



## 834BLV Flame Retardant Epoxy (Part A)

### MG Chemicals UK Limited

Version No: A-1.00

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

Issue Date: 13/06/2023

Revision Date: 13/06/2023

L.REACH.GB.EN

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

##### 1.1. Product Identifier

Product name	834BLV Flame Retardant Epoxy (Part A)
Synonyms	834BLV-450ML, 834BLV-3L, 834BLV-60L
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A diglycidyl ether resin, solid)
Other means of identification	834BLV13062023   UFI:VXQ0-00X5-1007-191N

##### 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	No specific uses advised against are identified.

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

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)	MG Chemicals (Head office)
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	1210 Corporate Drive Ontario L7L 5R6 Canada	1210 Corporate Drive Ontario L7L 5R6 Canada
Telephone	+(44) 1663 362888	+(1) 800-340-0772	+(1) 800-340-0772
Fax	Not Available	+(1) 800-340-0773	+(1) 800-340-0773
Website	Not Available	<a href="http://www.mgchemicals.com">www.mgchemicals.com</a>	<a href="http://www.mgchemicals.com">www.mgchemicals.com</a>
Email	<a href="mailto:sales@mgchemicals.com">sales@mgchemicals.com</a>	<a href="mailto:Info@mgchemicals.com">Info@mgchemicals.com</a>	<a href="mailto:Info@mgchemicals.com">Info@mgchemicals.com</a>

##### 1.4. Emergency telephone number

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

#### SECTION 2 Hazards identification

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

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H411 - Hazardous to the Aquatic Environment Long-Term Hazard Category 2, H315 - Skin Corrosion/Irritation Category 2, H319 - Serious Eye Damage/Eye Irritation Category 2, H317 - Sensitisation (Skin) Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567

##### 2.2. Label elements

Hazard pictogram(s)	
Signal word	Warning

##### Hazard statement(s)

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

##### Supplementary Phrases

## 834BLV Flame Retardant Epoxy (Part A)

EUH210	Safety data sheet available on request.
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## Precautionary statement(s) Prevention

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

## Precautionary statement(s) Response

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

## Precautionary statement(s) Storage

Not Applicable

## Precautionary statement(s) Disposal

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

Skin contact and/or ingestion may produce health damage\*.

Substance PBT/vPvB  $\geq 0.1\%$  = none

Cumulative effects may result following exposure\*.

Endocrine Disruptor substance  $\geq 0.1\%$  = none

May produce discomfort of the eyes and respiratory tract\*.

Limited evidence of a carcinogenic effect\*.

Possible respiratory sensitizer\*.

May possibly affect fertility\*.

Repeated exposure potentially causes skin dryness and cracking\*.

Vapours potentially cause drowsiness and dizziness\*.

bisphenol A diglycidyl ether resin, solid	Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors
propylene glycol monomethyl ether - mixture of isomers	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)
C14-20 aliphatics ( $\leq 2\%$ aromatics)	Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors
naphtha petroleum, light aromatic solvent	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)
xylene	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)

## SECTION 3 Composition / information on ingredients

## 3.1.Substances

See 'Composition on ingredients' in Section 3.2

## 3.2.Mixtures

1. CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	SCL / M-Factor	Nanoform Particle Characteristics
1. 25085-99-8 2.500-033-5 3.603-074-00-8 4.Not Available	53	bisphenol A diglycidyl ether resin, solid [e]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H315, H319, H317, H411 [2]	Eye Irrit. 2; H319: C $\geq 5\%$   Skin Irrit 2; H315: C $\geq 5\%$	Not Available
1. 21645-51-2 2.244-492-7 3.Not Available 4.Not Available	23	alumina hydrate	Not Applicable	Not Available	Not Available
1. 68333-79-9 2.269-789-9 3.Not Available 4.Not Available	16	ammonium polyphosphate	Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H413 [1]	Not Available	Not Available

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## 834BLV Flame Retardant Epoxy (Part A)

