



## 834B-A Black Flame Retardant Epoxy

MG Chemicals UK Limited

Version No: A-1.02

Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date: 10/05/2019

Revision Date: 26/03/2020

L.REACH.GBR.EN

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

#### 1.1. Product Identifier

Product name	834B-A
Synonyms	SDS Code: 834B-Part A; 834B-375ML, 834B-2.7L, 834B-10.8L, 834B-60L
Other means of identification	Black Flame Retardant Epoxy

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

Relevant identified uses	Resin for use with epoxy hardener
Uses advised against	Not Applicable

#### 1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 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)	Not Available
Emergency telephone numbers	+(44) 20 35147487	Not Available
Other emergency telephone numbers	+(0) 800 680 0425	Not Available

### SECTION 2 HAZARDS IDENTIFICATION

#### 2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	H411 - Chronic Aquatic Hazard Category 2, H315 - Skin Corrosion/Irritation Category 2, H319 - Eye Irritation Category 2, H361 - Reproductive Toxicity Category 2, H317 - Skin Sensitizer Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

#### 2.2. Label elements

Hazard pictogram(s)	
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SIGNAL WORD **WARNING**

#### Hazard statement(s)

H411	Toxic to aquatic life with long lasting effects.
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.

#### Supplementary statement(s)

EUH205	Contains epoxy constituents. May produce an allergic reaction.
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## Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

## Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.

## Precautionary statement(s) Storage

P405	Store locked up.
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## Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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## 2.3. Other hazards

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

## SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

## 3.1. Substances

See 'Composition on ingredients' in Section 3.2

## 3.2. Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.1675-54-3 2.216-823-5 3.603-073-00-2 603-074-00-8 4.01-2119456619-26-XXXX	30	<u>bisphenol A diglycidyl ether</u>	Eye Irritation Category 2, Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H319, H317, H315 [2]
1.21645-51-2 2.244-492-7 3.Not Available 4.01-2119529246-39-XXXX	21	<u>alumina hydrate</u>	EUH210 [1]
1.68333-79-9 2.269-789-9 3.Not Available 4.01-2120090300-70-XXXX	19	<u>ammonium polyphosphate</u>	Chronic Aquatic Hazard Category 4; H413 [1]
1.1344-28-1. 2.215-691-6 3.Not Available 4.01-2119529248-35-XXXX	16	<u>aluminium oxide</u>	EUH210 [1]
1.17557-23-2 2.241-536-7 3.603-094-00-7 4.01-2120759332-55-XXXX	6	<u>neopentyl glycol diglycidyl ether</u>	Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H317, H315 [2]
1.12767-90-7 2.215-566-6 3.Not Available 4.01-0000016699-53-XXXX 01-2119691658-19-XXXX 01-2120773328-46-XXXX	5	<u>zinc borate</u>	Eye Irritation Category 2, Chronic Aquatic Hazard Category 1, Acute Aquatic Hazard Category 1, Reproductive Toxicity Category 1B; H319, H410, H400, H360 [1]
1.25068-38-6 2.500-033-5 3.603-074-00-8 4.01-2119456619-26-XXXX	1	<u>bisphenol A/ diglycidyl ether resin, liquid</u>	Eye Irritation Category 2, Chronic Aquatic Hazard Category 2, Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H319, H411, H317, H315 [2]
1.1333-86-4 2.215-609-9 3.Not Available 4.01-2119384822-32-XXXX 01-2120767622-50-	0.4	<u>carbon black</u>	Carcinogenicity Category 2; H351 [1]

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XXXX[01-0000016864-62-XXXX

**Legend:**

1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; \* EU IOELVs available

**SECTION 4 FIRST AID MEASURES****4.1. Description of first aid measures**

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Wash out immediately with fresh running water.</li> <li>▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.</li> <li>▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area.</li> <li>▶ Other measures are usually unnecessary.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> </ul>

**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.

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

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

**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.

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<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Combustible.</li> <li>▶ Slight fire hazard when exposed to heat or flame.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> <li>▶ May emit acrid smoke.</li> <li>▶ Mists containing combustible materials may be explosive.</li> </ul> <p>Combustion products include:  carbon dioxide (CO<sub>2</sub>)  aldehydes  nitrogen oxides (NO<sub>x</sub>)  phosphorus oxides (PO<sub>x</sub>)  metal oxides  other pyrolysis products typical of burning organic material.</p> <p>When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.</p>
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**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**

