Kit Revision Date: 15/09/2021

FLAME RETARDANT STRUCTURAL 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*
A	9200FR-A	Epoxy resin for use with hardeners
B	9200FR-B	Epoxy hardener for use with resins

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 all parts listed above.

NOT REGULATED by ground, kits 9200FR-25ML and 9200FR-50ML.

9200FR-A Flame Retardant Structural Epoxy Adhesive (Part A)

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: **13/09/2021**
Revsion Date: **13/09/2021**
L.REACH.GB.EN

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

1.1. Product Identifier

Product name	9200FR-A
Synonyms	SDS Code: 9200FR-Part A, 9200FR-25ML, 9200FR-50ML | UFI:RMN0-C09E-R00V-529W
Other means of identification	Flame Retardant Structural Epoxy Adhesive (Part A)

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

Relevant identified uses	epoxy resine for use with hardeners
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	**MG Chemicals UK Limited**	**MG Chemicals (Head office)**
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	+(44) 1663 362888	+(1) 800-201-8822
Fax	Not Available	+(1) 800-708-9888
Website	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 - Hazardous to the Aquatic Environment Long-Term Hazard Category 2, H302 - Acute Toxicity (Oral) Category 4, H315 - Skin Corrosion/Irritation Category 2, H319 - Serious Eye Damage/Eye Irritation Category 2, H361 - Reproductive Toxicity Category 2, H317 - Sensitisation (Skin) 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.
H302	Harmful if swallowed.
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.

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Supplementary statement(s)

EUH205	Contains epoxy constituents. May produce an allergic reaction.

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.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
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.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

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

Cumulative effects may result following exposure*.

May produce discomfort of the respiratory system*.

Possible respiratory sensitizer*.

Possible cancer-causing agent*.

May produce genetic damage*.

phenol/ formaldehyde glycidyl 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**
1.9003-36-5 2.500-006-8 3.Not Available 4.Not Available	33	phenol/ formaldehyde glycidyl ether copolymer **[e]**	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Sensitisation (Skin) Category 1, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H315, H319, H317, H361fd, H411, EUH205 [1]	Not Available
1.21645-51-2 2.244-492-7 3.Not Available 4.Not Available	20	aluminium hydroxide	Serious Eye Damage/Eye Irritation Category 2; H319, EUH066 [1]	Not Available
1.68333-79-9 2.269-789-9 3.Not Available 4.Not Available	19	ammonium polyphosphate	Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H413 [1]	Not Available
1.1675-54-3 2.216-823-5 3.603-073-00-2|603-074-00-8 4.Not Available	19	bisphenol A diglycidyl ether	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Sensitisation (Skin) Category 1; H315, H319, H317 [2]	Not Available

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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
1.12767-90-7 2.235-804-2 3.Not Available 4.Not Available	6	zinc borate	Serious Eye Damage/Eye Irritation Category 2, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 1; H319, H360FD, H410 [1]	Not Available
1.60506-81-2 2.262-270-8 3.Not Available 4.Not Available	2	dipentaerythritol pentaacrylate	Serious Eye Damage/Eye Irritation Category 2, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3; H319, H317, H412 [1]	Not Available
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*			

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 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.
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.
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.

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

As in all cases of suspected poisoning, follow the ABCDEs of emergency medicine (airway, breathing, circulation, disability, exposure), then the ABCDEs of toxicology (antidotes, basics, change absorption, change distribution, change elimination).
For poisons (where specific treatment regime is absent):

BASIC TREATMENT

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

ADVANCED TREATMENT

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

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

- All treatments should be based on observed signs and symptoms of distress in the patient. Consideration should be given to the possibility that overexposure to materials other than this product may have occurred.

Continued...

- Ingestion of large quantities of phosphate salts (over 1.0 grams for an adult) may cause an osmotic catharsis resulting in diarrhoea and probable abdominal cramps. Larger doses such as 4-8 grams will almost certainly cause these effects in everyone. In healthy individuals most of the ingested salt will be excreted in the faeces with the diarrhoea and, thus, not cause any systemic toxicity. Doses greater than 10 grams hypothetically may cause systemic toxicity.
- Treatment should take into consideration both anionic and cation portion of the molecule.
- All phosphate salts, except calcium salts, have a hypothetical risk of hypocalcaemia, so calcium levels should be monitored.

Treat symptomatically.

SECTION 5 Firefighting measures

5.1. Extinguishing media

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

5.2. Special hazards arising from the substrate or mixture

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

5.3. Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ May be violently or explosively reactive. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Fight fire from a safe distance, with adequate cover. ‣ If safe, switch off electrical equipment until vapour fire hazard removed. ‣ Use water delivered as a fine spray to control the fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **Do not** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) aldehydes nitrogen oxides (NOx) phosphorus oxides (POx) metal oxides other pyrolysis products typical of burning organic material.

SECTION 6 Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. · 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. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb spill with sand, earth, inert material or vermiculite. ‣ Wipe up. ‣ Place in a suitable, labelled container for waste disposal.
Major Spills	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 LAND SPILL - SMALL cross-linked polymer - particulate | 1 | shovel | shovel | R, W, SS

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

LAND SPILL - MEDIUM

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

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

Safe handling

- Most acrylic monomers have low viscosity therefore pouring, material transfer and processing of these materials do not necessitate heating.
- Viscous monomers may require heating to facilitate handling. To facilitate product transfer from original containers, product must be heated to no more than 60 deg. C. (140 F.), for not more than 24 hours.
- Do NOT use localised heat sources such as band heaters to heat/ melt product.
- Do NOT use steam.
- Hot boxes or hot rooms are recommended for heating/ melting material. The hot box or hot room should be set a maximum temperature of 60 deg. C. (140 F.).
- Do NOT overheat - this may compromise product quality and /or result in an uncontrolled hazardous polymerisation.
- If product freezes, heat as indicated above and mix gently to redistribute the inhibitor. Product should be consumed in its entirety after heating/ melting; avoid multiple 'reheats' which may affect product quality or result in product degradation.
- Product should be packaged with inhibitor(s). Unless inhibited, product may polymerise, raising temperature and pressure, possibly rupturing container. Check inhibitor level periodically, adding to bulk material if needed. In addition, the product's inhibitor(s) require the presence of dissolved oxygen. Maintain, at a minimum, the original headspace in the product container and do NOT blanket or mix with oxygen-free gas as it renders the inhibitor ineffective. Ensure air space (oxygen) is present during product heating / melting.
- Store product indoors at temperatures greater than the product's freeing point (or greater than 0 deg. C. (32 F).) if no freezing point available and below 38 deg. C (100 F.).
- Avoid prolonged storage (longer than shelf-life) storage temperatures above 38 deg. C (100 F.).
- Store in tightly closed containers in a properly vented storage area away from heat, sparks, open flame, strong oxidisers, radiation and other initiators.
- Prevent contamination by foreign materials.
- Prevent moisture contact.
- Use only non-sparking tools and limit storage time. Unless specified elsewhere, shelf-life is 6 months from receipt.
- 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.

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	▸ 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	▸ 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.
Storage incompatibility	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 metals, 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 react like non-metals when they react with metals and act like metals when they react with non-metals. Epoxides: ▸ are highly reactive with acids, bases, and oxidising and reducing agents. ▸ react, possibly violently, with anhydrous metal chlorides, ammonia, amines and group 1 metals. ▸ may polymerise in the presence of peroxides or heat - polymerisation may be violent ▸ may react, possibly violently, with water in the presence of acids and other catalysts. ▸ Phenols are incompatible with strong reducing substances such as hydrides, nitrides, alkali metals, and sulfides. ▸ Avoid use of aluminium, copper and brass alloys in storage and process equipment. ▸ Heat is generated by the acid-base reaction between phenols and bases. ▸ Phenols are sulfonated very readily (for example, by concentrated sulfuric acid at room temperature), these reactions generate heat. ▸ Phenols are nitrated very rapidly, even by dilute nitric acid. ▸ Nitrated phenols often explode when heated. Many of them form metal salts that tend toward detonation by rather mild shock. for multifunctional acrylates: ▸ Avoid exposure to free radical initiators (peroxides, persulfates) , iron, rust, oxidisers, and strong acids and strong bases. ▸ Avoid heat, flame, sunlight, X-rays or ultra-violet radiation. ▸ Storage beyond expiration date, may initiate polymerisation. Polymerisation of large quantities may be violent (even explosive) ▸ Avoid strong acids, bases. Glycidyl 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
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
ammonium polyphosphate	Inhalation 18.06 mg/m³ (Systemic, Chronic) *Inhalation 4.45 mg/m³ (Systemic, Chronic)* * *Oral 1.28 mg/kg bw/day (Systemic, Chronic)* *	Not Available

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Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
bisphenol A diglycidyl ether	Dermal 0.75 mg/kg bw/day (Systemic, Chronic) Inhalation 4.93 mg/m³ (Systemic, Chronic) *Dermal 89.3 µg/kg bw/day (Systemic, Chronic) ** *Inhalation 0.87 mg/m³ (Systemic, Chronic) ** *Oral 0.5 mg/kg bw/day (Systemic, Chronic) **	0.006 mg/L (Water (Fresh)) 0.001 mg/L (Water - Intermittent release) 0.018 mg/L (Water (Marine)) 0.341 mg/kg sediment dw (Sediment (Fresh Water)) 0.034 mg/kg sediment dw (Sediment (Marine)) 0.065 mg/kg soil dw (Soil) 10 mg/L (STP) 11 mg/kg food (Oral)
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)
dipentaerythritol pentaacrylate	Dermal 0.5 mg/kg bw/day (Systemic, Chronic) Inhalation 1.76 mg/m³ (Systemic, Chronic)	0.013 mg/L (Water (Fresh)) 0.001 mg/L (Water - Intermittent release) 0.13 mg/L (Water (Marine)) 2.8 mg/kg sediment dw (Sediment (Fresh Water)) 0.28 mg/kg sediment dw (Sediment (Marine)) 0.22 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 Available	Not Available	Not Available	Not Available	Not Available	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
bisphenol A diglycidyl ether	39 mg/m3	430 mg/m3	2,600 mg/m3
bisphenol A diglycidyl ether	90 mg/m3	990 mg/m3	5,900 mg/m3

Ingredient	Original IDLH	Revised IDLH
phenol/ formaldehyde glycidyl ether copolymer	Not Available	Not Available
aluminium hydroxide	Not Available	Not Available
ammonium polyphosphate	Not Available	Not Available
bisphenol A diglycidyl ether	Not Available	Not Available
zinc borate	Not Available	Not Available
dipentaerythritol pentaacrylate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
phenol/ formaldehyde glycidyl ether copolymer	E	≤ 0.1 ppm
aluminium hydroxide	E	≤ 0.01 mg/m³
bisphenol A diglycidyl ether	E	≤ 0.1 ppm
zinc borate	E	≤ 0.01 mg/m³
dipentaerythritol pentaacrylate	E	≤ 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

CEL TWA: 1 mg/m3 [compare WEEL-TWA* for multifunctional acrylates (MFAs)]
(CEL = Chemwatch Exposure Limit)
Exposure to MFAs has been reported to cause contact dermatitis in humans and serious eye injury in laboratory animals. Exposure to some MFA-resin containing aerosols has also been reported to cause dermatitis. As no assessment of the possible effects of long-term exposure to aerosols was found, a conservative Workplace Environmental Exposure Level (WEEL) was suggested by the American Industrial Hygiene Association (AIHA).
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)

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8.2. Exposure controls

8.2.1. Appropriate engineering 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.)

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

- Chemical goggles.
- Full face shield may be required for supplementary but never for primary protection of eyes.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- 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.
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
General warning: Do NOT use latex gloves! Use only recommended gloves - using the wrong gloves may increase the risk:

Exposure condition Short time use; (few minutes less than 0.5 hour) Little physical stress	Use of thin nitrile rubber gloves: Nitrile rubber (0.1 mm) Excellent tactibility ('feel'), powder-free Disposable Inexpensive Give adequate protection to low molecular weigh acrylic monomers
Exposure condition Medium time use; less than 4 hours Physical stress (opening drums, using tools, etc.)	Use of medium thick nitrile rubber gloves Nitrile rubber, NRL (latex) free; <0.45 mm Moderate tactibility ('feel'), powder-free Disposable Moderate price Gives adequate protection for most acrylates up to 4 hours Do NOT give adequate protection to low molecular weight monomers at exposures longer than 1 hour
Exposure condition Long time Cleaning operations	Nitrile rubber, NRL (latex) free; >0.56 mm low tactibility ('feel'), powder free High price Gives adequate protection for most acrylates in combination with commonly used solvents up to 8 hours Do NOT give adequate protection to low molecular weight monomers at exposures longer than 1 hour Avoid use of ketones and acetates in wash-up solutions.