1. CAS No 2. EC No 3. Index No 4. REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	SCL / M-Factor	Nanoform Particle Characteristics
1. 108-65-6 2. 203-603-9 3. 603-064-00-3 607-195-00-7 603-106-00-0 4. Not Available	0.9	<u>propylene glycol monomethyl ether - mixture of isomers</u> *	Flammable Liquids Category 3; H226 [2]	Not Available	Not Available
1. 1333-86-4 2. 215-609-9 422-130-0 435-640-3 3. Not Available 4. Not Available	0.8	<u>carbon black</u>	Carcinogenicity Category 2; H351 [1]	Not Available	Not Available
1. 8052-41-3 2. 265-149-8 232-489-3 3. 649-422-00-2 649-345-00-4 4. Not Available	0.4	<u>Stoddard Solvent</u>	Flammable Liquids Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Aspiration Hazard Category 1; H226, H336, H304, EUH066 [1]	Not Available	Not Available
1. 162627-21-6 2. Not Available 3. Not Available 4. Not Available	0.3	<u>caprolactone, ethoxylated polyphosphates</u>	Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H318, H413 [1]	Not Available	Not Available
1. 64742-47-8 2. 265-093-4 265-148-2 265-149-8 3. 649-214-00-1 649-221-00-X 649-422-00-2 4. Not Available	0.2	<u>C14-20 aliphatics (&lt;=2% aromatics)</u> [e]	Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Aspiration Hazard Category 1; H336, H304, EUH066 [1]	Not Available	Not Available
1. 64742-95-6 2. 265-199-0 3. 649-356-00-4 4. Not Available	0.1	<u>naphtha petroleum, light aromatic solvent</u>	Germ Cell Mutagenicity Category 1B, Carcinogenicity Category 1B, Aspiration Hazard Category 1; H340, H350, H304 [2]	Not Available	Not Available
1. 1330-20-7 2. 215-535-7 3. 601-022-00-9 4. Not Available	0.1	<u>xylene</u> *	Flammable Liquids Category 3, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2; H226, H312, H332, H315 [2]	*	Not Available
<b>Legend:</b>		1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties			

## SECTION 4 First aid measures

## 4.1. Description of first aid measures

<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>Immediately give a glass of water.</li> <li>First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.</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.

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

Treat symptomatically.

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## 834BLV Flame Retardant Epoxy (Part A)

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

[Ellenhorn and Barceloux: Medical Toxicology]

## SECTION 5 Firefighting measures

## 5.1. Extinguishing media

- ▶ 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

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

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear full body protective clothing with breathing apparatus.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ 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>

## SECTION 6 Accidental release measures

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

See section 8

## 6.2. Environmental precautions

See section 12

## 6.3. Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <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>																														
Major Spills	<p>Environmental hazard - contain spillage.</p> <p>Chemical Class: aromatic hydrocarbons</p> <p>For release onto land: recommended sorbents listed in order of priority.</p> <table><tr><th>SORBENT TYPE</th><th>RANK</th><th>APPLICATION</th><th>COLLECTION</th><th>LIMITATIONS</th></tr></table> <p>LAND SPILL - SMALL</p> <table><tr><td>Feathers - pillow</td><td>1</td><td>throw</td><td>pitchfork</td><td>DGC, RT</td></tr><tr><td>cross-linked polymer - particulate</td><td>2</td><td>shovel</td><td>shovel</td><td>R,W,SS</td></tr><tr><td>cross-linked polymer- pillow</td><td>2</td><td>throw</td><td>pitchfork</td><td>R, DGC, RT</td></tr><tr><td>sorbent clay - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, I, P,</td></tr><tr><td>treated clay/ treated natural organic - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, I</td></tr></table>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	Feathers - pillow	1	throw	pitchfork	DGC, RT	cross-linked polymer - particulate	2	shovel	shovel	R,W,SS	cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	3	shovel	shovel	R, I, P,	treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I
SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS																											
Feathers - pillow	1	throw	pitchfork	DGC, RT																											
cross-linked polymer - particulate	2	shovel	shovel	R,W,SS																											
cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT																											
sorbent clay - particulate	3	shovel	shovel	R, I, P,																											
treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I																											

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## 834BLV Flame Retardant Epoxy (Part A)

wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT
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## LAND SPILL - MEDIUM

cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
feathers - pillow	3	throw	skiploader	DGC, RT
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC

## Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT:Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

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

Moderate hazard.

- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Absorb remaining product with sand, earth or vermiculite.
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

## 6.4. Reference to other sections

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

## SECTION 7 Handling and storage

## 7.1. Precautions for safe handling

Safe handling	<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>
Fire and explosion protection	See section 5
Other information	<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>

## 7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<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>
Storage incompatibility	<p>For aluminas (aluminium oxide):</p> <p>Incompatible with hot chlorinated rubber.</p> <p>In the presence of chlorine trifluoride may react violently and ignite.</p> <p>-May initiate explosive polymerisation of olefin oxides including ethylene oxide.</p> <p>-Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.</p> <p>-Produces exothermic reaction with oxygen difluoride.</p>

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	<p>-May form explosive mixture with oxygen difluoride.</p> <p>-Forms explosive mixtures with sodium nitrate.</p> <p>-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>Xylenes:</p> <ul style="list-style-type: none"> <li>▶ may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride</li> <li>▶ attack some plastics, rubber and coatings</li> <li>▶ may generate electrostatic charges on flow or agitation due to low conductivity.</li> <li>▶ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents.</li> <li>▶ Aromatics can react exothermically with bases and with diazo compounds.</li> </ul> <p>For alkyl aromatics:</p> <p>The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.</p> <ul style="list-style-type: none"> <li>▶ Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen</li> <li>▶ Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.</li> <li>▶ Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.</li> <li>▶ Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.</li> <li>▶ Alkali metals accelerate the oxidation while CO<sub>2</sub> as co-oxidant enhances the selectivity.</li> <li>▶ Microwave conditions give improved yields of the oxidation products.</li> <li>▶ Photo-oxidation products may occur following reaction with hydroxyl radicals and NO<sub>x</sub> - these may be components of photochemical smogs.</li> </ul> <p>Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007</p> <ul style="list-style-type: none"> <li>▶ Avoid reaction with amines, mercaptans, strong acids and oxidising agents</li> <li>▶ Phosphates are incompatible with oxidising and reducing agents.</li> <li>▶ Phosphates are susceptible to formation of highly toxic and flammable phosphine gas in the presence of strong reducing agents such as hydrides.</li> <li>▶ Partial oxidation of phosphates by oxidizing agents may result in the release of toxic phosphorus oxides.</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>
<b>Hazard categories in accordance with Regulation (EC) No 1272/2008</b>	E2: Hazardous to the Aquatic Environment in Category Chronic 2
<b>Qualifying quantity (tonnes) of dangerous substances as referred to in Article 3(10) for the application of</b>	E2 Lower- / Upper-tier requirements: 200 / 500

## 7.3. Specific end use(s)

See section 1.2

## SECTION 8 Exposure controls / personal protection

## 8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
alumina hydrate	Dermal 4.063 mg/kg bw/day (Systemic, Chronic) Inhalation 10.76 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 10.76 mg/m <sup>3</sup> (Local, Chronic) Oral 4.74 mg/kg bw/day (Systemic, Chronic) *	Not Available
ammonium polyphosphate	Inhalation 18.06 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 4.45 mg/m <sup>3</sup> (Systemic, Chronic) * Oral 1.28 mg/kg bw/day (Systemic, Chronic) *	Not Available
propylene glycol monomethyl ether - mixture of isomers	Dermal 183 mg/kg bw/day (Systemic, Chronic) Inhalation 275 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 553.5 mg/m <sup>3</sup> (Systemic, Acute) Inhalation 550 mg/m <sup>3</sup> (Local, Acute) Dermal 78 mg/kg bw/day (Systemic, Chronic) * Inhalation 33 mg/m <sup>3</sup> (Systemic, Chronic) * Oral 33 mg/kg bw/day (Systemic, Chronic) * Inhalation 33 mg/m <sup>3</sup> (Local, Chronic) *	0.635 mg/L (Water (Fresh)) 0.064 mg/L (Water - Intermittent release) 6.35 mg/L (Water (Marine)) 3.29 mg/kg sediment dw (Sediment (Fresh Water)) 0.329 mg/kg sediment dw (Sediment (Marine)) 0.29 mg/kg soil dw (Soil) 100 mg/L (STP)
carbon black	Inhalation 1 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 0.5 mg/m <sup>3</sup> (Local, Chronic) Inhalation 0.06 mg/m <sup>3</sup> (Systemic, Chronic) *	1 mg/L (Water (Fresh)) 0.1 mg/L (Water - Intermittent release) 10 mg/L (Water (Marine))
Stoddard Solvent	Dermal 80 mg/kg bw/day (Systemic, Chronic) Inhalation 44 mg/m <sup>3</sup> (Systemic, Chronic) Dermal 7.56 mg/cm <sup>2</sup> (Local, Chronic) Inhalation 44 mg/m <sup>3</sup> (Local, Chronic) Dermal 30 mg/kg bw/day (Systemic, Acute) Inhalation 55 mg/m <sup>3</sup> (Systemic, Acute) Inhalation 55 mg/m <sup>3</sup> (Local, Acute) Dermal 40 mg/kg bw/day (Systemic, Chronic) *	0.14 mg/L (Water (Fresh)) 0.35 mg/L (Water - Intermittent release) 0.014 mg/L (Water (Marine)) 1.14 mg/kg sediment dw (Sediment (Fresh Water)) 0.14 mg/kg sediment dw (Sediment (Marine))

