LAND SPILL - MEDIUM

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	W, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	4	throw	skiploader	DGC, RT

Legend

DGC: Not effective where ground cover is dense

Major Spills

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

NOTE:

- P Organic absorbents have been known to ignite when contaminated with amines in closed containers. Certain cellulosic materials used for spill cleanup such as wood chips or sawdust have shown reactivity with ethyleneamines and should be avoided.
- 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.

For amines:

- 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.
- Fightlis 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 plan
- 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.
- Rubber gloves
- 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.
- Use in a well-ventilated area.
- WARNING: To avoid violent reaction, ALWAYS add material to water and NEVER water to material.
- 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

See section 5

Other information

Safe handling

- ▶ 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

- 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

- 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

Ingredient	DNELs	PNECs
	Exposure Pattern Worker	Compartment

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
aluminium hydroxide	Inhalation 10.76 mg/m³ (Systemic, Chronic) Inhalation 10.76 mg/m³ (Local, Chronic) Oral 4.74 mg/kg bw/day (Systemic, Chronic) *	Not Available
2,4,6- tris[(dimethylamino)methyl]phenol	Not Available	0.084 mg/L (Water (Fresh)) 0.008 mg/L (Water - Intermittent release) 0.84 mg/L (Water (Marine)) 0.2 mg/L (STP)

^{*} Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Not Available						

Not Applicable

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
aluminium hydroxide	8.7 mg/m3	73 mg/m3	440 mg/m3
2,4,6- tris[(dimethylamino)methyl]phenol	6.5 mg/m3	72 mg/m3	430 mg/m3

Ingredient	Original IDLH	Revised IDLH
aluminium hydroxide	Not Available	Not Available
trimercaptan ether, propoxylated	Not Available	Not Available
2,4,6- tris[(dimethylamino)methyl]phenol	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit		
aluminium hydroxide	E	≤ 0.01 mg/m³		
trimercaptan ether, propoxylated	D	> 0.1 to ≤ 1 ppm		
2,4,6- tris[(dimethylamino)methyl]phenol	С	> 1 to ≤ 10 parts per million (ppm)		
Notes:	, , , , , , , , , , , , , , , , , , , ,	exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the h outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a sure concentrations that are expected to protect worker health.		

MATERIAL DATA

Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.

OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

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.

8.2.1. Appropriate engineering controls

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.

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









- Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient 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.
- ▶ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles 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:

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

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

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

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

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.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

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

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

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

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

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

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

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

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

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.
- Eyewash unit.
- Ensure there is ready access to a safety shower.

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator	
up to 10	1000	AK-AUS / Class1 P2	-	
up to 50	1000	-	AK-AUS / Class 1 P2	
up to 50	5000	Airline *	-	
up to 100	5000	-	AK-2 P2	
up to 100	10000	-	AK-3 P2	
100+			Airline**	

* - Continuous Flow ** - Continuous-flow or positive pressure demand

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

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

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

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	Beige to light yellow			
Physical state	Liquid	Relative density (Water = 1)	1.5	
Odour	Not Available	Partition coefficient n-octanol / water	Not Available	
Odour threshold	Not Available	Auto-ignition temperature (°C)	365	
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)	118	Molecular weight (g/mol)	Not Available	

Flash point (°C)	124	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	Immiscible	pH as a solution (%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	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

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.

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.

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.

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. Inhalation hazard is increased at higher temperatures.

Ingestion

effects of stricture formation.

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

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.

	corroborating animal or human evidence. The material may still pre-existing organ (e.g liver, kidney) damage is evident. Present	classification systems as 'harmful by ingestion'. This is because of the lack of be damaging to the health of the individual, following ingestion, especially where t definitions of harmful or toxic substances are generally based on doses ase, ill-health). Gastrointestinal tract discomfort may produce nausea and		
Skin Contact	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. 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.			
Еуе	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. 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.			
Chronic	Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and neore (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. 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. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nos asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are like become hyper-responsive. Chronic Chronic			
8329TFF-B Thermally Conductive Epoxy Adhesive (Part B		IRRITATION Not Available		
aluminium hydroxidd	TOXICITY Inhalation(Rat) LC50; >2.3 mg/l4h ^[1] Oral(Rat) LD50; >2000 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]		
trimercaptan ether, propoxylated	TOXICITY Dermal (rabbit) LD50: >10200 mg/kg ^[2] Oral(Rat) LD50; 2600 mg/kg ^[2]	IRRITATION Not Available		

IRRITATION

dermal (rat) LD50: >973 mg/kg^[1]

Oral(Rat) LD50; 2169 mg/kg^[1]

2,4,6tris[(dimethylamino)methyl]phenol Eye (rabbit): 0.05 mg/24h - SEVERE

Skin (rabbit): 2 mg/24h - SEVERE

Eye: adverse effect observed (irreversible damage) [1]

Continued...

Skin: adverse effect observed (corrosive)[1]

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

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture .

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers

Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69

TRIMERCAPTAN ETHER, PROPOXYLATED

Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations.

Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of

http://doi.org/10.5487/TR.2015.31.2.105

2.4.6 TRIS[(DIMETHYLAMINO)METHYL]PHENOL

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.

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

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

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

Inhalation:

Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.

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

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

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

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

8329TFF-B Thermally Conductive Epoxy Adhesive (Part B) & 2,4,6-TRIS[(DIMETHYLAMINO)METHYL]PHENOL

include asthma, bronchitis, and emphysema.

Skin Contact:

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

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

Eye Contact:

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

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

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

The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.

Ingestion:

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

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

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

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

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

8329TFF-B Thermally Conductive Epoxy Adhesive (Part B) & TRIMERCAPTAN ETHER, PROPOXYLATED 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.