Where none of this gloves ensure safe handling (for example in long term handling of acrylates containing high levels of acetates and/ or ketones, use laminated multilayer gloves.
Guide to the Classification and Labelling of UV/EB Acrylates Third edition, 231 October 2007 - Cefic

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	-

Continued...

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	Off White

Physical state	Liquid	**Relative density (Water = 1)**	1.30
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	>20.5
Initial boiling point and boiling range (°C)	>150	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	113	**Taste**	Not Available
Evaporation rate	Not Available BuAC = 1	**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	Partly miscible	**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

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10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	No report of respiratory illness in humans as a result of exposure to multifunctional acrylates has been found. Similarly evidence of systemic damage does not appear to exist. 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. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion. 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. Male rats exposed to a single oral dose of bisphenol A diglycidyl ether (BADGE) at 750, 1000, and 2000 mg/kg/day showed a significantly increase in the number of immature and maturing sperm on the testis. There were no significant differences with respect to sperm head count, sperm motility, and sperm abnormality in the BADGE treatment groups 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). Inorganic polyphosphates are used extensively in domestic and industrial products. Rats fed 10% sodium trimetaphosphate for a month exhibited transient tubular necrosis; those given 10% sodium metaphosphate exhibited growth retardation; 10% sodium hexametaphosphate produced pale and swollen kidneys. Salts of this type appear to be hydrolysed in the bowel to produce phosphoric acid and systemic acidosis may result following absorption. Higher molecular weight species, absorbed from the alimentary canal, may produce hypocalcaemic tetany due to binding of ionised calcium by the absorbed phosphate. This is reported in at least one case following ingestion of sodium tripolyphosphate. 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.
Skin Contact	The material can produce chemical burns following direct contact with the skin. All multifunctional acrylates (MFA) produce skin discomfort and are known or suspected skin sensitisers. Aerosols generated in the industrial process are reported to produce dermatitis - vapours generated by the heat of milling may also occur in sufficient concentration to produce dermatitis. Because exposure to industrial aerosols of MFA may also include exposure to various resin systems, photo-initiators, solvents, hydrogen-transfer agents, stabilisers, surfactants, fillers and polymerisation inhibitors, toxic effects may arise due to a range of chemical actions. Bisphenol A diglycidyl ether (BADGE) may produce contact dermatitis characterised by erythema and oedema, with weeping followed by crusting and scaling. A liquid resin with a molecular weight of 350 produced severe skin irritation in rabbits when applied daily for 4 hours over 20 days. Following the initial contact there may be a discrete erythematous lesion, confined to the point of contact, which may persist for 48 hours to 10 days; the erythema may give way to a papular, vesicular rash with scaling. In animals uncured resin produces moderate ante-mortem depression, loss of body weight and diarrhoea. Local irritation, inflammation and death resulting from respiratory system depression are recorded. Higher molecular weight resins generally produce lower toxicity. 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 ▸ produces 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	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury.
Chronic	Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or 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

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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 n-butyl glycidyl ether, induced morphological transformation in mammalian cells in vitro. n-Butyl glycidyl ether induced micronuclei in mice in vivo following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations in vivo or chromosomal aberrations in animal cells in vitro. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in Drosophila. The glycidyl ethers were generally mutagenic to bacteria.
Bisphenol A diglycidyl ethers (BADGEs) produce sensitisation dermatitis characterised by a papular, vesicular eczema with considerable itching of the back of the hand, the forearm and face and neck. This lesion may persist for 10-14 days after withdrawal from exposure and recur immediately on re-exposure. This dermatitis may persist for longer periods following each exposure but is unlikely to become more intense. Lesions may develop a brownish colour and scaling occurs frequently. Lower molecular weight species produce sensitisation more readily.
In mice technical grades of bisphenol A diglycidyl ether produced epidermal tumours and a small increase in the incidence kidney tumours in males and of lymphoreticular/ haematopoietic tumours in females. Subcutaneous injection produced a small number of fibrosarcomas in rats.
BADGE is listed as an IARC Group 3 carcinogen, meaning it is 'not classifiable as to its carcinogenicity to humans'. Concern has been raised over this possible carcinogenicity because BADGE is used in epoxy resins in the lining of some tin cans for foodstuffs, and unreacted BADGE may end up in the contents of those cans.
In chronic animal studies inorganic polyphosphates produced growth inhibition, increased kidney weights (with calcium deposition and desquamation), bone decalcification, parathyroid hypertrophy and hyperplasia, inorganic phosphaturia, hepatic focal necrosis and alterations to the size of muscle fibres.
Inorganic phosphates are not genotoxic in bacterial systems nor are they carcinogenic in rats. No reproductive or developmental toxicity was seen in studies using rats exposed to sodium hexametaphosphate or sodium trimetaphosphate.
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.
There are reports of lung damage due to excessive inhalation of alumina dust. Ingestion of large amounts of aluminium hydroxide for prolonged periods may cause phosphate depletion, especially if phosphate intake is low. This may cause loss of appetite, muscle weakness, muscular disease and even softening of the bones. These effects have not been reported in people occupationally exposed to aluminium hydroxide.
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

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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.
All multifunctional acrylates (MFA) produce skin discomfort and are known or suspected skin sensitisers. Aerosols generated in the industrial process are reported to produce dermatitis - vapours generated by the heat of milling may also occur in sufficient concentration to produce dermatitis. Because exposure to industrial aerosols of MFA may also include exposure to various resin systems, photo-initiators, solvents, hydrogen-transfer agents, stabilisers, surfactants, fillers and polymerisation inhibitors, toxic effects may arise due to a range of chemical actions.

9200FR-A Flame Retardant Structural Epoxy Adhesive (Part A)

TOXICITY	IRRITATION
Not Available	Not Available

phenol/ formaldehyde glycidyl 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]

aluminium hydroxide

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]

ammonium polyphosphate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >3160 mg/kg[2]	Not Available
Inhalation(Rat) LC50; >4.85 mg/l4h[1]	
Oral(Rat) LD50; >=300<=2000 mg/kg[1]	

bisphenol A diglycidyl ether

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 2 mg/24h - SEVERE
Oral(Rat) LD50; >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit): 500 mg - mild
	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 *

dipentaerythritol pentaacrylate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[1]	Not Available

Continued...

	Oral(Rat) LD50; >2000 mg/kg[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*

9200FR-A Flame Retardant Structural Epoxy Adhesive (Part A)	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
PHENOL/ FORMALDEHYDE GLYCIDYL ETHER COPOLYMER	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.
BISPHENOL A DIGLYCIDYL ETHER	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. 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

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	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 n-butyl glycidyl ether, induced morphological transformation in mammalian cells in vitro. n-Butyl glycidyl ether induced micronuclei in mice in vivo following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations in vivo or chromosomal aberrations in animal cells in vitro. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in Drosophila. The glycidyl ethers were generally mutagenic to bacteria. 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 55badger The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
DIPENTAERYTHRITOL PENTAACRYLATE	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.
9200FR-A Flame Retardant Structural Epoxy Adhesive (Part A) & PHENOL/ FORMALDEHYDE GLYCIDYL ETHER COPOLYMER & BISPHENOL A DIGLYCIDYL ETHER & DIPENTAERYTHRITOL PENTAACRYLATE	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.
9200FR-A Flame Retardant Structural Epoxy Adhesive (Part A) & BISPHENOL A DIGLYCIDYL ETHER	In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg). **Reproductive and Developmental Toxicity**: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg. **Carcinogenicity:** IARC concluded that 'there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals.' Its overall evaluation was 'Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3). In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997). **Genotoxicity**: In S. typhimurium strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg). **Immunotoxicity:** Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs - **Consumer exposure** to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/ kg/body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weigh/day (male rats) from the 2-year carcinogenicity study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of

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	reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs. 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.
9200FR-A Flame Retardant Structural Epoxy Adhesive (Part A) & PHENOL/ FORMALDEHYDE GLYCIDYL 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.
9200FR-A Flame Retardant Structural Epoxy Adhesive (Part A) & DIPENTAERYTHRITOL PENTAACRYLATE	UV (ultraviolet)/ EB (electron beam) acrylates are generally of low toxicity UV/EB acrylates are divided into two groups; 'stenomeric' and 'eurymeric' acrylates. The first group consists of well-defined acrylates which can be described by a simple idealised chemical;they are low molecular weight species with a very narrow weight distribution profile. The eurymeric acrylates cannot be described by an idealised structure and may differ fundamentally between various suppliers; they are of relatively high molecular weigh and possess a wide weight distribution. Stenomeric acrylates are usually more hazardous than the eurymeric substances. Stenomeric acrylates are also well defined which allows comparison and exchange of toxicity data - this allows more accurate classification. The stenomerics cannot be classified as a group; they exhibit substantial variation. Based on the available oncogenicity data and without a better understanding of the carcinogenic mechanism the Health and Environmental Review Division (HERD), Office of Toxic Substances (OTS), of the US EPA previously concluded that all chemicals that contain the acrylate or methacrylate moiety (CH2=CHCOO or CH2=C(CH3)COO) should be considered to be a carcinogenic hazard unless shown otherwise by adequate testing. This position has now been revised and acrylates and methacrylates are no longer *de facto* carcinogens. Where no 'official' classification for acrylates and methacrylates exists, there has been cautious attempts to create classifications in the absence of contrary evidence. For example Monalkyl or monoarylesters of acrylic acids should be classified as R36/37/38 and R51/53 Monoalkyl or monoaryl esters of methacrylic acid should be classified as R36/37/38
ALUMINIUM HYDROXIDE & DIPENTAERYTHRITOL PENTAACRYLATE	No significant acute toxicological data identified in literature search.