Continued...

## 834BLV Flame Retardant Epoxy (Part A)

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
	<i>Inhalation 22 mg/m³ (Systemic, Chronic) *</i> <i>Oral 10.56 mg/kg bw/day (Systemic, Chronic) *</i> <i>Dermal 3.78 mg/cm² (Local, Chronic) *</i> <i>Inhalation 22 mg/m³ (Local, Chronic) *</i> <i>Dermal 60 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 55 mg/m³ (Systemic, Acute) *</i> <i>Oral 50 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 55 mg/m³ (Local, Acute) *</i>	
C14-20 aliphatics (<=2% aromatics)	Dermal 2.91 mg/kg bw/day (Systemic, Chronic) Inhalation 16.4 mg/m³ (Systemic, Chronic) Inhalation 5 002.67 mg/m³ (Systemic, Acute) <i>Dermal 1.25 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 4.85 mg/m³ (Systemic, Chronic) *</i> <i>Oral 1.25 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 3 001.6 mg/m³ (Systemic, Acute) *</i>	17 g/kg food (Oral)
naphtha petroleum, light aromatic solvent	Inhalation 837.5 mg/m³ (Local, Chronic) Inhalation 1 286.4 mg/m³ (Systemic, Acute) Inhalation 1 066.67 mg/m³ (Local, Acute) <i>Inhalation 178.57 mg/m³ (Local, Chronic) *</i> <i>Inhalation 1 152 mg/m³ (Systemic, Acute) *</i> <i>Inhalation 640 mg/m³ (Local, Acute) *</i>	Not Available
xylene	Dermal 212 mg/kg bw/day (Systemic, Chronic) Inhalation 221 mg/m³ (Systemic, Chronic) Inhalation 221 mg/m³ (Local, Chronic) Inhalation 442 mg/m³ (Systemic, Acute) Inhalation 442 mg/m³ (Local, Acute) <i>Dermal 125 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 65.3 mg/m³ (Systemic, Chronic) *</i> <i>Oral 12.5 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 65.3 mg/m³ (Local, Chronic) *</i> <i>Inhalation 260 mg/m³ (Systemic, Acute) *</i> <i>Inhalation 260 mg/m³ (Local, Acute) *</i>	0.327 mg/L (Water (Fresh)) 0.327 mg/L (Water - Intermittent release) 0.327 mg/L (Water (Marine)) 12.46 mg/kg sediment dw (Sediment (Fresh Water)) 12.46 mg/kg sediment dw (Sediment (Marine)) 2.31 mg/kg soil dw (Soil) 6.58 mg/L (STP)

\* Values for General Population

## Occupational Exposure Limits (OEL)

## INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs).	propylene glycol monomethyl ether - mixture of isomers	1-Methoxypropan-2-ol	100 ppm / 375 mg/m3	560 mg/m3 / 150 ppm	Not Available	Sk
UK Workplace Exposure Limits (WELs).	propylene glycol monomethyl ether - mixture of isomers	1-Methoxypropyl acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Sk
UK Workplace Exposure Limits (WELs).	carbon black	Carbon black	3.5 mg/m3	7 mg/m3	Not Available	Not Available
UK Workplace Exposure Limits (WELs).	xylene	Xylene, o-,m-,p- or mixed isomers	50 ppm / 220 mg/m3	441 mg/m3 / 100 ppm	Not Available	Sk, BMGV

## Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
bisphenol A diglycidyl ether resin, solid	90 mg/m3	990 mg/m3	5,900 mg/m3
bisphenol A diglycidyl ether resin, solid	30 mg/m3	330 mg/m3	2,000 mg/m3
alumina hydrate	8.7 mg/m3	73 mg/m3	440 mg/m3
propylene glycol monomethyl ether - mixture of isomers	100 ppm	160 ppm	660 ppm
propylene glycol monomethyl ether - mixture of isomers	Not Available	Not Available	Not Available
carbon black	9 mg/m3	99 mg/m3	590 mg/m3
Stoddard Solvent	300 mg/m3	1,800 mg/m3	29500** mg/m3
C14-20 aliphatics (<=2% aromatics)	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
xylene	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
bisphenol A diglycidyl ether resin, solid	Not Available	Not Available
alumina hydrate	Not Available	Not Available
ammonium polyphosphate	Not Available	Not Available
propylene glycol monomethyl ether - mixture of isomers	Not Available	Not Available

Continued...

## 834BLV Flame Retardant Epoxy (Part A)

Ingredient	Original IDLH	Revised IDLH
carbon black	1,750 mg/m <sup>3</sup>	Not Available
Stoddard Solvent	20,000 mg/m <sup>3</sup>	Not Available
caprolactone, ethoxylated polyphosphates	Not Available	Not Available
C14-20 aliphatics (<=2% aromatics)	2,500 mg/m <sup>3</sup>	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
xylene	900 ppm	Not Available

## Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
bisphenol A diglycidyl ether resin, solid	E	≤ 0.01 mg/m <sup>3</sup>
caprolactone, ethoxylated polyphosphates	C	> 1 to ≤ 10 parts per million (ppm)
naphtha petroleum, light aromatic solvent	E	≤ 0.1 ppm

**Notes:** Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

## MATERIAL DATA

IFRA Prohibited Fragrance Substance

The International Fragrance Association (IFRA) Standards form the basis for the globally accepted and recognized risk management system for the safe use of fragrance ingredients and are part of the IFRA Code of Practice. This is the self-regulating system of the industry, based on risk assessments carried out by an independent Expert Panel

**WARNING:** This substance is classified by the NOHSC as Category 2 Probable Human Carcinogen

These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.

CR = Cancer Risk/10000; UF = Uncertainty factor:

TLV believed to be adequate to protect reproductive health:

LOD: Limit of detection

Toxic endpoints have also been identified as:

D = Developmental; R = Reproductive; TC = Transplacental carcinogen

Jankovic J., Drake F.: A Screening Method for Occupational Reproductive

American Industrial Hygiene Association Journal 57: 641-649 (1996)

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 propylene glycol monomethyl ether (PGME)

Odour Threshold: 10 ppm.

The TLV-TWA is protective against discomfort caused by odour, against eye and skin irritation, and chronic effects (including possible liver and kidney damage).

Individuals exposed to 100 ppm reported a transient unpleasant odour with slight eye irritation after about 1 or 2 hours. At 300 ppm, mild irritation of the eyes and nose developed within 5 minutes; some individuals found the irritation hardly bearable after about an hour. A concentration of 750 ppm was highly irritating. Signs of central nervous system depression developed at 1000 ppm. Neurological, clinical chemical and general medical examinations showed no other conspicuous toxicity.

Concentrations of the beta-isomer, 2-methoxy-1-propyl acetate are low in commercial grades of PGME and teratogenic effects associated with this isomer are expected to be absent.

Odour Safety Factor(OSF)

OSF=10 (propylene glycol monomethyl ether)

The TLV is based on the exposures to aluminium chloride and the amount of hydrolysed acid and the corresponding acid TLV to provide the same degree of freedom from irritation.

Workers chronically exposed to aluminium dusts and fumes have developed severe pulmonary reactions including fibrosis, emphysema and pneumothorax. A much rarer encephalopathy has also been described.