<b>Minor Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>• 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.</li> <li>• If irritating vapors are present, an approved air-purifying respirator with organic vapor canister is recommended for cleaning up spills and leaks.</li> <li>• For small spills, reactive diluents should be absorbed with sand.</li> </ul> <ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>▶ Wipe up.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> </ul>
<b>Major Spills</b>	<p>Environmental hazard - contain spillage.</p> <p>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.</p> <p>Moderate hazard.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Absorb remaining product with sand, earth or vermiculite.</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

**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**

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ When handling, <b>DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> </ul>
<b>Fire and explosion protection</b>	See section 5

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<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ Store in a cool, dry, well-ventilated area.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>
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## 7.2. Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Metal can or drum</li> <li>▶ Packaging as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
<b>Storage incompatibility</b>	<p>For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate.</p> <p>Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.</p> <p>Epoxides:</p> <ul style="list-style-type: none"> <li>▶ are highly reactive with acids, bases, and oxidising and reducing agents.</li> <li>▶ react, possibly violently, with anhydrous metal chlorides, ammonia, amines and group 1 metals.</li> <li>▶ may polymerise in the presence of peroxides or heat - polymerisation may be violent</li> <li>▶ may react, possibly violently, with water in the presence of acids and other catalysts.</li> </ul> <p>Glycidyl ethers:</p> <ul style="list-style-type: none"> <li>▶ 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</li> <li>▶ may polymerise in contact with heat, organic and inorganic free radical producing initiators</li> <li>▶ may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines</li> <li>▶ react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide</li> <li>▶ attack some forms of plastics, coatings, and rubber</li> </ul> <p>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.</p> <ul style="list-style-type: none"> <li>▶ Avoid cross contamination between the two liquid parts of product (kit).</li> <li>▶ If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur.</li> <li>▶ This excess heat may generate toxic vapour</li> <li>▶ Avoid reaction with amines, mercaptans, strong acids and oxidising agents</li> </ul>

## 7.3. Specific end use(s)

See section 1.2

## SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

## 8.1. Control parameters

**DERIVED NO EFFECT LEVEL (DNEL)**

Not Available

**PREDICTED NO EFFECT LEVEL (PNEC)**

Not Available

**OCCUPATIONAL EXPOSURE LIMITS (OEL)****INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: respirable dust	4 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: inhalable dust	10 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m <sup>3</sup>	7 mg/m <sup>3</sup>	Not Available	Not Available

**EMERGENCY LIMITS**

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
bisphenol A diglycidyl ether	Bisphenol A diglycidyl ether	39 mg/m <sup>3</sup>	430 mg/m <sup>3</sup>	2,600 mg/m <sup>3</sup>
bisphenol A diglycidyl ether	Epoxy resin includes EPON 1001, 1007, 820, ERL-2795	90 mg/m <sup>3</sup>	990 mg/m <sup>3</sup>	5,900 mg/m <sup>3</sup>
alumina hydrate	Aluminum hydroxide	8.7 mg/m <sup>3</sup>	73 mg/m <sup>3</sup>	440 mg/m <sup>3</sup>
aluminium oxide	Aluminum oxide; (Alumina)	5.7 mg/m <sup>3</sup>	15 mg/m <sup>3</sup>	25 mg/m <sup>3</sup>
bisphenol A/ diglycidyl ether resin, liquid	Epoxy resin includes EPON 1001, 1007, 820, ERL-2795	90 mg/m <sup>3</sup>	990 mg/m <sup>3</sup>	5,900 mg/m <sup>3</sup>
carbon black	Carbon black	9 mg/m <sup>3</sup>	99 mg/m <sup>3</sup>	590 mg/m <sup>3</sup>

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Ingredient	Original IDLH	Revised IDLH
bisphenol A diglycidyl ether	Not Available	Not Available
alumina hydrate	Not Available	Not Available
ammonium polyphosphate	Not Available	Not Available
aluminium oxide	Not Available	Not Available
neopentyl glycol diglycidyl ether	Not Available	Not Available
zinc borate	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Not Available	Not Available
carbon black	1,750 mg/m <sup>3</sup>	Not Available