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

Legend: ✘ *– Data either not available or does not fill the criteria for classification*
✔ *– Data available to make classification*

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

9200FR-A Flame Retardant Structural Epoxy Adhesive (Part A)

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

phenol/ formaldehyde glycidyl ether copolymer

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

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

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ammonium polyphosphate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	3.57mg/l	2
EC50	72h	Algae or other aquatic plants	>97.1mg/l	2
LC50	96h	Fish	>100mg/l	2
EC50	48h	Crustacea	>100mg/l	2

bisphenol A diglycidyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	9.4mg/l	2
LC50	96h	Fish	1.2mg/l	2
EC50	48h	Crustacea	1.1mg/l	2
NOEC(ECx)	504h	Crustacea	0.3mg/l	2

zinc borate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	40.2mg/l	2
LC50	96h	Fish	1.793mg/l	2
EC50	48h	Crustacea	1mg/l	2
NOEC(ECx)	768h	Fish	0.009mg/l	2
EC50	96h	Algae or other aquatic plants	15.4mg/l	2

dipentaerythritol pentaacrylate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	21mg/l	2
LC50	96h	Fish	8.9mg/l	2
EC50	48h	Crustacea	18mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	6.6mg/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*

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
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.
Liquid epoxy resins and some reactive diluents are not readily biodegradable, although its epoxy functional groups are hydrolysed in contact with water, they have the potential to bio-accumulate and are moderately toxic to aquatic organisms. They are generally classified as dangerous for the environment according to the European Union classification criteria.
Uncured solid resins on the other hand are not readily bio-available, not toxic to aquatic and terrestrial organisms, not readily biodegradable, but hydrolysable.They present no significant hazard for the environment.

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.
Ecotoxicity:
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

Continued...

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

* *Persistence and Bioaccumulation Regulations* (Canada 2000).
Substances containing unsaturated carbons are ubiquitous in indoor environments. They result from many sources (see below). Most are reactive with environmental ozone and many produce stable products which are thought to adversely affect human health.The potential for surfaces in an enclosed space to facilitate reactions should be considered.

Source of unsaturated substances	Unsaturated substances (Reactive Emissions)	Major Stable Products produced following reaction with ozone.
Occupants (exhaled breath, ski oils, personal care products)	Isoprene, nitric oxide, squalene, unsaturated sterols, oleic acid and other unsaturated fatty acids, unsaturated oxidation products	Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.
Soft woods, wood flooring, including cypress, cedar and silver fir boards, houseplants	Isoprene, limonene, alpha-pinene, other terpenes and sesquiterpenes	Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles
Carpets and carpet backing	4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters	Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonenal
Linoleum and paints/polishes containing linseed oil	Linoleic acid, linolenic acid	Propanal, hexanal, nonanal, 2-heptenal, 2-nonenal, 2-decenal, 1-pentene-3-one, propionic acid, n-butyric acid
Latex paint	Residual monomers	Formaldehyde
Certain cleaning products, polishes, waxes, air fresheners	Limonene, alpha-pinene, terpinolene, alpha-terpineol, linalool, linalyl acetate and other terpenoids, longifolene and other sesquiterpenes	Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and organic peroxides, acetone, benzaldehyde, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H)-furanone, 4-AMC, SOAs including ultrafine particles
Natural rubber adhesive	Isoprene, terpenes	Formaldehyde, methacrolein, methyl vinyl ketone
Photocopier toner, printed paper, styrene polymers	Styrene	Formaldehyde, benzaldehyde
Environmental tobacco smoke	Styrene, acrolein, nicotine	Formaldehyde, benzaldehyde, hexanal, glyoxal, N-methylformamide, nicotinaldehyde, cotinine
Soiled clothing, fabrics, bedding	Squalene, unsaturated sterols, oleic acid and other saturated fatty acids	Acetone, geranyl acetone, 6MHO, 40PA, formaldehyde, nonanal, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid
Soiled particle filters	Unsaturated fatty acids from plant waxes, leaf litter, and other vegetative debris; soot; diesel particles	Formaldehyde, nonanal, and other aldehydes; azelaic acid; nonanoic acid; 9-oxo-nonanoic acid and other oxo-acids; compounds with mixed functional groups (=O, -OH, and -COOH)
Ventilation ducts and duct liners	Unsaturated fatty acids and esters, unsaturated oils, neoprene	C5 to C10 aldehydes
'Urban grime'	Polycyclic aromatic hydrocarbons	Oxidized polycyclic aromatic hydrocarbons
Perfumes, colognes, essential oils (e.g. lavender, eucalyptus, tea tree)	Limonene, alpha-pinene, linalool, linalyl acetate, terpinene-4-ol, gamma-terpinene	Formaldehyde, 4-AMC, acetone, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H) furanone, SOAs including ultrafine particles
Overall home emissions	Limonene, alpha-pinene, styrene	Formaldehyde, 4-AMC, pinonaldehyde, acetone, pinic acid, pinonic acid, formic acid, benzaldehyde, SOAs including ultrafine particles

Abbreviations: 4-AMC, 4-acetyl-1-methylcyclohexene; 6MHQ, 6-methyl-5-heptene-2-one, 4OPA, 4-oxopentanal, SOA, Secondary Organic Aerosols
Reference: Charles J Weschler; Environmental Helath Perspectives, Vol 114, October 2006
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

Continued...

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 pH. 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-, B5O6(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.
In air ammonia is persistent whilst, in water, it biodegrades rapidly to nitrate, producing a high oxygen demand. Ammonia is strongly adsorbed to soil. Ammonia is non-persistent in water (half-life 2 days) and is moderately toxic to fish under normal temperature and pH conditions. Ammonia is harmful to aquatic life at low concentrations but does not concentrate in the food chain. Ammonium ions may be toxic to fish at 0.3 mg/l
Drinking Water Standards:
0.5 mg/l (UK max.)
1.5 mg/l (WHO Levels)
Soil Guidelines: none available.
Air Quality Standards: none available.

The principal problems of phosphate contamination of the environment relates to eutrophication processes in lakes and ponds. Phosphorus is an essential plant nutrient and is usually the limiting nutrient for blue-green algae. A lake undergoing eutrophication shows a rapid growth of algae in surface waters. Planktonic algae cause turbidity and flotation films. Shore algae cause ugly muddying, films and damage to reeds. Decay of these algae causes oxygen depletion in the deep water and shallow water near the shore. The process is self-perpetuating because anoxic conditions at the sediment/water interface causes the release of more adsorbed phosphates from the sediment. The growth of algae produces undesirable effects on the treatment of water for drinking purposes, on fisheries, and on the use of lakes for recreational purposes.
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

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

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
bisphenol A diglycidyl ether	MEDIUM (LogKOW = 3.8446)

12.4. Mobility in soil

Ingredient	Mobility
bisphenol A diglycidyl ether	LOW (KOC = 1767)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗

PBT Criteria fulfilled?	No
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

Continued...

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	▸ 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. Waste Management Production waste from epoxy resins and resin systems should be treated as hazardous waste in accordance with National regulations. Fire retarded resins containing halogenated compounds should also be treated as special waste. Accidental spillage of resins, curing agents and their formulations should be contained and absorbed by special mineral absorbents to prevent them from entering the environment. Contaminated or surplus product should not be washed down the sink, but preferably be fully reacted to form cross-linked solids which is non-hazardous and can be more easily disposed. Finished articles made from fully cured epoxy resins are hard, infusible solids presenting no hazard to the environment. However, finished articles from flame-retarded material containing halogenated resins should be considered hazardous waste, and disposed as required by National laws. Articles made from epoxy resins, like other thermosets, can be recycled by grinding and used as fillers in other products. Another way of disposal and recovery is combustion with energy recovery. 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. ▸ **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 bisphenol A diglycidyl ether and zinc borate)
14.3. **Transport hazard class(es)**	Class: 9 Subrisk: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Hazard identification (Kemler): 90 Classification code: M6 Hazard Label: 9 Special provisions: 274 335 375 601 Limited quantity: 5 L Tunnel Restriction Code: 3 (-)

Continued...

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	Environmentally hazardous substance, liquid, n.o.s. * (contains bisphenol A diglycidyl ether and zinc borate)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A97 A158 A197 A215 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 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 bisphenol A diglycidyl ether and zinc borate)
14.3. **Transport hazard class(es)**	IMDG Class: 9 IMDG Subrisk: Not Applicable
14.4. **Packing group**	III
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 bisphenol A diglycidyl ether and zinc borate)
14.3. **Transport hazard class(es)**	9 | Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Classification code: M6 Special provisions: 274; 335; 375; 601 Limited quantity: 5 L Equipment required: PP Fire cones number: 0

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
phenol/ formaldehyde glycidyl ether copolymer	Not Available
aluminium hydroxide	Not Available
ammonium polyphosphate	Not Available
bisphenol A diglycidyl ether	Not Available
zinc borate	Not Available
dipentaerythritol pentaacrylate	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type

Continued...

Product name	Ship Type
phenol/ formaldehyde glycidyl ether copolymer	Not Available
aluminium hydroxide	Not Available
ammonium polyphosphate	Not Available
bisphenol A diglycidyl ether	Not Available
zinc borate	Not Available
dipentaerythritol pentaacrylate	Not Available

SECTION 15 Regulatory information

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

phenol/ formaldehyde glycidyl ether copolymer is found on the following regulatory lists

Europe EC Inventory

aluminium hydroxide is found on the following regulatory lists

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

ammonium polyphosphate is found on the following regulatory lists

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

bisphenol A diglycidyl ether is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List | European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances | European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

Europe EC Inventory | International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

zinc borate is found on the following regulatory lists

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

dipentaerythritol pentaacrylate is found on the following regulatory lists

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

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 (phenol/ formaldehyde glycidyl ether copolymer; aluminium hydroxide; ammonium polyphosphate; bisphenol A diglycidyl ether; dipentaerythritol pentaacrylate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (phenol/ formaldehyde glycidyl ether copolymer; ammonium polyphosphate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (ammonium polyphosphate; bisphenol A diglycidyl ether; dipentaerythritol pentaacrylate)
Vietnam - NCI	Yes
Russia - FBEPH	No (dipentaerythritol pentaacrylate)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	13/09/2021
Initial Date	09/02/2018

Continued...

Full text Risk and Hazard codes

H360FD	May damage fertility. May damage the unborn child.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H410	Very toxic to aquatic life with long lasting effects.
H412	Harmful to aquatic life with long lasting effects.
H413	May cause long lasting harmful effects to aquatic life.

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 - Add UFI number and format changes to safety data sheet

end of SDS

9200FR-B Flame Retardant Structural 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: **15/09/2021**
Revision Date: **15/09/2021**
L.REACH.GB.EN

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

1.1. Product Identifier

Product name	9200FR-B
Synonyms	SDS Code: 9200FR-B, 9200FR-25ML, 9200FR-50ML | UFI:RPN0-U0YU-200C-TDVY
Other means of identification	Flame Retardant Structural Epoxy Adhesive (Part B)

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

Relevant identified uses	epoxy adhesive hardener for use with resins
Uses advised against	Not 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 - Hazardous to the Aquatic Environment Long-Term Hazard Category 2, H318 - Serious Eye Damage/Eye Irritation Category 1, H302 - Acute Toxicity (Oral) Category 4, H315 - Skin Corrosion/Irritation Category 2, H361 - Reproductive Toxicity Category 2, H317 - Sensitisation (Skin) 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	**Danger**

Hazard statement(s)

H411	Toxic to aquatic life with long lasting effects.
H318	Causes serious eye damage.
H302	Harmful if swallowed.
H315	Causes skin irritation.
H361	Suspected of damaging fertility or the unborn child.
H317	May cause an allergic skin reaction.

Page 1 continued...

Supplementary statement(s)

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.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

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 serious health damage*.

Cumulative effects may result following exposure*.

May produce discomfort of the respiratory system*.

Limited evidence of a carcinogenic effect*.