For trimethyl benzene as mixed isomers (of unstated proportions)

Odour Threshold Value: 2.4 ppm (detection)

Use care in interpreting effects as a single isomer or other isomer mix. Trimethylbenzene is an eye, nose and respiratory irritant. High concentrations cause central nervous system depression. Exposed workers show CNS changes, asthmatic bronchitis and blood dyscrasias at 60 ppm. The TLV-TWA is thought to be protective against the significant risk of CNS

excitation, asthmatic bronchitis and blood dyscrasias associated with exposures above the limit.

Odour Safety Factor (OSF)

OSF=10 (1,2,4-TRIMETHYLBENZENE)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

ClassOSF Description

A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities

B 26-550 As "A" for 50-90% of persons being distracted

C 1-26 As "A" for less than 50% of persons being distracted

D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached

E <0.18 As "D" for less than 10% of persons aware of being tested

for xylenes:

IDLH Level: 900 ppm

Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)

NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).

Continued...



## 834BLV Flame Retardant Epoxy (Part A)

Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.

Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially contribute to the dose received by inhalation.

Odour Safety Factor(OSF)

OSF=4 (XYLENE)

NOTE N: The classification as a carcinogen need not apply if the full refining history is known and it can be shown that the substance from which it is produced is not a carcinogen.

This note applies only to certain complex oil-derived substances in Annex VI.


European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also

apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

## 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"> <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"> <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
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<p><b>8.2.2. Individual protection measures, such as personal protective equipment</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. [AS/NZS 1337.1, EN166 or national equivalent]</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].</li> </ul>																				
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>																				
<p><b>Hands/feet protection</b></p>	<p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>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>																				

## 834BLV Flame Retardant Epoxy (Part A)

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

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

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

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

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

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

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

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

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

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

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

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

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

The performance, based on breakthrough times, of:

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

As defined in ASTM F-739-96

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

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

· **DO NOT** use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin).

· **DO NOT** use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use.

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

**Body protection** See Other protection below

**Other protection**

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

## Recommended material(s)

### GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

**"Forsberg Clothing Performance Index".**

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

834BLV Flame Retardant Epoxy (Part A)

Material	CPI
PE/EVAL/PE	A
PVA	A
TEFLON	A
VITON	A
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C

## 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 ^

^ - 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

Continued...

## 834BLV Flame Retardant Epoxy (Part A)

NITRILE+PVC	C
PVC	C
PVDC/PE/PVDC	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

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

- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
  - The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
  - Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
  - Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
  - Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
  - Use approved positive flow mask if significant quantities of dust becomes airborne.
  - Try to avoid creating dust conditions.
- Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.
- P2 is a respiratory filter rating under various international standards, Filters at least 94% of airborne particles
- Suitable for:
- Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
  - Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
  - Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS

## 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)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	220
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	110	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 (%)	6.89	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.36	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Not Available	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	1.51	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

## 9.2. Other information

Not Available

Continued...

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## 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>A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias. High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement.</p> <p>Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.</p> <p>Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.</p> <p>Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue.</p> <p>Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p>
Ingestion	<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> <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>The material has <b>NOT</b> been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>Phosphates are slowly and incompletely absorbed from the gastrointestinal tract and are unlikely (other than in abuse) to produce the systemic effects which occur when introduced by other routes. Such effects include vomiting, lethargy, fever, diarrhoea, falls in blood pressure, slow pulse, cyanosis, carpal spasm, coma and tetany. These effects result following sequestration of blood calcium.</p> <p>Ingestion of large amounts of phosphate salts (over 1 gm for an adult) may produce osmotic catharsis resulting in diarrhoea and probably, abdominal cramp. Large doses (4-8 gm) will almost certainly produce these effects in most individuals. Most of the ingested salt will be excreted in the faeces of healthy individuals without producing systemic toxicity. Doses in excess of 10 gm may produce systemic toxicity.</p>
Skin Contact	<p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</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>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.</p> <p>Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>

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	<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>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by 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>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</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>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</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 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.</p> <p>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.</p> <p>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</p>

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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 pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

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

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

Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. The presence of the p-hydroxy group on the benzene rings is thought to be responsible for the oestradiol mimicry.

Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review.

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

Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/ litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the 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"

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

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

A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, "these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls". Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells.[whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and 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.