**MATERIAL DATA**

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1.2 µm) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m<sup>3</sup> over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

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

For aluminium oxide:

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

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

For epichlorohydrin

Odour Threshold Value: 0.08 ppm

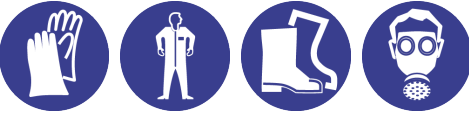
NOTE: Detector tubes for epichlorohydrin, measuring in excess of 5 ppm, are commercially available.

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 (EPICHLOROXYDRIN)

**8.2. Exposure controls**

<p><b>8.2.1. Appropriate engineering controls</b></p>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear 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.</p> <table border="1" data-bbox="391 1310 1484 1568"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="391 1624 1484 1792"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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.)																				
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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																				
<p><b>8.2.2. Personal protection</b></p>																					
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing</li> </ul>																				

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	<ul style="list-style-type: none"> <li>▶ of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> </ul>
<b>Skin protection</b>	See Hand protection below
<b>Hands/feet protection</b>	<p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>- frequency and duration of contact,</li> <li>- chemical resistance of glove material,</li> <li>- glove thickness and</li> <li>- dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>- Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>- Excellent when breakthrough time &gt; 480 min</li> <li>- Good when breakthrough time &gt; 20 min</li> <li>- Fair when breakthrough time &lt; 20 min</li> <li>- Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>- 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.</li> <li>- 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</li> </ul> <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>When handling liquid-grade epoxy resins wear chemically protective gloves, boots and aprons.</p> <p>The performance, based on breakthrough times, of:</p> <ul style="list-style-type: none"> <li>- Ethyl Vinyl Alcohol (EVAL laminate) is generally excellent</li> <li>- Butyl Rubber ranges from excellent to good</li> <li>- Nitrile Butyl Rubber (NBR) from excellent to fair.</li> <li>- Neoprene from excellent to fair</li> <li>- Polyvinyl (PVC) from excellent to poor</li> </ul> <p>As defined in ASTM F-739-96</p> <ul style="list-style-type: none"> <li>- Excellent breakthrough time &gt; 480 min</li> <li>- Good breakthrough time &gt; 20 min</li> <li>- Fair breakthrough time &lt; 20 min</li> <li>- Poor glove material degradation</li> </ul> <p>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)</p> <ul style="list-style-type: none"> <li>- <b>DO NOT</b> use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin).</li> <li>- <b>DO NOT</b> use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use.</li> </ul> <p>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</p>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C. apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eye wash unit.</li> </ul>

**Respiratory protection**

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the 'Exposure Standard' (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS	-	A-PAPR-AUS / Class 1
up to 50 x ES	-	A-AUS / Class 1	-
up to 100 x ES	-	A-2	A-PAPR-2 ^

Continued...

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## ^ - Full-face

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

- ▶ 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	Black		
Physical state	Liquid	Relative density (Water = 1)	1.69
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)	16300
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

## 9.2. Other information

Not Available

## SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> <li>▶ Unstable in the presence of incompatible materials.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
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	<p>The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>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.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p>
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Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual. Reactive diluents exhibit a range of ingestion hazards. Small amounts swallowed incidental to normal handling operations are not likely to cause injury. However, swallowing larger amounts may cause injury.</p> <p>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</p> <p>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.</p> <p>Acute toxic responses to aluminium are confined to the more soluble forms.</p>
Skin Contact	<p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Skin contact with reactive diluents may cause slight to moderate irritation with local redness. Repeated or prolonged skin contact may cause burns.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> <li>▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▶ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.</li> </ul> <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
Eye	<p>Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury. Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>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.</p> <p>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.</p> <p>Very fine Al<sub>2</sub>O<sub>3</sub> powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.</p> <p>When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.</p> <p>The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.</p> <p>Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.</p> <p>There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.</p> <p>Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.</p> <p>Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.</p> <p>After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for</p>

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a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

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

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans.

However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramidal-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of 'tau' a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau *in vitro*. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade.

Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia*, August 1995]

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.

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

Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals.

Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether.

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

Chemicals containing epoxy groups are of concern for cancer effects, though the concern is lower for epoxy groups with di-substituted carbons (US EPA 1994)

The epoxide group is an alkylating agent and thus may produce damage to nucleotides found within the cell; such damage is potentially tumourigenic.

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) needs months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.

Chemicals containing epoxy functional groups are of concern for reproductive effects, though the concern for epoxy groups with di-substituted carbons is lower than that for singly substituted epoxy groups (US EPA, 1994).

834B-A Black Flame Retardant Epoxy	TOXICITY	IRRITATION
	Not Available	Not Available
bisphenol A diglycidyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 2 mg/24h - SEVERE
	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 500 mg - mild
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
alumina hydrate	TOXICITY	IRRITATION

## 834B-A Black Flame Retardant Epoxy

	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
ammonium polyphosphate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >3160 mg/kg <sup>[2]</sup>	Not Available
	Oral (rat) LD50: >=300-2000 mg/kg <sup>[1]</sup>	
aluminium oxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
neopentyl glycol diglycidyl ether	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (rat) LD50: 4500 mg/kg <sup>[2]</sup>	Skin (human): Sensitiser [Shell]
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
zinc borate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): mild *
	Oral (rat) LD50: >5000 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin: non-irritant *
bisphenol A diglycidyl ether resin, liquid	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >1200 mg/kg <sup>[2]</sup>	Eye (rabbit): 100mg - Mild
	Oral (rat) LD50: >1000 mg/kg <sup>[2]</sup>	
carbon black	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (rat) LD50: >15400 mg/kg <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>Legend:</b>	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	

834B-A Black Flame Retardant Epoxy	For aluminium compounds: Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex. Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate). Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability. For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe. Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium. The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium-containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account. Systemic toxicity after repeated exposure No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium. When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose. The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent.
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## 834B-A Black Flame Retardant Epoxy

	<p>Reproductive and developmental toxicity:</p> <p>Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity</p> <p>High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI.</p> <p>Genotoxicity</p> <p>Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.</p> <p>Carcinogenicity.</p> <p>The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons.</p> <p>Neurodegenerative diseases.</p> <p>Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. Some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.</p> <p>Contact sensitivity:</p> <p>It has been suggested that the body burden of aluminium may be linked to different diseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome.</p> <p>Aluminium acts not only as an adjuvant, stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitiser causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be able to act as a contact allergen, then it has to undergo haptensation to be immunogenic and to initiate an immune response. Once inside the skin, the metal ions must bind to proteins to become immunologically reactive. The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminium</p> <p>Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste</p> <p>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.</p> <p>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.</p> <p>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.</p>
BISPHENOL A DIGLYCIDYL ETHER	55badger
NEOPENTYL GLYCOL DIGLYCIDYL ETHER	* Anchor SDS]
BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	Foetotoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity); NOEL (maternal) 60 mg/kg
CARBON BLACK	<b>WARNING:</b> This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Inhalation (rat) TCLo: 50 mg/m <sup>3</sup> /6h/90D-I Nil reported
834B-A Black Flame Retardant Epoxy & BISPHENOL A DIGLYCIDYL ETHER & NEOPENTYL GLYCOL	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

## 834B-A Black Flame Retardant Epoxy

<p><b>DIGLYCIDYL ETHER &amp; BISPENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID</b></p>	<p>opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
<p><b>834B-A Black Flame Retardant Epoxy &amp; BISPENOL A DIGLYCIDYL ETHER &amp; BISPENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID</b></p>	<p>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 &gt;100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg).</p> <p><b>Reproductive and Developmental Toxicity:</b> 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.</p> <p><b>Carcinogenicity:</b> 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).</p> <p>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).</p> <p><b>Genotoxicity:</b> In <i>S. typhimurium</i> 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).</p> <p><b>Immunotoxicity:</b> 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</p> <p>-</p> <p><b>Consumer exposure</b> 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 reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs.</p>
<p><b>834B-A Black Flame Retardant Epoxy &amp; BISPENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID</b></p>	<p>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</p> <p>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.</p> <p>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.</p>
<p><b>834B-A Black Flame Retardant Epoxy &amp; BISPENOL A DIGLYCIDYL ETHER</b></p>	<p>Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review.</p> <p>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.</p> <p>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 hypospadias 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'</p> <p>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'</p> <p>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.</p> <p>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 potentially 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.</p> <p>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 xenestrogens in humans and may be the cause of additional concerns in children.</p> <p>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.</p> <p>Many drugs, including naproxen, salicylic acid, carbamazepine and mefenamic acid can, in vitro, significantly inhibit bisphenol A glucuronidation (detoxification).</p>