Possible respiratory sensitizer*.

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

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	**%[weight]**	**Name**	**Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567**	**Nanoform Particle Characteristics**
1.68683-29-4 2.Not Available 3.Not Available 4.Not Available	23	acrylonitrile/ butadiene copolymer amine terminated	Acute Toxicity (Inhalation) Category 4; H332, EUH032 [1]	Not Available
1.21645-51-2 2.244-492-7 3.Not Available 4.Not Available	22	aluminium hydroxide	Serious Eye Damage/Eye Irritation Category 2; H319, EUH066 [1]	Not Available
1.68333-79-9 2.269-789-9 3.Not Available 4.Not Available	20	ammonium polyphosphate	Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H413 [1]	Not Available
1.68410-23-1 2.Not Available 3.Not Available 4.Not Available	17	C18 fatty acid dimers/ tetraethylenepentamine polyamides	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3; H315, H318, H335 [1]	Not Available

Continued...

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
1.68082-29-1* 2.500-191-5 3.Not Available 4.01-2119972320-44-XXXX	7	tall oil/ triethylenetetramine polyamides	Serious Eye Damage/Eye Irritation Category 2; H319 [1]	Not Available
1.12767-90-7 2.235-804-2 3.Not Available 4.Not Available	6	zinc borate	Serious Eye Damage/Eye Irritation Category 2, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 1; H319, H360FD, H410 [1]	Not Available
1.112-24-3 2.203-950-6 3.612-059-00-5 4.Not Available	1	triethylenetetramine	Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3; H312, H314, H317, H412 [2]	Not Available
1.140-31-8 2.205-411-0 3.612-105-00-4 4.Not Available	1	N-aminoethylpiperazine	Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3; H302, H312, H314, H317, H412 [2]	Not Available
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*			

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 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.
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.
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.

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

for phosphate salts intoxication:

- All treatments should be based on observed signs and symptoms of distress in the patient. Consideration should be given to the possibility that overexposure to materials other than this product may have occurred.
- Ingestion of large quantities of phosphate salts (over 1.0 grams for an adult) may cause an osmotic catharsis resulting in diarrhoea and probable abdominal cramps. Larger doses such as 4-8 grams will almost certainly cause these effects in everyone. In healthy individuals most of the ingested salt will be excreted in the faeces with the diarrhoea and, thus, not cause any systemic toxicity. Doses greater than 10 grams hypothetically may cause systemic toxicity.
- Treatment should take into consideration both anionic and cation portion of the molecule.
- All phosphate salts, except calcium salts, have a hypothetical risk of hypocalcaemia, so calcium levels should be monitored.

Treat symptomatically.

For cyanide intoxication (and for certain nitriles which produce cyanide ion)

- Signs symptoms of acute cyanide poisoning reflect cellular hypoxia and are often non-specific.
- Cyanosis may be a late finding.
- *A bradycardic*, hypertensive and tachypneic patient suggests poisoning especially if CNS and cardiovascular depression subsequently occurs.
- Immediate attention should be directed towards assisted ventilation, administration of 100% oxygen, insertion of intravenous lines and institution of cardiac monitoring.
- Obtain an arterial blood gas immediately and correct any severe metabolic acidosis (pH below 7.15).
- Mildly symptomatic patients generally require supportive care alone. Nitrites should not be given indiscriminately - in all cases of moderate to severe poisoning, they should be given in conjunction with thiosulfate. As a temporizing measure supply amyl nitrite perles (0.2ml inhaled 30 seconds every minute) until intravenous lines for sodium nitrite are established. 10 ml of a 3% solution is administered over 4 minutes to produce 20% methaemoglobin in adults. Follow directly with 50 ml of 25% sodium thiosulfate, at the same rate, IV. If symptoms reappear or persist within 1/2-1 hour, repeat nitrite and thiosulfate at 50% of initial dose. As the mode of action involves the metabolic conversion of the thiosulfate to thiocyanate, renal failure may enhance thiocyanate toxicity.

Continued...

- Methylene blue is not an antidote. [Ellenhorn and Barceloux: Medical Toxicology]

If amyl nitrite intervention is employed then Medical Treatment Kits should contain the following:
- One box containing one dozen amyl nitrite ampoules
- Two sterile ampoules of sodium nitrite solution (10 mL of a 3% solution in each)
- Two sterile ampoules of sodium thiosulfate solution (50 mL of a 25% solution in each)
- One 10 mL sterile syringe. One 50 mL sterile syringe. Two sterile intravenous needles. One tourniquet.
- One dozen gauze pads.
- Latex gloves
- A 'Biohazard' bag for disposal of bloody/contaminated equipment.
- A set of cyanide instructions on first aid and medical treatment.

- Notes on the use of amyl nitrite:-
- AN is highly volatile and flammable - do not smoke or use around a source of ignition.
- If treating patient in a windy or draughty area provide some shelter or protection (shirt, wall, drum, cupped hand etc.) to prevent amyl nitrite vapour from being blown away. Keep ampoule upwind from the nose, the objective is to get amyl nitrite into the patients lungs.
- Rescuers should avoid AN inhalation to avoid becoming dizzy and losing competence.
- Lay the patient down. Since AN dilates blood vessels and lowers blood pressure, lying down will help keep patient conscious.
- **DO NOT** overuse - excessive use might put the patient into shock. Experience at DuPont plants has not shown any serious after-effects from treatment with amyl nitrite.

ADDITIONAL NOTES:
- Major medical treatment procedures may vary e.g. US (FDA method as recommended by DuPont) uses amyl nitrite as a methaemoglobin generator, followed by treatment with sodium nitrite and then sodium thiosulfate.

MODES OF ACTION: Amyl nitrite (AN) reacts with haemoglobin (HB) to form about 5% methaemoglobin (MHB). Sodium nitrite ($NaNO_2$) reacts with haemoglobin to form approximately 20-30% methaemoglobin. Methaemoglobin attracts cyanide ions (CN) from tissue and binds with them to become cyanmethaemoglobin (CNMHB). Sodium thiosulfate ($Na_2S_2O_3$) converts cyanmethaemoglobin to thiocyanate (HSCN) which is excreted by the kidneys. i.e. AN + HB = MHB $NaNO_2$ + HB = MHB CN + MHB = CNMHB $Na_2S_2O_3$ + CNMHB + O_2 = HSCN
- The administration of the antidote salts is intravenous in normal saline, Ringers lactate or other available IV fluid.
- European practice may use 4-dimethylaminophenol (DMAP) as a methaemoglobin generator. Also hydroxycobalamin (Vitamin B12a) is used. Hydroxycobalamin works by reacting with cyanide to form cyanocobalamin (Vitamin B12) which is excreted in the urine.
- European and Australian NOHSC (ASCC) propose dicobalt edetate (Kelocyanor) as antidote. This acts by chelating cyanide to form stable cobalticyanide, which is excreted in the urine. In all cases hyperbaric therapy may increase the efficiency of a cyanide antidote kit.

SECTION 5 Firefighting measures

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

5.2. Special hazards arising from the substrate or mixture

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

5.3. Advice for firefighters

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.
Fire/Explosion Hazard	Once acrylic fibre is ignited, an exothermic reaction can occur in the absence of oxygen with evolution of hazardous materials. ‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) nitrogen oxides (NOx) phosphorus oxides (POx) metal oxides other pyrolysis products typical of burning organic material.

SECTION 6 Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures
See section 8

6.2. Environmental precautions
See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage.

Continued...

	▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ If contamination of drains or waterways occurs, advise emergency services.

6.4. Reference to other sections

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

SECTION 7 Handling and storage

7.1. Precautions for safe handling

Safe handling	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ 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	▸ 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.
Storage incompatibility	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 metals, 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 react like non-metals when they react with metals and act like metals when they react with non-metals. ▸ Phosphates are incompatible with oxidising and reducing agents. ▸ Phosphates are susceptible to formation of highly toxic and flammable phosphine gas in the presence of strong reducing agents such as hydrides. ▸ Partial oxidation of phosphates by oxidizing agents may result in the release of toxic phosphorus oxides. ▸ Avoid strong acids, bases.

Continued...

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
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
ammonium polyphosphate	Inhalation 18.06 mg/m³ (Systemic, Chronic) *Inhalation 4.45 mg/m³ (Systemic, Chronic)* * *Oral 1.28 mg/kg bw/day (Systemic, Chronic)* *	Not Available
C18 fatty acid dimers/ tetraethylenepentamine polyamides	Dermal 1.1 mg/kg bw/day (Systemic, Chronic) Inhalation 3.9 mg/m³ (Systemic, Chronic) *Dermal 0.56 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 0.97 mg/m³ (Systemic, Chronic)* * *Oral 0.56 mg/kg bw/day (Systemic, Chronic)* *	0.004 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.041 mg/L (Water (Marine)) 411.01 mg/kg sediment dw (Sediment (Fresh Water)) 41.1 mg/kg sediment dw (Sediment (Marine)) 82.18 mg/kg soil dw (Soil) 3.14 mg/L (STP)
tall oil/ triethylenetetramine polyamides	Dermal 1.1 mg/kg bw/day (Systemic, Chronic) Inhalation 3.9 mg/m³ (Systemic, Chronic) *Dermal 0.56 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 0.97 mg/m³ (Systemic, Chronic)* * *Oral 0.56 mg/kg bw/day (Systemic, Chronic)* *	0.004 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.043 mg/L (Water (Marine)) 434.02 mg/kg sediment dw (Sediment (Fresh Water)) 43.4 mg/kg sediment dw (Sediment (Marine)) 86.78 mg/kg soil dw (Soil) 3.84 mg/L (STP)
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)
N-aminoethylpiperazine	Dermal 3.33 mg/kg bw/day (Systemic, Chronic) Inhalation 10.6 mg/m³ (Systemic, Chronic) Inhalation 15 µg/m³ (Local, Chronic) Inhalation 10.6 mg/m³ (Systemic, Acute) Inhalation 80 mg/m³ (Local, Acute)	0.058 mg/L (Water (Fresh)) 0.006 mg/L (Water - Intermittent release) 0.58 mg/L (Water (Marine)) 215 mg/kg sediment dw (Sediment (Fresh Water)) 21.5 mg/kg sediment dw (Sediment (Marine)) 1 mg/kg soil dw (Soil) 250 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Not Available	Not Available	Not Available	Not Available	Not Available	Not Available	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
C18 fatty acid dimers/ tetraethylenepentamine polyamides	30 mg/m3	330 mg/m3	2,000 mg/m3
triethylenetetramine	3 ppm	14 ppm	83 ppm
N-aminoethylpiperazine	6.4 mg/m3	71 mg/m3	420 mg/m3

Ingredient	Original IDLH	Revised IDLH
acrylonitrile/ butadiene copolymer amine terminated	Not Available	Not Available
aluminium hydroxide	Not Available	Not Available
ammonium polyphosphate	Not Available	Not Available
C18 fatty acid dimers/ tetraethylenepentamine polyamides	Not Available	Not Available
tall oil/ triethylenetetramine polyamides	Not Available	Not Available
zinc borate	Not Available	Not Available
triethylenetetramine	Not Available	Not Available
N-aminoethylpiperazine	Not Available	Not Available

Continued...