ALUMINIUM HYDROXIDE & TRIMERCAPTAN ETHER, PROPOXYLATED & 2,4,6-TRIS[(DIMETHYLAMINO)METHYL]PHENOL

No significant acute toxicological data identified in literature search.

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

Legend:

X - Data either not available or does not fill the criteria for classification

– Data available to make classification

11.2.1. Endocrine Disruption Properties

Not Available

12.1. Toxicity

SECTION 12 Ecological information

8329TFF-B Thermally Conductive	Endpoint	Test Duration (hr)	Species	Value		Source	
Epoxy Adhesive (Part B)	Not Available	Not Available	Not Available	Not Available		Not Availa	able
	Endpoint	Test Duration (hr)	Species		Value		Source

aluminium hydroxide

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
LC50	96h	Fish	0.57mg/l	2
EC50	48h	Crustacea	>0.065mg/l	4
EC50	96h	Algae or other aquatic plants	0.46mg/l	2

trimercaptan ether, propoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

2,4,6tris[(dimethylamino)methyl]phenol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	72h	Algae or other aquatic plants	2.8mg/l	2
EC50	72h	Algae or other aquatic plants	2.8mg/l	2
LC50	96h	Fish	175mg/l	2

Legend:

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

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

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.

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
2,4,6- tris[(dimethylamino)methyl]phenol	нієн	HIGH

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation	
2,4,6- tris[(dimethylamino)methyl]phenol	LOW (LogKOW = 0.773)	

12.4. Mobility in soil

Ingredient	Mobility
2,4,6- tris[(dimethylamino)methyl]phenol	LOW (KOC = 15130)

12.5. Results of PBT and vPvB assessment

	P	В	Т	
Relevant available data	Not Available	Not Available	Not Available	
PBT	X	×	X	
vPvB	×	×	×	
PBT Criteria fulfilled?				
vPvB			No	

12.6. Endocrine Disruption Properties

Not Available

12.7. Other adverse effects

Not 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
- Disposal (if all else fails)

Product / Packaging disposal

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

- 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 licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed 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

Continued...

SECTION 14 Transport information

Labels Required



Excepted Quantity
Code E2 for all modes of transport.
On air waybill, write "Dangerous Goods in Excepted Quantity"

Land transport (ADR-RID)

	1		
14.1. UN number	2735		
14.2. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. (contains 2,4,6-tris[(dimethylamino)methyl]phenol)		
14.3. Transport hazard class(es)	Class 8 Subrisk Not Applicable		
14.4. Packing group			
14.5. Environmental hazard	Not Applicable		
	Hazard identification (Kemler) Classification code	80 C7	
14.6. Special precautions for	Hazard Label	8	
user	Special provisions	274	
	Limited quantity	1L	
	Tunnel Restriction Code	2 (E)	

Air transport (ICAO-IATA / DGR)

14.1. UN number	2735					
14.2. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. (contains 2,4,6-tris[(dimethylamino)methyl]phenol)					
	ICAO/IATA Class	8				
14.3. Transport hazard class(es)	ICAO / IATA Subrisk	Not Applicable				
(,	ERG Code	8L				
14.4. Packing group	П					
14.5. Environmental hazard	Not Applicable					
	Special provisions		A3 A803			
	Cargo Only Packing Ir	nstructions	855			
	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		1 L			
	Passenger and Cargo Limited Quantity Packing Instructions		Y840			
	Passenger and Cargo Limited Maximum Qty / Pack		0.5 L			

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	2735		
14.2. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. (contains 2,4,6-tris[(dimethylamino)methyl]phenol)		
14.3. Transport hazard class(es)	IMDG Class 8 IMDG Subrisk Not Applicable		
14.4. Packing group	П		
14.5. Environmental hazard	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. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. (contains 2,4,6-tris[(dimethylamino)methyl]phenol)

14.3. Transport hazard class(es)	8 Not Applicable		
14.4. Packing group	II.		
14.5. Environmental hazard	Not Applicable		
	Classification code Special provisions	C7 274	
14.6. Special precautions for user	Limited quantity	1 L	
	Equipment required	PP, EP	
	Fire cones number	0	

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

Not Applicable

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

Product name	Group
aluminium hydroxide	Not Available
trimercaptan ether, propoxylated	Not Available
2,4,6- tris[(dimethylamino)methyl]phenol	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
aluminium hydroxide	Not Available
trimercaptan ether, propoxylated	Not Available
2,4,6- tris[(dimethylamino)methyl]phenol	Not Available

SECTION 15 Regulatory information

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

aluminium hydroxide is found on the following regulatory lists	
Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
trimercaptan ether, propoxylated is found on the following regulatory lists	
Not Applicable	
2,4,6-tris[(dimethylamino)methyl]phenol is found on the following regulatory lists	
Furone FC Inventory	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and

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

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 - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (aluminium hydroxide; trimercaptan ether, propoxylated; 2,4,6-tris[(dimethylamino)methyl]phenol)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (trimercaptan ether, propoxylated)
Japan - ENCS	No (trimercaptan ether, propoxylated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (trimercaptan ether, propoxylated)
Vietnam - NCI	Yes
Russia - FBEPH	No (trimercaptan ether, propoxylated)

National Inventory	Status
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 Other information

Revision Date	19/07/2021
Initial Date	07/02/2019

Full text Risk and Hazard codes

H302	Harmful if swallowed.
H315	Causes skin irritation.
H319	Causes serious eye irritation.

Other information

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

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

ES: Exposure Standard

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value BCF: BioConcentration Factors

BEI: Biological Exposure Index

AIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory

NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act

TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

Reason For Change

A-3.00 - Format change to the SDS