## 834B-A Black Flame Retardant Epoxy

	<p>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.</p> <p>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.</p> <p>A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not <i>n</i>-butyl glycidyl ether, induced morphological transformation in mammalian cells <i>in vitro</i>. <i>n</i>-Butyl glycidyl ether induced micronuclei in mice <i>in vivo</i> following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations <i>in vivo</i> or chromosomal aberrations in animal cells <i>in vitro</i>. 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 <i>Drosophila</i>. The glycidyl ethers were generally mutagenic to bacteria</p> <p>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.</p> <p>for 1,2-butylene oxide (ethyloxirane):</p> <p>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/m<sup>3</sup> 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/m<sup>3</sup>) 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</p>
<b>BISPHENOL A DIGLYCIDYL ETHER &amp; BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID</b>	<p>The substance is classified by IARC as Group 3:  <b>NOT</b> classifiable as to its carcinogenicity to humans.  Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
<b>ALUMINA HYDRATE &amp; ALUMINIUM OXIDE &amp; CARBON BLACK</b>	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

## SECTION 12 ECOLOGICAL INFORMATION

## 12.1. Toxicity

834B-A Black Flame Retardant Epoxy	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

bisphenol A diglycidyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.2mg/L	2
	EC50	48	Crustacea	1.1mg/L	2
	EC50	72	Algae or other aquatic plants	9.4mg/L	2
	EC0	48	Crustacea	<1mg/L	2
	NOEC	504	Crustacea	0.3mg/L	2

alumina hydrate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.134mg/L	2
	EC50	48	Crustacea	0.7364mg/L	2
	EC50	72	Algae or other aquatic plants	0.001-0.05mg/L	2
NOEC	168	Crustacea	0.001-mg/L	2	

ammonium polyphosphate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	70mg/L	4
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>97.1mg/L	2
NOEC	72	Algae or other aquatic plants	3.57mg/L	2	

Continued...

## 834B-A Black Flame Retardant Epoxy

aluminium oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.134mg/L	2
	EC50	48	Crustacea	0.7364mg/L	2
	EC50	72	Algae or other aquatic plants	0.001-0.799mg/L	2
	NOEC	240	Crustacea	0.001-0.1002mg/L	2

neopentyl glycol diglycidyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	12.318mg/L	3
	EC50	96	Algae or other aquatic plants	ca.1-73.67mg/L	2

zinc borate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.65mg/L	2
	EC50	48	Crustacea	0.001-0.014mg/L	2
	EC50	96	Algae or other aquatic plants	15.4mg/L	2
	NOEC	72	Algae or other aquatic plants	0.000001mg/L	2

bisphenol A diglycidyl ether resin, liquid	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	48	Crustacea	ca.2mg/L	2

carbon black	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>10-mg/L	2
	EC10	72	Algae or other aquatic plants	>10-mg/L	2
	NOEC	96	Fish	>=1-mg/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.

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Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F ([bis(4-hydroxyphenyl)methane; BPF], which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem, Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe<sup>3+</sup> 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 (t<sub>1/2</sub>water : t<sub>1/2</sub>soil : t<sub>1/2</sub>sediment = 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).

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

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.

For aluminium and its compounds and salts:

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

#### Environmental fate:

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

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

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

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

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

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

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

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

Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminium to above-ground parts. Tea leaves may contain very high concentrations of aluminium, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminium include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae).

Aluminium is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminium and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminium is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminium in the plant/concentration of aluminium in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminium is not taken up in plants from soil, but is instead biologically diluted.