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
acrylonitrile/ butadiene copolymer amine terminated	E	≤ 0.1 ppm
aluminium hydroxide	E	≤ 0.01 mg/m³
C18 fatty acid dimers/ tetraethylenepentamine polyamides	E	≤ 0.1 ppm
tall oil/ triethylenetetramine polyamides	E	≤ 0.1 ppm
zinc borate	E	≤ 0.01 mg/m³
triethylenetetramine	E	≤ 0.1 ppm
N-aminoethylpiperazine	D	> 0.1 to ≤ 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 1,3-butadiene:
Odour Threshold Value: 0.45 ppm (detection), 1.1 ppm (recognition)
Exposure at or below the TLV-TWA is thought to provide significant protection for workers against systemic toxicity including cancer.
US rubber workers reached an accord in 1996 to limit exposure to 1 ppm with a 15-minute, short-term limit of 5 ppm. This TLV-TWA is currently under review in light of a report of animal carcinogenicity at 6.25 ppm.
Odour Safety Factor(OSF)
OSF=1.3 ('1,3-BUTADIENE')

Phenol and catechol produce similar toxic actions although catechol is considerably less toxic than phenol in animal tests following inhalation. By other exposure routes catechol is considerably more toxic. The TLV-TWA is thought to be protective against the significant risk of dermal and upper respiratory tract irritation and central nervous system effects, including convulsion.

TLV TWA: 0.001 mg/m3 (as total proteins) Inhalable fraction skin sensitiser
as rubber processing fume:
MEL-TWA: 0.6 mg/m3 as cyclohexane solubles [HSE, UK]
BRMA-TWA: 0.25 mg/m3 as cyclohexane solubles [BRMA Code of Practice]
Rubber fume is a complex and indeterminate mixture of substances and is defined as 'fume evolved in the mixing, milling and blending of natural rubber and synthetic polymers combined with chemicals, and in the processes which convert the resultant blend into finished products or parts thereof, and including any inspection procedures where fume continues to be evolved'.
'Fume' generally describes solid particles generated by chemical reactions, or by condensation from the gaseous state, usually after volatilisation from melted substances, and often accompanied by a chemical reaction such as oxidation or thermal breakdown.
Several chemical agents may occur in rubber fume which are experimental or animal carcinogens, however, given the number of chemicals used or formed during rubber making, difficulties arise in attributing a particular effect to a given exposure.
Stomach cancer has been associated with work in jobs early in the production line; lung and lower oesophagus cancer with all work processes; and lymphomas with jobs where co-exposure to solvents occurs. Other cancers have also been reported with liver tumours appearing as a secondary phenomenon. No no-effect levels have been determined.
Two studies showed no excess of bladder cancer in workers entering the industry after 1950: the excess risk before that date is thought to result from exposure to residual beta-naphthylamines previously used as anti-oxidants.
as rubber process dust:
MEL-TWA: 6 mg/m3 [HSE, UK]
Rubber process dust is a complex, variable mixture of particulates defined as 'dust arising in the stages of rubber manufacture where ingredients are handled, weighed, added to or mixed with natural or synthetic elastomers. It does not include dusts arising from the abrasion of cured rubber but occurs during the preparation of compounds of either synthetic or natural rubber.
There is some evidence that occupational exposure to rubber dusts produces an excess incidence of stomach cancer. HSE data concluded that there was a small but significant excess of stomach cancer associated with the initial processes in rubber manufacture. Stomach cancer shows a marked social class gradient, which may lead to an over-estimation of the risk.
One report from the USA stated that exposure in rubber processing areas produces pulmonary disease but this has not been supported by UK epidemiology nor reports from the industry.
No no-effect level has been determined. The MEL was considered appropriate because it was felt reasonably practical for industry to comply with this value.
Polyamide hardeners have much reduced volatility, toxicity and are much less irritating to the skin and eyes than amine hardeners. However commercial polyamides may contain a percentage of residual unreacted amine and all unnecessary contact should be avoided.

8.2. Exposure controls

8.2.1. Appropriate engineering 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.)

Continued...

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

Eye and face protection

- Chemical goggles.
- Full face shield may be required for supplementary but never for primary protection of eyes.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- 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.
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.

Continued...

	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.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
'Forsberg Clothing Performance Index'.
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
9200FR-B Flame Retardant Structural Epoxy Adhesive (Part B)

Material	CPI
BUTYL	A
NEOPRENE	C
NITRILE	C
PE/EVAL/PE	C
VITON	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as 'feel' or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	Light yellow

Physical state	Liquid	**Relative density (Water = 1)**	1.27
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available

Continued...

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)	122	**Taste**	Not Available
Evaporation rate	Not Available BuAC = 1	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	<0.001	**Gas group**	Not Available
Solubility in water	Partly miscible	**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

Inhaled	Inhalation of epoxy resin amine hardener vapours (including polyamines and amine adducts) may produce bronchospasm and coughing episodes lasting days after cessation of the exposure. Even faint traces of these vapours may trigger an intense reaction in individuals showing 'amine asthma'. The literature records several instances of systemic intoxications following the use of amines in epoxy resin systems. Excessive exposure to the vapours of epoxy amine curing agents may cause both respiratory irritation and central nervous system depression. Signs and symptoms of central nervous system depression, in order of increasing exposure, are headache, dizziness, drowsiness, and incoordination. In short, a single prolonged (measured in hours) or excessive inhalation exposure may cause serious adverse effects, including death. Exposure to toxic levels of butadiene has also produced chromosome damage. Human volunteers exposed at 2000-8000 ppm 1,3-butadiene for 6-8 hours showed slight smarting of the eyes, difficulty in focusing on instrument scales and a transient objection to butadiene odour. Characteristics of exposure include dry nose/mouth/throat, fatigue, headache, vertigo, nausea, narcosis, respiratory paralysis, and central nervous system depression. Very high concentrations may cause loss of consciousness or death. Repeated and prolonged exposure to 1,3-butadiene vapour may cause kidney and liver damage. Deep anaesthesia was induced in rabbits in 8 to 10 minutes at 200000 to 250000 ppm. Recovery from brief periods of anaesthesia occurred within two minutes of terminating the exposure. The intensity and time of exposure to hydrogen cyanide determines effects, symptoms. Short term inhalation of 20 to 40 ppm hydrogen cyanide may result in slight symptoms. Higher concentrations can cause death within minutes or hours; a concentration of 270 ppm can be fatal in one minute. Acute exposure to cyanides can cause death by cyanosis, asphyxia. At very low doses, symptoms of hydrogen cyanide exposure may be weakness, headaches, confusion, giddiness, dizziness, confusion, anxiety, nausea and vomiting. Normal blood pressure with rapid pulse is usual in mild cases. The respiratory rate varies with the intensity of exposure: rapid with mild exposure, or slow and gasping with severe exposure. Symptoms of mild exposure to hydrogen cyanide are completely reversed when exposure ceases. In severe cases, breathing is rapid and deep, then becomes slow and gasping. The victim may feel an irregular heartbeat and tightness in the chest. The skin appears bright pink or red. Fluid may fill the lungs and interfere with breathing. Unconsciousness, convulsions and death can quickly follow depending on the degree of exposure. Massive exposures may produce sudden collapse and death. concentration of 270 If death does not result, recovery is usually complete. There have however been a few reports of after-effects. These are similar to those seen in people deprived of oxygen, e.g. near-drowning victims

Continued...

	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.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion. Ingestion of amine epoxy-curing agents (hardeners) may cause severe abdominal pain, nausea, vomiting or diarrhoea. The vomitus may contain blood and mucous. If death does not occur within 24 hours there may be an improvement in the patients condition for 2-4 days only to be followed by the sudden onset of abdominal pain, board-like abdominal rigidity or hypo-tension; this indicates that delayed gastric or oesophageal corrosive damage has occurred. Inorganic polyphosphates are used extensively in domestic and industrial products. Rats fed 10% sodium trimetaphosphate for a month exhibited transient tubular necrosis; those given 10% sodium metaphosphate exhibited growth retardation; 10% sodium hexametaphosphate produced pale and swollen kidneys. Salts of this type appear to be hydrolysed in the bowel to produce phosphoric acid and systemic acidosis may result following absorption. Higher molecular weight species, absorbed from the alimentary canal, may produce hypocalcaemic tetany due to binding of ionised calcium by the absorbed phosphate. This is reported in at least one case following ingestion of sodium tripolyphosphate. 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.
Skin Contact	The material can produce chemical burns following direct contact with the skin. Skin contact is not thought to produce harmful health effects (as classified under EC Directives using animal models). Systemic harm, however, has been identified following exposure of animals by at least one other route and the material may still produce health damage following entry through wounds, lesions or abrasions. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur. Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions. Individuals exhibiting 'amine dermatitis' may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis. NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided. The diepoxide of butadiene (1,2:3,4-diepoxybutane), a probable metabolite, has been reported to be a mild skin tumourigen when applied topically to the skin of mice Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or 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.

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Acrylonitrile is a skin and respiratory sensitiser. Chronic exposures may produce severe liver inflammation. Chronic effects of occupational exposure include skin and eye irritation, nausea, vomiting, weakness, fatigue, jaundice, anaemia, leukocytosis, bilirubinaemia, increased serum thiocyanate concentrations, and hepatic and renal irritation. When administered orally (by gavage or in drinking water), acrylonitrile induced increased incidences of fore-stomach squamous cell papillomas, central nervous system microgliomas, mammary gland carcinomas and Zymbal gland carcinomas, forestomach papillomas and acanthomas, and central nervous system neoplasms in rats of both sexes. An epidemiological study of textile-plant workers potentially exposed to acrylonitrile and observed for 20 years, showed an increased incidence of cancers of the lung; further follow-up of this cohort revealed a continued excess of lung cancer, although during the actual 5-year follow-up period, there was no excess. This follow-up showed a significant excess of cancer of the prostate. *NTP*
In chronic animal studies inorganic polyphosphates produced growth inhibition, increased kidney weights (with calcium deposition and desquamation), bone decalcification, parathyroid hypertrophy and hyperplasia, inorganic phosphaturia, hepatic focal necrosis and alterations to the size of muscle fibres.
Inorganic phosphates are not genotoxic in bacterial systems nor are they carcinogenic in rats. No reproductive or developmental toxicity was seen in studies using rats exposed to sodium hexametaphosphate or sodium trimetaphosphate.
Amongst humans occupationally exposed to 1,3-butadiene several cancer sites with high statistically significant mortality ratios were identified. These included cancer of the testes, cancers of the digestive system (oesophagus, stomach, large intestine), larynx and Hodgkin's disease.
Exposure by rats to 1,3-butadiene gas at 1000 ppm/6hrs/day, 5 days /week (105 weeks for females and 111 weeks for males) caused significant increases in the incidence of tumours at various sites; mammary gland adenomas and sarcomas; uterine sarcomas; Zymbal gland carcinomas; thyroid adenomas and pancreatic adenomas. A high incidence of malignant lymphoma was found amongst a group of exposed rats in a second study
There are reports of lung damage due to excessive inhalation of alumina dust. Ingestion of large amounts of aluminium hydroxide for prolonged periods may cause phosphate depletion, especially if phosphate intake is low. This may cause loss of appetite, muscle weakness, muscular disease and even softening of the bones. These effects have not been reported in people occupationally exposed to aluminium hydroxide.
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.
The toxic properties of cyanide result from its ability to inhibit enzymes required for the respiration of cells within the body.
Chronic exposure to cyanides, at levels too low to produce clinical complaints, may cause dermatitis, itching, scarlet rash, perforation of nasal septum, throat irritation, muscular cramps, weight loss and enlargement of the thyroid gland. Workers with pre-existing CNS, heart and lung disorders are at significant risk.
A wide range of symptoms are thought to be caused by long-term, low-level (often less than 10 ppm) exposure to cyanides. Symptoms include persistent runny nose, weakness, dizziness, giddiness, headache, nausea, vomiting, abdominal pain, throat irritation, changes in taste and smell, muscle cramps, weight loss, flushing of the face, itching and irritation of the upper respiratory tract, throat and eyes and enlargement of the thyroid gland. These symptoms are not specific to cyanide exposure; therefore it has been difficult to prove that chronic cyanide toxicity exists.
Repeated minor contact with cyanides produces a characteristic scarlet rash with itching, papules (small, superficial raised spots on the skin), perforation of the nasal septum and possible sensitisation. Concerns have been expressed that low-level, long term exposures may result in damage to the nerves of the eye.
Chronic exposure to cyanides and certain nitriles may result in interference to iodine uptake by thyroid gland and its consequent enlargement. This occurs following metabolic conversion of the cyanide moiety to the less toxic thiocyanate which is excreted in the urine. Thyroid insufficiency may also occur as a result of metabolic conversion of cyanides to the corresponding thiocyanate..
A small amount of cyanide is excreted, unchanged, in the breath, sweat and urine.
Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