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

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

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

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

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

Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity..

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

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

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	<p>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 <i>Drosophila</i>. The glycidyl ethers were generally mutagenic to bacteria.</p> <p>Dogs given daily doses of sodium phosphate dibasic for 9-22 weeks showed calcium deposits in the kidneys (nephrocalcinosis) with disseminated atrophy of the proximal tubule. Animals fed on sodium phosphate dibasic and potassium dihydrogen phosphate, in both short- and long-term studies, showed increased bone porosity; hyperparathyroidism and soft tissue calcification were also evident.</p> <p>Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.</p> <p>Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers. Xylene has been classed as a developmental toxin in some jurisdictions.</p> <p>Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.</p>	
834BLV Flame Retardant Epoxy (Part A)	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
bisphenol A diglycidyl ether resin, solid	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >1200 mg/kg <sup>[2]</sup>	Not Available
	Oral (Mouse) LD50; >500 mg/kg <sup>[2]</sup>	
alumina hydrate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation(Rat) LC50: >2.3 mg/l4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >2000 mg/kg <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
	Inhalation(Rat) LC50: >4.85 mg/l4h <sup>[1]</sup>	
	Oral (Rat) LD50: >=300<=2000 mg/kg <sup>[1]</sup>	
propylene glycol monomethyl ether - mixture of isomers	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit) 230 mg mild
	Oral (Rat) LD50: 3739 mg/kg <sup>[2]</sup>	Eye (rabbit) 500 mg/24 h. - mild
		Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin (rabbit) 500 mg open - mild
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
carbon black	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
Stoddard Solvent	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >3000 mg/kg <sup>[1]</sup>	Eye (hmn) 470 ppm/15m irrit.
	Inhalation(Rat) LC50: >5.5 mg/l4h <sup>[1]</sup>	Eye (rabbit) 500 mg/24h moderate
	Oral (Rat) LD50: >5000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
caprolactone, ethoxylated polyphosphates	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
C14-20 aliphatics (<=2% aromatics)	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[2]</sup>	Eye : Not irritating (OECD 405) *
	Inhalation(Rat) LC50: 4.6 mg/l4h <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: 7400 mg/kg <sup>[2]</sup>	Skin : Not irritating (OECD 404)*
		Skin: adverse effect observed (irritating) <sup>[1]</sup>

## 834BLV Flame Retardant Epoxy (Part A)

naphtha petroleum, light aromatic solvent	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation(Rat) LC50: >4.42 mg/L4h <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >4500 mg/kg <sup>[1]</sup>	
xylene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg <sup>[2]</sup>	Eye (human): 200 ppm irritant
	Inhalation(Rat) LC50: 5000 ppm4h <sup>[2]</sup>	Eye (rabbit): 5 mg/24h SEVERE
	Oral (Mouse) LD50: 2119 mg/kg <sup>[2]</sup>	Eye (rabbit): 87 mg mild
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit):500 mg/24h moderate
		Skin: adverse effect observed (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	

834BLV Flame Retardant Epoxy (Part A)	<p>Data demonstrate that during inhalation exposure, aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure.</p> <p>Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism.</p> <p>The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.</p> <p>For aluminium compounds:</p> <p>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).</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Systemic toxicity after repeated exposure</p> <p>No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium.</p> <p>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.</p> <p>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.</p> <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</p>
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high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate. This study was used by JECFA as key study to derive the PTWI.

#### Genotoxicity

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

#### Carcinogenicity

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

#### Neurodegenerative diseases

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

#### Contact sensitivity

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.

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 ionised to 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.

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. In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg).

**Reproductive and Developmental Toxicity:** BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg.

**Carcinogenicity:** IARC concluded that "there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals." Its overall evaluation was "Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3).

In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997).

**Genotoxicity:** In *S. typhimurium* strains TA100 and TA1535, BADGE (10-10,000 µg/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg).