Aluminium concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminium residue analyses in brook trout have shown that whole-body aluminium content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminium uptake, to eliminate aluminium at a rate that exceeds uptake, or to maintain approximately the same amount of aluminium while the body mass increases. The decline in whole-body aluminium residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminium than did the other tissues.

The greatest fraction of the gill-associated aluminium was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminium transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminium in whole-body tissue of the Atlantic salmon exposed to high



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concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.

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

**Ecotoxicity:****Freshwater species pH >6.5**

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

Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L

Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

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

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

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

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

Alga: 1 sp NOEC growth 2.0 mg/L

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

The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4-5.4, with a maximum toxicity occurring around pH 5.0-5.2. The inorganic single unit aluminium species (Al(OH)<sub>2</sub><sup>+</sup>) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H<sup>+</sup> concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.

Drinking Water Standards:

aluminium: 200 ug/l (UK max.)

200 ug/l (WHO guideline)

chloride: 400 mg/l (UK max.)

250 mg/l (WHO guideline)

fluoride: 1.5 mg/l (UK max.)

1.5 mg/l (WHO guideline)

nitrate: 50 mg/l (UK max.)

50 mg/l (WHO guideline)

sulfate: 250 mg/l (UK max.)

Soil Guideline: none available.

Air Quality Standards: none available.

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

**12.2. Persistence and degradability**

Ingredient	Persistence: Water/Soil	Persistence: Air
bisphenol A diglycidyl ether	HIGH	HIGH
neopentyl glycol diglycidyl ether	HIGH	HIGH
bisphenol A/ diglycidyl ether resin, liquid	HIGH	HIGH

**12.3. Bioaccumulative potential**

Ingredient	Bioaccumulation
bisphenol A diglycidyl ether	MEDIUM (LogKOW = 3.8446)
neopentyl glycol diglycidyl ether	LOW (LogKOW = 0.2342)
bisphenol A/ diglycidyl ether resin, liquid	LOW (LogKOW = 2.6835)

**12.4. Mobility in soil**

Ingredient	Mobility
bisphenol A diglycidyl ether	LOW (KOC = 1767)
neopentyl glycol diglycidyl ether	LOW (KOC = 10)
bisphenol A/ diglycidyl ether resin, liquid	LOW (KOC = 51.43)

**12.5. Results of PBT and vPvB assessment**

	P	B	T
Relevant available data	Not Applicable	Not Applicable	Not Applicable
PBT Criteria fulfilled?	Not Applicable	Not Applicable	Not Applicable

**12.6. Other adverse effects**

No data available

**SECTION 13 DISPOSAL CONSIDERATIONS****13.1. Waste treatment methods**

Product / Packaging disposal	
	▶ Containers may still present a chemical hazard/ danger when empty.

Continued...

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	<ul style="list-style-type: none"> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> </ul> <p>Waste Management</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>▶ Reduction</li> <li>▶ Reuse</li> <li>▶ Recycling</li> <li>▶ Disposal (if all else fails)</li> </ul> <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Authority for disposal.</li> <li>▶ Bury or incinerate residue at an approved site.</li> <li>▶ Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>
Waste treatment options	Not Available
Sewage disposal options	Not Available

## SECTION 14 TRANSPORT INFORMATION

## Labels Required

	<p>For 834B-375ML, 834B-2.7L</p> <p>NOT REGULATED by Ground ADR Special Provision 375</p> <p>NOT REGULATED by Air IATA Special Provision A197</p> <p>NOT REGULATED by Sea IMDG per 2.10.2.7</p> <p>NOT REGULATED by ADN Special Provision 274 (The provision of 3.1.2.8 apply)</p>
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## Land transport (ADR)

14.1. UN number	3082										
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S.										
14.3. Transport hazard class(es)	<table border="0"> <tr> <td>Class</td> <td>9</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	9	Subrisk	Not Applicable						
Class	9										
Subrisk	Not Applicable										
14.4. Packing group	III										
14.5. Environmental hazard	Environmentally hazardous										
14.6. Special precautions for user	<table border="0"> <tr> <td>Hazard identification (Kemler)</td> <td>90</td> </tr> <tr> <td>Classification code</td> <td>M6</td> </tr> <tr> <td>Hazard Label</td> <td>9</td> </tr> <tr> <td>Special provisions</td> <td>274 335 375 601</td> </tr> <tr> <td>Limited quantity</td> <td>5 L</td> </tr> </table>	Hazard identification (Kemler)	90	Classification code	M6	Hazard Label	9	Special provisions	274 335 375 601	Limited quantity	5 L
Hazard identification (Kemler)	90										
Classification code	M6										
Hazard Label	9										
Special provisions	274 335 375 601										
Limited quantity	5 L										