9200FR-B Flame Retardant Structural Epoxy Adhesive (Part B)

TOXICITY	IRRITATION
Not Available	Not Available

acrylonitrile/ butadiene copolymer amine terminated

TOXICITY	IRRITATION
dermal (rat) LD50: >3000 mg/kg[2]	Eye (rabbit): irritant *
Inhalation(Rat) LC50; 5.61 mg/L4h[2]	Skin: irritant, Draize Score 3.6*
Oral(Rat) LD50; >15380 mg/kg[2]	

aluminium hydroxide

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]

ammonium polyphosphate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >3160 mg/kg[2]	Not Available
Inhalation(Rat) LC50; >4.85 mg/l4h[1]	
Oral(Rat) LD50; >=300<=2000 mg/kg[1]	

C18 fatty acid dimers/ tetraethylenepentamine polyamides

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Not Available
Oral(Rat) LD50; >2000 mg/kg[1]	

tall oil/ triethylenetetramine polyamides

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Not Available
Oral(Rat) LD50; >2000 mg/kg[1]	

Continued...

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 *

triethylenetetramine

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 550 mg/kg[2]	Eye (rabbit):20 mg/24 h - moderate
Oral(Mouse) LD50; 38.5 mg/kg[2]	Eye (rabbit); 49 mg - SEVERE
	Skin (rabbit): 490 mg open SEVERE
	Skin (rabbit): 5 mg/24 SEVERE

N-aminoethylpiperazine

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 866 mg/kg[1]	Eye (rabbit): 20 mg/24h - mod
Oral(Rat) LD50; >1000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit): 0.1 mg/24h - mild
	Skin (rabbit): 5 mg/24h - SEVERE
	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*

ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED

* B.F. Goodrich
The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.
Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

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

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

ALUMINIUM HYDROXIDE

No significant acute toxicological data identified in literature search.

C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES

**[Valspar]

TRIETHYLENETETRAMINE

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
For alkyl polyamines:
The alkyl polyamines cluster consists of organic compounds containing two terminal primary amine groups and at least one secondary amine group.Typically these substances are derivatives of ethylenediamine, propylenediamine or hexanediamine. The molecular weight range for the entire cluster is relatively narrow, ranging from 103 to 232
Acute toxicity of the alkyl polyamines cluster is low to moderate via oral exposure and a moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin sensitisers in experimental animals. Repeated exposure in rats via the oral route indicates a range of toxicity from low to high hazard. Most cluster members gave positive results in tests for potential genotoxicity.
Limited carcinogenicity studies on several members of the cluster showed no evidence of carcinogenicity. Unlike aromatic amines, aliphatic amines are not expected to be potential carcinogens because they are not expected to undergo metabolic activation, nor would activated intermediates be stable enough to reach target macromolecules.
Polyamines potentiate NMDA induced whole-cell currents in cultured striatal neurons
Triethylenetetramine (TETA) is a severe irritant to skin and eyes and induces skin sensitisation.
TETA is of moderate acute toxicity: LD50(oral, rat) > 2000 mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure to saturated vapour via inhalation was tolerated without impairment. Exposure to to aerosol leads to reversible irritations of the mucous membranes in the respiratory tract.
Following repeated oral dosing via drinking water only in mice but not in rats at concentration of 3000 ppm there were signs of impairment. The NOAEL is 600 ppm [92 mg/kg bw (oral, 90 days)]. Lifelong dermal application to mice (1.2 mg/mouse) did not result in tumour formation.
There are differing results of the genetic toxicity for TETA. The positive results of the in vitro tests may be the result of a direct genetic action as well as a result of an interference with essential metal ions. Due to this uncertainty of the in vitro tests, the genetic toxicity of TETA has to be assessed on the basis of in vivo tests.
The in vivo micronucleus tests (i.p. and oral) and the SLRL test showed negative results.
There are no human data on reproductive toxicity (fertility assessment). The analogue diethylenetriamine had no effects on reproduction. TETA shows developmental toxicity in animal studies if the chelating property of the substance is effective. The NOEL is 830 mg/kg bw (oral).
Experience with female patients suffering from Wilson´s disease demonstrated that no miscarriages and no foetal abnormalities occur during treatment with TETA..
In rats, there are several studies concerning developmental toxicity. The oral treatment of rats with 75, 375 and 750 mg/kg resulted in no effects on dams and fetuses, except slight increased fetal body weight After oral treatment of rats with 830 or 1670 mg/kg bw only in the highest dose group increased foetal abnormalities in 27/44 fetus (69,2 %) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significant the fetal abnormalities of the highest dose group to 3/51 (6,5 % foetus. These findings suggest that the developmental toxicity is produced as a secondary consequence of the chelating properties of TETA.
Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

Continued...

N-AMINOETHYLPIPERAZINE	for piperazine: Exposure to piperazine and its salts has clearly been demonstrated to cause asthma in occupational settings. No NOAEL can be estimated for respiratory sensitisation (asthma). Although the LD50 levels indicate a relatively low level of oral acute toxicity (LD50 1-5 g/kg bw), signs of neurotoxicity may appear in humans after exposure to lower doses. Based on exposure levels of up to 3.4 mg/kg/day piperazine base and a LOAEL of 110 mg/kg, there is no concern for acute toxicity In pigs, piperazine is readily absorbed from the gastrointestinal tract, and the major part of the resorbed compound is excreted as unchanged piperazine during the first 48 hours. The principal route of excretion of piperazine and its metabolites is via urine, with a minor fraction recovered from faeces (16%). In humans the kinetics of the uptake and excretion of piperazine and its metabolites with urine appear to be roughly similar to that in the pig, and the nature and extent of conversion to metabolites has not been determined. Piperazine has demonstrated a low acute toxicity (LD50 = 1-5 g/kg bw) by the oral, dermal, and subcutaneous route of administration to rodents, whereas adequate inhalation toxicity data have not been found. However, there are findings of EEG (electroencephalogram) changes in 37% of 89 children administrated 90-130 mg/kg piperazine (two doses during one day), corroborated by a proposed GABA (gamma-aminobutyric acid) receptor agonism exerted by piperazine. Since clinical symptoms of neurotoxicity may occur after exposure to higher doses, a LOAEL of 110 mg/kg piperazine base for acute neurotoxicity in humans after acute exposure is proposed. Piperazine, as concentrated aqueous solution, has strongly irritating properties with regard to skin, and should be regarded as corrosive with respect to the eye. Exposure to piperazine and it salts has been demonstrated to cause allergic dermatitis as well as respiratory sensitisation in humans. As shown by the LLNA, piperazine has a sensitising potential in animals. Although piperazine is clearly sensitising, no NOAEL can be set for this effect from the present database. A NOAEL of 25 mg/kg/day of piperazine for liver toxicity in the beagle dog has been chosen after repeated exposure. A LOAEL of 30 mg/kg/day of piperazine for neurotoxicity is proposed based on documentation of (rare cases) of neurotoxicity from human clinical practice. Neurotoxicity also appears in other species (e.g., rabbits, dogs, cats, tigers, and horses), but not in rodents. For reproductive effects of piperazine, there is a NOAEL of 125 mg/kg/day for effects on fertility, i.e., reduced pregnancy index, decreased number of implantation sites, and decreased litter sizes in rats. The teratogenic properties have been investigated in rats and rabbits in adequate studies. In rabbit, such effects may be elicited at a dose level that is also toxic to the dam. The LOAEL is 94 mg/kg/day, and the NOAEL 42 mg/kg/day piperazine base (maternal and embryotoxic). In the rat study, there were decreases in body weight of both dams and offspring at the top dose (2,100 mg/kg/day piperazine base), but there were no signs of any malformations. The genotoxic properties have been investigated both *in vitro* (in the Ames test, in a nonstandard study on Saccharomyces cervisiae and in Chinese hamster ovary cells) and *in vivo*, in a micronuclei assay on mice, all with negative results. There are no solid indications of a carcinogenic effect of piperazine, neither in animal studies, nor from the investigation on humans. In view of lack of genotoxic action, it appears unlikely that piperazine poses a carcinogenic risk. There seems to be an additional cancer risk due to the formation of N-mononitrosopiperazine (NPZ) from piperazine. It is possible to calculate a hypothetical additional cancer risk posed by NPZ after exposure to piperazine, but the calculation would depend on several assumptions. We conclude that there seems to be an additional cancer risk due to the formation of NPZ from piperazine, and although it is difficult to estimate, it is probably small.
9200FR-B Flame Retardant Structural Epoxy Adhesive (Part B) & TRIETHYLENETETRAMINE & N-AMINOETHYLPIPERAZINE	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.
9200FR-B Flame Retardant Structural Epoxy Adhesive (Part B) & ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED	Occupational exposures in the rubber-manufacturing industry are carcinogenic to humans (Group 1).IARC Working Groups There is sufficient evidence in humans for the carcinogenicity of occupational exposures in the rubber-manufacturing industry. Occupational exposures in the rubber-manufacturing industry cause leukaemia, lymphoma, and cancers of the urinary bladder, lung, and stomach. Also, a positive association has been observed between occupational exposures in the rubber-manufacturing industry and cancers of the prostate, oesophagus, and larynx.IARC Working Group. The multiple genetic and cytogenetic effects observed among workers employed in the rubber-manufacturing industry provide strong evidence to support genotoxicity as one mechanism for the observed increase in cancer risks. However, due to the complexity and changing nature of the exposure mixture and the potential interactions between exposures in the rubber-manufacturing industry, other mechanisms are also likely to play a role. While it is clear that exposure to some agents in the rubber-manufacturing industry has been reduced over time, the results of recent cytogenetic studies continue to raise concerns about cancer risks. The rubber-manufacturing industry has used and still uses a wide variety of substances that belong to many different chemical categories, e.g. carbon black, aromatic amines, PAH, N-nitrosamines, mineral oils, other volatile organic compounds from curing fumes, trace amounts of monomers from synthetic rubber like 1,3-butadiene, acetonitrile, styrene, vinyl chloride, ethylene oxide, etc.. For this reason, it has been difficult to relate the observed cancer hazards in the rubber-manufacturing industry to exposure to specific chemicals.
9200FR-B Flame Retardant Structural Epoxy Adhesive (Part B) & C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES	For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides) The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented. The Fatty nitrogen-derived amides (FND amides) comprise four categories: Subcategory I: Substituted Amides Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components) Subcategory III: Imidazole Derivatives Subcategory IV: FND Amphoterics Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies. Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II. Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories. Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonella reverse mutation assay exist for all of the subcategories. Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II. In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30 chemicals tested.