## Air transport (ICAO-IATA / DGR)

14.1. UN number	3082						
14.2. UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. *						
14.3. Transport hazard class(es)	<table border="0"> <tr> <td>ICAO/IATA Class</td> <td>9</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>9L</td> </tr> </table>	ICAO/IATA Class	9	ICAO / IATA Subrisk	Not Applicable	ERG Code	9L
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	<table border="0"> <tr> <td>Special provisions</td> <td>A97 A158 A197</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>964</td> </tr> </table>	Special provisions	A97 A158 A197	Cargo Only Packing Instructions	964		
Special provisions	A97 A158 A197						
Cargo Only Packing Instructions	964						

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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.
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.
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

## SECTION 15 REGULATORY INFORMATION

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

## BIPHENOL A DIGLYCIDYL ETHER(1675-54-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
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 - Chemwatch Standard Format
Europe EC Inventory	European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	GESAMP/EHS Composite List - GESAMP Hazard Profiles
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)	IMO IBC Code Chapter 17: Summary of minimum requirements
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)	IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
European Customs Inventory of Chemical Substances ECICS (English)	International Air Transport Association (IATA) Dangerous Goods Regulations
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation	International FOSFA List of Banned Immediate Previous Cargoes
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	International Maritime Dangerous Goods Requirements (IMDG Code)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31	Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)

## ALUMINA HYDRATE(21645-51-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Europe EC Inventory	Europe European Customs Inventory of Chemical Substances ECICS (Romanian)
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
Europe European Customs Inventory of Chemical Substances - ECICS (Slovak)	European Customs Inventory of Chemical Substances ECICS (English)
Europe European Customs Inventory of Chemical Substances ECICS (Bulgarian)	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
Europe European Customs Inventory of Chemical Substances ECICS (Czech)	

Continued...

## 834B-A Black Flame Retardant Epoxy

**AMMONIUM POLYPHOSPHATE(68333-79-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	IMO IBC Code Chapter 17: Summary of minimum requirements
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
European Customs Inventory of Chemical Substances ECICS (English)	

**ALUMINIUM OXIDE(1344-28-1.) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Europe EC Inventory	European Customs Inventory of Chemical Substances ECICS (English)
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	UK Workplace Exposure Limits (WELs)

**NEOPENTYL GLYCOL DIGLYCIDYL ETHER(17557-23-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Europe EC Inventory	European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	International FOSFA List of Banned Immediate Previous Cargoes

**ZINC BORATE(12767-90-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways	European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products - Annex III - List of Substances which cosmetic products must not contain except subject to the restrictions laid down (Czech)	European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products - Annex III - List of Substances which cosmetic products must not contain except subject to the restrictions laid down (Estonian)	European Customs Inventory of Chemical Substances ECICS (English)
EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products - Annex III - List of Substances which cosmetic products must not contain except subject to the restrictions laid down (German)	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products - Annex III - List of Substances which cosmetic products must not contain except subject to the restrictions laid down (Latvian)	European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products - Annex III - List of Substances which cosmetic products must not contain except subject to the restrictions laid down (Lithuanian)	International Air Transport Association (IATA) Dangerous Goods Regulations
EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products - Annex III - List of Substances which cosmetic products must not contain except subject to the restrictions laid down (Portuguese)	International Maritime Dangerous Goods Requirements (IMDG Code)
Europe EC Inventory	Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)	

**BIPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID(25068-38-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
Europe EC Inventory	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)	International Air Transport Association (IATA) Dangerous Goods Regulations
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)	International FOSFA List of Banned Immediate Previous Cargoes
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	International Maritime Dangerous Goods Requirements (IMDG Code)
European Customs Inventory of Chemical Substances ECICS (English)	Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)	

**CARBON BLACK(1333-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances	European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
European Customs Inventory of Chemical Substances ECICS (English)	UK Workplace Exposure Limits (WELs)
European List of Notified Chemical Substances (ELINCS)	

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) 2015/830; 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**

Continued...