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	For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole. Some typical applications of FND Amides are: masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers. The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health. The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals.
9200FR-B Flame Retardant Structural Epoxy Adhesive (Part B) & ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED & C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES & TRIETHYLENETETRAMINE & N-AMINOETHYLPIPERAZINE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED & C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES & N-AMINOETHYLPIPERAZINE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
TRIETHYLENETETRAMINE & N-AMINOETHYLPIPERAZINE	Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds. Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines. In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper The material may produce severe 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.

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

Legend: ✘ *– Data either not available or does not fill the criteria for classification*
✔ *– Data available to make classification*

11.2.1. Endocrine Disruption Properties

Not Available

SECTION 12 Ecological information

12.1. Toxicity

9200FR-B Flame Retardant Structural Epoxy Adhesive (Part B)

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

acrylonitrile/ butadiene copolymer amine terminated

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

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

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	EC50	96h	Algae or other aquatic plants	0.46mg/l	2

ammonium polyphosphate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	3.57mg/l	2
EC50	72h	Algae or other aquatic plants	>97.1mg/l	2
LC50	96h	Fish	>100mg/l	2
EC50	48h	Crustacea	>100mg/l	2

C18 fatty acid dimers/ tetraethylenepentamine polyamides

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	1.25mg/l	2
EC50	72h	Algae or other aquatic plants	4.11mg/l	2
LC50	96h	Fish	7.07mg/l	2
EC50	48h	Crustacea	5.18mg/l	2

tall oil/ triethylenetetramine polyamides

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	0.5mg/l	2
EC50	72h	Algae or other aquatic plants	4.34mg/l	2
LC50	96h	Fish	7.07mg/l	2
EC50	48h	Crustacea	7.07mg/l	2

zinc borate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	40.2mg/l	2
LC50	96h	Fish	1.793mg/l	2
EC50	48h	Crustacea	1mg/l	2
NOEC(ECx)	768h	Fish	0.009mg/l	2
EC50	96h	Algae or other aquatic plants	15.4mg/l	2

triethylenetetramine

Endpoint	Test Duration (hr)	Species	Value	Source
ErC50	72h	Algae or other aquatic plants	2.5mg/l	1
LC50	96h	Fish	180mg/l	1
EC50	72h	Algae or other aquatic plants	2.5mg/l	1
EC50	48h	Crustacea	31.1mg/l	1
BCF	1008h	Fish	<0.5	7
EC10(ECx)	72h	Algae or other aquatic plants	0.67mg/l	1

N-aminoethylpiperazine

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	495mg/l	1
LC50	96h	Fish	>100mg/l	2
EC50	48h	Crustacea	32mg/l	1
NOEC(ECx)	48h	Crustacea	18mg/l	1

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*

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
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.
A team analyzed the effects of washing a number of synthetic materials at different temperatures in domestic washing machines, using different combinations of detergents, to quantify the microfibres shed. They found that acrylic was responsible for releasing nearly 730,000 tiny synthetic particles (microplastics) per wash, five times more than polyester-cotton blend fabric, and nearly 1.5 times as many as pure polyester.
Fiber wastes has been found in sediments along shorelines around the world. predicted. They have been found to be tiny, synthetic, and all over the coastline, with the greatest concentration near sewage outflows. Of the man-made material found on the shoreline, 85% were microfibers and matched the types of material (such as nylon and acrylic) used in clothing.
The alkali metal cyanides (and other metal cyanides) are very soluble in water. As a result, they readily dissociate into their respective anions and cations when released into water. Depending on the pH of the water, the resulting cyanide ion may then form hydrogen cyanide or react with various metals in natural water. The proportion of hydrogen cyanide formed from soluble cyanides increases as the water pH decreases. At pH <7, >99% of the cyanide ions in water are converted to hydrogen cyanide. As the pH increases, cyanide ions in the water may form complex metallocyanides in the presence of excess cyanides; however, if metals are prevalent, simple metal cyanides are formed. Volatilization is the dominant mechanism for the removal of free cyanide. At pH >9.2, most of the free cyanide should exist as HCN, a volatile form of cyanide. Wide variations in the rate of volatilization are expected since this process is affected by a number of parameters such as temperature, pH, wind speed, and cyanide concentration. Volatilization of free cyanide from concentrated solutions is most effective under conditions of high temperatures, high dissolved oxygen levels, and at increased concentrations of atmospheric carbon dioxide
Unlike water-soluble alkali metal cyanides, insoluble metal cyanides are not expected to degrade to hydrogen cyanide. Cyanide occurs most commonly as hydrogen cyanide in water,

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although it can also occur as the cyanide ion, alkali and alkaline earth metal cyanides (potassium cyanide, sodium cyanide, calcium cyanide), relatively stable metallocyanide complexes (ferricyanide complex [Fe(CN)6]-3), moderately stable metallocyanide complexes (complex nickel and copper cyanide), or easily decomposable metallocyanide complexes (zinc cyanide [Zan(CN)2], cadmium cyanide [Cd(CN)2]). Oxidation, hydrolysis, and photolysis (photodegradation) are the three predominant chemical processes that may cause loss of simple cyanides in aquatic media.
Certain cyanides are oxidised to isocyanates by strong oxidising agents; the isocyanates may be further hydrolysed to ammonia and carbon dioxide. However, it has not yet been determined whether such oxidation and subsequent hydrolysis of isocyanate is a significant fate process in natural waters known to contain peroxy radicals. In water, hydrogen cyanide and cyanide ion exist in equilibrium with their relative concentrations primarily dependent on pH and temperature. At pH <8, >93% of the free cyanide in water will exist as undissociated hydrogen cyanide. Hydrogen cyanide can be hydrolysed to formamide, which is subsequently hydrolysed to ammonium and formate ions. However, the relatively slow rates of hydrolysis reported for hydrogen cyanide in acidic solution and of cyanides under alkaline conditions indicate that hydrolysis is not competitive with volatilisation and biodegradation for removal of free cyanide from ambient waters At pH <9.2, most of the free cyanide in solution should exist as hydrogen cyanide, a volatile cyanide form. On the basis of Henry's law constant and the volatility characteristics associated with various ranges of Henry's law constant, volatilization is a significant and probably dominant fate process for hydrogen cyanide in surface water. The most common alkali metal cyanides (e.g., sodium and potassium cyanide) may also be lost from surface water primarily through volatilization; whereas, the sparingly soluble metal cyanides such as copper (I) cyanide are removed from water predominantly by sedimentation and biodegradation. Because volatilisation is not an important fate process for cyanide in groundwater, cyanide would be expected to persist for considerably longer periods of time in underground aquifers than in surface water.
The significance of photolysis in the fate of cyanides in water has not been fully investigated. Hydrogen cyanide and cyanide ions in aqueous solution have been found to be very resistant to photolysis by natural sunlight, except under heterogeneous photocatalytic conditions. Photocatalytic oxidation may not be significant in natural waters, however, because of significant light reduction at increasingly greater depths. In clear water or at water surfaces, some metallocyanides, such as ferrocyanides and ferricyanides, may decompose to the cyanide ion by photodissociation and subsequently form hydrogen cyanide.
Biodegradation is an important transformation process for cyanide in natural surface waters, and is dependent on such factors as cyanide concentrations, pH, temperature, availability of nutrients, and acclimation of microbes. Although the cyanide ion is toxic to microorganisms at concentrations as low as 5-10 mg/L, acclimation increases tolerance to this compound. Mixed microorganisms in sewage sludge or activated sludge acclimated to cyanide also significantly biodegrade concentrations <=100 mg/L of most simple and complex cyanides. It is known that there is a natural attentuation of the cyanide ion and thiocyanide concentrations in waste waters, for example those obtained gold mill tails, that is due the acclimation of indigenous microflora in the tailings. A number of microorganisms have been identified that are capable of uptake, conversion, sorption, and/or precipitation of the cyanide ion, cyanate, and thiocyanate, including species of the genera, *Actinomyces, Alcaligenes, Arthrobacter, Bacillus, Micrococcus, Neisseria, Paracoccus, Pseudomonas*, and *Thiobacillus*. Some of these species, for example *Pseudomonas*, are capable of using the cyanide ion and thiocyanate as the sole source of carbon and nitrogen and therefore, are particularly effective at cyanide degradation. In fact, *Pseudomonas* is the basis of commercial applications for degrading the cyanide ion to ammonia and carbonate in waste waters generated in mining operations that use the cyanide ion to leach gold and other precious metals for low-grade ores. Sulfur transferases such as rhodanese are involved in substitution reactions that result in the conversion of the cyanide ion to the less toxic thiocyanate, whereas pyridoxal phosphate enzymes are involved in substitution/addition reactions that result in production of nitrile derivatives of a-amino acids. These organic nitriles may then be ultimately degraded via enzyme catalysed hydrolysis to either the corresponding amino acid and ammonia or the carboxylic acid and ammonia. The cyanide hydratase and cyanidase enzymes catalyse the hydrolysis of the cyanide ion to formamide or formic acid and ammonia, respectively In soil, cyanide present at low concentrations would biodegrade under aerobic conditions with the initial formation of ammonia, which would be converted to nitrite and nitrate in the presence of nitrifying bacteria. Under anaerobic conditions, the cyanides ion will denitrify to gaseous nitrogen. Upper limits of 200 and 2 ppm (mg/kg CN–), respectively, have been reported for uninhibited aerobic and anaerobic biodegradation of cyanide in soil; however, these limits have not been confirmed in other studies. Cyanide ions in soil are not involved in oxidation-reduction reactions but may undergo complexation reactions with metal ions in soil
Cyanides are sorbed by various natural media, including clays, biological solids and sediments Hydrogen cyanide and the alkali metal cyanides are not likely to be strongly sorbed onto sediments and suspended solids because of their high water solubilities. Soluble metal cyanides may show somewhat stronger sorption than hydrogen cyanide, with the extent of sorption increasing with decreasing pH and increasing iron oxide, clay, and organic material contents of sediment and suspended solids. However, sorption is probably insignificant even for metal cyanides when compared to volatilisation and biodegradation. Cyanides are fairly mobile in soil. Mobility is lowest in soils with low pH and high concentrations of free iron oxides, positively charged particles, and clays (e.g., chlorite, kaolin, gibbsite), and highest in soils with high pH, high concentrations of free CaCO3 and negatively charged particles, and low clay content. Although cyanide has a low soil sorption capability, it is usually not detected in groundwater, probably because of fixation by trace metals through complexation or transformation by soil microorganisms. In soils where cyanide levels are high enough to be toxic to microorganisms (i.e., landfills, spills), this compound may leach into groundwater. Leaching of cyanide into a shallow aquifer has been demonstrated. Volatilisation of hydrogen cyanide would be a significant loss mechanism for cyanides from soil surfaces at a pH <9.2.
Most cyanide in the atmosphere exists almost entirely as hydrogen cyanide gas, although small amounts of metal cyanides may be present as particulate matter in the air. Hydrogen cyanide is very resistant to photolysis at wavelengths of normal sunlight. The most important reaction of hydrogen cyanide in air is the reaction with photochemically-generated hydroxyl radicals and subsequent rapid oxidation to carbon monoxide (CO) and nitric oxide (NO); photolysis and reaction with ozone are not important transformation processes, and reaction with singlet oxygen is not a significant transformation process except at stratospheric altitudes where singlet oxygen is present in significant concentrations. The rate of hydroxyl radical reaction with hydrogen cyanide in the atmosphere depends on the altitude, and the rate of the reaction is at least an order of magnitude faster at lower tropospheric altitudes (0–8 km) than at upper tropospheric altitudes (10–12 km). Based on a reaction rate constant of 3x10-14 cm3/(molecule-sec) at 25 °C and assuming an average hydroxyl radical concentration of 5x105 molecules/cm3, the residence time for the reaction of hydrogen cyanide vapor with hydroxyl radicals in the atmosphere is approximately 2 years
There is some evidence that certain metal cyanide complexes bioaccumulate in aquatic organisms. Fish from water with soluble silver and copper cyanide complexes were found to have metal cyanides in their tissues at concentrations ranging up to 168 and 304 µg/g, respectively (wet or dry weight not specified). It is difficult to evaluate the toxicologic significance of bioaccumulation of metal cyanide complexes because these compounds are much less toxic than soluble hydrogen cyanide, sodium cyanide, or potassium cyanide. There is no evidence of biomagnification of cyanides in the food chain. Accumulation of cyanide in food webs is not expected, considering the rapid detoxification of cyanide by most species and the lethal effects of large doses of cyanide
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 butadiene:
Kow: 1.99
Koc : 72-228
Half-life (hr) air : 4.9
Henry's Pa m3 /mol: 2.57
Henry's atm m3 /mol: 7.24E-02
BCF : 19.1
Environmental fate:
The high volatility of this compound suggests that it will partition predominantly to the atmospheric compartment, where it is not expected to be adsorbed to particulate matter to any significant extent.
Terrestrial Fate: If spilled on land, 1,3-butadiene will predominately volatilise very rapidly due to its very low boiling point. Dissolved in water, it may leach through soil into ground water due to its high water solubility and low estimated soil adsorption coefficient. It will not appreciably hydrolyse but may be subject to biodegradation based on screening tests. 1,3-Butadiene is expected to volatilize rapidly from either moist or dry soil to the atmosphere. This follows from the estimated lack of any appreciable adsorption to soil, and consideration of 1,3-butadiene's calculated Henry's law constant for moist soil or its vapor pressure, 2,100 mm Hg at 25 C,for dry soil. Both values suggest a rapid rate of volatilisation from their respective media. The calculated soil adsorption coefficient of 288 suggests that 1,3-butadiene may display moderate mobility in soil. However, the expected rapid rate of volatilisation and the possibility of rapid degradation in soil suggest that there is little potential for 1,3-butadiene to leach into groundwater. Methane-utilizing bacteria isolated from the soil of an oil refinery epoxidised 1,3-butadiene under aerobic conditions
Aquatic Fate: When released into water, 1,3-butadiene will volatilise rapidly with a half-life estimated to be several hours. It will not hydrolyse appreciably, but may be subject to biodegradation, based on screening tests.
Atmospheric Fate: Butadiene is a reactive, electron-rich chemical that is expected to undergo rapid reactions with the electrophilic oxidants typically present in the atmosphere: ozone, photochemically produced hydroxyl radicals, nitrate radicals, and molecular oxygen. Among these, the most rapid reaction in the atmosphere is with photochemically produced hydroxyl radicals. The atmospheric destruction of 1,3-butadiene by photo-initiated processes has been established empirically by early studies There are four gas-phase pathways that can destroy 1,3-butadiene in the atmosphere. Depending on local conditions, any one or all of these reactions may occur. Destruction of atmospheric 1,3-butadiene by the gas-phase reaction with photochemically produced hydroxyl radicals is expected to be the dominant photo-initiated pathway. Destruction by nitrate radicals is expected to be a significant night-time process in urban areas.
Reaction with hydroxyl radicals is the dominant removal mechanism, with an estimated half-life of several hours. Reaction with ozone and nitrate radicals may also contribute to the degradation of the chemical. Polluted urban atmospheres increase the rate of degradation somewhat during daylight hours as suggested by the detection of the highest atmospheric levels of the chemical in the early morning hours. Acetaldehyde and acrolein have been identified as products of photooxidation. Washout may contribute to removal of 1,3-butadiene from the atmosphere; however, evaporation from the rain may be rapid and the compound returned to the atmosphere relatively quickly unless it leaches into the soil.