## 834B-A Black Flame Retardant Epoxy

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (bisphenol A/ diglycidyl ether resin, liquid; neopentyl glycol diglycidyl ether; bisphenol A diglycidyl ether; aluminium oxide; alumina hydrate; carbon black; ammonium polyphosphate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (ammonium polyphosphate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (neopentyl glycol diglycidyl ether; bisphenol A diglycidyl ether; ammonium polyphosphate)
Vietnam - NCI	Yes
Russia - ARIPS	No (neopentyl glycol diglycidyl ether)
Thailand - TECl	No (bisphenol A/ diglycidyl ether resin, liquid; neopentyl glycol diglycidyl ether; zinc borate; bisphenol A diglycidyl ether)
<b>Legend:</b>	Yes = All declared ingredients are on the inventory No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

## SECTION 16 OTHER INFORMATION

Revision Date	10/05/2019
Initial Date	12/10/2017

## Full text Risk and Hazard codes

<b>H351</b>	Suspected of causing cancer.
<b>H360</b>	May damage fertility or the unborn child.
<b>H400</b>	Very toxic to aquatic life.
<b>H410</b>	Very toxic to aquatic life with long lasting effects.
<b>H413</b>	May cause long lasting harmful effects to aquatic life.

## SDS Version Summary

Version	Issue Date	Sections Updated
4.7.1.1.1	10/05/2019	Acute Health (inhaled), Acute Health (swallowed), Advice to Doctor, Chronic Health, Classification, First Aid (swallowed), Ingredients

## Other information

## Ingredients with multiple cas numbers

Name	CAS No
bisphenol A diglycidyl ether	1675-54-3, 116161-20-7, 170962-54-6, 47424-12-4, 85101-00-4, 25068-38-6
alumina hydrate	14762-49-3, 21645-51-2
aluminium oxide	1344-28-1, 1011245-20-7, 1022097-81-9, 107462-07-7, 107874-14-6, 1097999-44-4, 1197416-35-5, 122784-35-4, 1234495-70-5, 1239586-42-5, 12522-88-2, 127361-04-0, 12737-16-5, 131689-14-0, 1346644-15-2, 135152-65-7, 1355357-83-3, 135667-70-8, 138361-58-7, 148619-39-0, 152743-26-5, 153858-98-1, 157516-29-5, 163581-50-8, 165390-91-0, 170448-81-4, 190401-78-6, 200295-99-4, 205316-36-5, 209552-43-2, 230616-05-4, 252756-35-7, 253606-46-1, 253606-47-2, 253606-45-0, 268724-08-9, 39354-49-9, 457654-46-5, 488831-46-5, 521982-71-8, 53809-96-4, 54352-04-4, 546141-61-1, 663170-52-3, 67853-35-4, 67894-14-8, 67894-42-2, 68189-68-4, 68389-42-4, 68389-43-5, 74871-10-6, 76363-81-0, 84149-21-3, 90669-62-8, 916225-60-0, 960377-08-6, 11092-32-3
zinc borate	1332-07-6, 108749-27-5, 13826-88-5, 12767-90-7, 139354-75-9, 14720-55-9, 12230-20-5, 12536-65-1, 12007-67-9, 115887-05-3, 12007-72-6, 12008-25-2
bisphenol A/ diglycidyl ether resin, liquid	25068-38-6, 25085-99-8

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

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

## 834B-A Black Flame Retardant Epoxy

STEL: Short Term Exposure Limit  
TEEL: Temporary Emergency Exposure Limit,  
IDLH: Immediately Dangerous to Life or Health Concentrations  
OSF: Odour Safety Factor  
NOAEL :No Observed Adverse Effect Level  
LOAEL: Lowest Observed Adverse Effect Level  
TLV: Threshold Limit Value  
LOD: Limit Of Detection  
OTV: Odour Threshold Value  
BCF: BioConcentration Factors  
BEI: Biological Exposure Index

### Reason for Change

A-1.02 - Update to the emergency contact number.