Continued...

Biodegradation: No data concerning the biodegradation of 1,3-butadiene in natural systems could be found in the literature. 1,3-Butadiene was listed in a group of chemicals which should be biodegraded by biological sewage treatment, as long as suitable acclimatization is achieved. Screening tests suggest that 1,3-butadiene may be biodegradable in the environment with 1,2-epoxybutene being a potential product .
Soil Adsorption/Mobility: The range of estimated adsorption coefficients for 1,3-butadiene from the soils and sediments is 72-228 based on its octanol/water partition coefficient or its water solubility and would therefore not be expected to appreciably adsorb in soils and sediments.
Volatilization from Water/Soil: Using the Henry's Law constant, the estimated half-life for evaporation of 1,3-butadiene from a river 1 m deep with a 1 m/sec current and a 3 m/sec wind is 3.8 hours . Due to its low boiling point, 1,3-butadiene would be expected to rapidly evaporate from soils.
Ecotoxicity:
Fish LC50 (24 h): 71.5 mg/L
1,3-Butadiene is moderately toxic to aquatic life in the short term and slightly toxic in the long term. There is not enough information to predict additional short or long-term effects of 1,3-butadiene on plants, birds, or other animals. 1,3-Butadiene is not expected to accumulate in fish. Animal studies have reported development effects such as skeletal abnormalities and decreased foetal weights, and reproductive effects, including an increased incidence of shrinkage of the ovaries and testicles. Animal studies have also reported tumours at a variety of sites from inhalation of 1,3-butadiene.
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 pH. 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-, B5O6(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.
In air ammonia is persistent whilst, in water, it biodegrades rapidly to nitrate, producing a high oxygen demand. Ammonia is strongly adsorbed to soil. Ammonia is non-persistent in water (half-life 2 days) and is moderately toxic to fish under normal temperature and pH conditions. Ammonia is harmful to aquatic life at low concentrations but does not concentrate in the food chain. Ammonium ions may be toxic to fish at 0.3 mg/l
Drinking Water Standards:
0.5 mg/l (UK max.)
1.5 mg/l (WHO Levels)
Soil Guidelines: none available.
Air Quality Standards: none available.

The principal problems of phosphate contamination of the environment relates to eutrophication processes in lakes and ponds. Phosphorus is an essential plant nutrient and is usually the limiting nutrient for blue-green algae. A lake undergoing eutrophication shows a rapid growth of algae in surface waters. Planktonic algae cause turbidity and flotation films. Shore algae cause ugly muddying, films and damage to reeds. Decay of these algae causes oxygen depletion in the deep water and shallow water near the shore. The process is self-perpetuating because anoxic conditions at the sediment/water interface causes the release of more adsorbed phosphates from the sediment. The growth of algae produces undesirable effects on the treatment of water for drinking purposes, on fisheries, and on the use of lakes for recreational purposes.
Soil Guidelines: Dutch Criteria:

free cyanide: 1 mg/kg (target)

20 mg/kg (intervention)

complex cyanide (pH 5): 5 mg/kg (target)

50 mg/kg (intervention)

Air Quality Standards: no safe guidelines recommended due to carcinogenic properties.
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

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

Continued...

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
triethylenetetramine	LOW (BCF = 5)
N-aminoethylpiperazine	LOW (LogKOW = -1.5677)

12.4. Mobility in soil

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

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗

PBT Criteria fulfilled?	No
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

Product / Packaging disposal	▸ 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. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.
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 C18 fatty acid dimers/ tetraethylenepentamine polyamides)

Continued...

14.3. **Transport hazard class(es)**	Class: 9 Subrisk: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Hazard identification (Kemler): 90 Classification code: M6 Hazard Label: 9 Special provisions: 274 335 375 601 Limited quantity: 5 L 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 C18 fatty acid dimers/ tetraethylenepentamine polyamides)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A97 A158 A197 A215 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 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 C18 fatty acid dimers/ tetraethylenepentamine polyamides)
14.3. **Transport hazard class(es)**	IMDG Class: 9 IMDG Subrisk: Not Applicable
14.4. **Packing group**	III
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 C18 fatty acid dimers/ tetraethylenepentamine polyamides)
14.3. **Transport hazard class(es)**	9 | Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Classification code: M6 Special provisions: 274; 335; 375; 601 Limited quantity: 5 L Equipment required: PP Fire cones number: 0

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

Not Applicable

Continued...

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

Product name	Group
acrylonitrile/ butadiene copolymer amine terminated	Not Available
aluminium hydroxide	Not Available
ammonium polyphosphate	Not Available
C18 fatty acid dimers/ tetraethylenepentamine polyamides	Not Available
tall oil/ triethylenetetramine polyamides	Not Available
zinc borate	Not Available
triethylenetetramine	Not Available
N-aminoethylpiperazine	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
acrylonitrile/ butadiene copolymer amine terminated	Not Available
aluminium hydroxide	Not Available
ammonium polyphosphate	Not Available
C18 fatty acid dimers/ tetraethylenepentamine polyamides	Not Available
tall oil/ triethylenetetramine polyamides	Not Available
zinc borate	Not Available
triethylenetetramine	Not Available
N-aminoethylpiperazine	Not Available

SECTION 15 Regulatory information

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

acrylonitrile/ butadiene copolymer amine terminated is found on the following regulatory lists

Not Applicable

aluminium hydroxide is found on the following regulatory lists

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

ammonium polyphosphate is found on the following regulatory lists

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

C18 fatty acid dimers/ tetraethylenepentamine polyamides is found on the following regulatory lists

Not Applicable

tall oil/ triethylenetetramine polyamides is found on the following regulatory lists

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)

triethylenetetramine is found on the following regulatory lists

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

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

N-aminoethylpiperazine is found on the following regulatory lists

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

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

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

Continued...

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (acrylonitrile/ butadiene copolymer amine terminated; aluminium hydroxide; ammonium polyphosphate; C18 fatty acid dimers/ tetraethylenepentamine polyamides; tall oil/ triethylenetetramine polyamides; triethylenetetramine; N-aminoethylpiperazine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (acrylonitrile/ butadiene copolymer amine terminated; C18 fatty acid dimers/ tetraethylenepentamine polyamides)
Japan - ENCS	No (acrylonitrile/ butadiene copolymer amine terminated; ammonium polyphosphate; tall oil/ triethylenetetramine polyamides)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (acrylonitrile/ butadiene copolymer amine terminated; ammonium polyphosphate)
Vietnam - NCI	Yes
Russia - FBEPH	No (acrylonitrile/ butadiene copolymer amine terminated; C18 fatty acid dimers/ tetraethylenepentamine polyamides; tall oil/ triethylenetetramine polyamides)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	15/09/2021
Initial Date	11/02/2018

Full text Risk and Hazard codes

H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.
H360FD	May damage fertility. May damage the unborn child.
H410	Very toxic to aquatic life with long lasting effects.
H412	Harmful to aquatic life with long lasting effects.
H413	May cause long lasting harmful effects to aquatic life.

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

Continued...

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 - Add UFI number and format changes to safety data sheet