**Immunotoxicity:** Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs

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**Consumer exposure** to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 µg/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 weight/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 µg/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

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	<p>contact with foodstuffs.</p> <p>The various members of the bisphenol family produce hormone like effects, seemingly as a result of binding to estrogen receptor-related receptors (ERRs; not to be confused with estrogen receptors)</p> <p>A suspected estrogen-related receptors (ERR) binding agent:</p> <p>Estrogen-related receptors (ERR, oestrogen-related receptors) are so named because of sequence homology with estrogen receptors but do not appear to bind estrogens or other tested steroid hormones. The ERR family have been demonstrated to control energy homeostasis, oxidative metabolism and mitochondrial biogenesis, while effecting mammalian physiology in the heart, brown adipose tissue, white adipose tissue, placenta, macrophages, and demonstrated additional roles in diabetes and cancer.</p> <p>ERRs bind enhancers throughout the genome where they exert effects on gene regulation</p> <p>Although their overall functions remain uncertain, they also share DNA-binding sites, co-regulators, and target genes with the conventional estrogen receptors ERalpha and ERbeta and may function to modulate estrogen signaling pathways.</p> <ul style="list-style-type: none"> <li>· ERR-alpha has wide tissue distribution but it is most highly expressed in tissues that preferentially use fatty acids as energy sources such as kidney, heart, brown adipose tissue, cerebellum, intestine, and skeletal muscle. ERRalpha has been detected in normal adrenal cortex tissues, in which its expression is possibly related to adrenal development, with a possible role in fetal adrenal function, in dehydroepiandrosterone (DHEAS) production in adrenarche, and also in steroid production of post-adrenarche/adult life. DHEA and other adrenal androgens such as androstenedione, although relatively weak androgens, are responsible for the androgenic effects of adrenarche, such as early pubic and axillary hair growth, adult-type body odor, increased oiliness of hair and skin, and mild acne.</li> <li>· ERR-beta is a nuclear receptor. Its function is unknown; however, a similar protein in mouse plays an essential role in placental development</li> <li>· ERR-gamma is a nuclear receptor that behaves as a constitutive activator of transcription. There is evidence that bisphenol A functions as an endocrine disruptor by binding strongly to ERRgamma BPA as well as its nitrated and chlorinated metabolites seems to binds strongly to ERR-gamma (dissociation constant = 5.5 nM), but not to the estrogen receptor (ER). BPA binding to ERR-gamma preserves its basal constitutive activity. Different expression of ERR-gamma in different parts of the body may account for variations in bisphenol A effects. For instance, ERR-gamma has been found in high concentration in the placenta, explaining reports of high bisphenol A accumulation there</li> </ul> <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>
<b>BISPHENOL A DIGLYCIDYL ETHER RESIN, SOLID</b>	<p>CAUTION: Epoxy resin products may contain sensitising glycidyl ethers, even when these are not mentioned in the information given for the product. The likely occurrence of these is greatly reduced in solid grades of the resin.</p>
<b>PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS</b>	<p>NOTE: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Fetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.</p> <p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<b>CARBON BLACK</b>	<p>Inhalation (rat) TCLo: 50 mg/m<sup>3</sup>/6h/90D-I Nil reported</p> <p><b>WARNING:</b> This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
<b>STODDARD SOLVENT</b>	<p>For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation.</p> <p>Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans.</p> <p>Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants).</p> <p>Reproductive toxicity: Animal studies show that high concentrations of toluene (&gt;0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus.</p> <p>Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials.</p> <p>Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.</p>
<b>CAPROLACTONE, ETHOXYLATED POLYPHOSPHATES</b>	<p>Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.</p> <p>Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.</p> <p>On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.</p> <p>Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.</p> <p>Ann-Therese Karlberg et al; Chem. Res. Toxicol. 2008, 21, 53-69</p> <p>Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.</p> <p>PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used</p> <p>Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology <a href="http://doi.org/10.5487/TR.2015.31.2.105">http://doi.org/10.5487/TR.2015.31.2.105</a></p>
<b>C14-20 ALIPHATICS (&lt;=2% AROMATICS)</b>	<p>*Exxsol D 100 SDS</p> <p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein</p>

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	<p>particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.</p>
<b>NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT</b>	<p>* [Devoe] .</p> <p>For C9 aromatics (typically trimethylbenzenes - TMBs)</p> <p>Acute Toxicity</p> <p>Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50 s range from 6,000 to 10,000 mg/m<sup>3</sup> for C9 aromatic naphtha and 18,000 to 24,000 mg/m<sup>3</sup> for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is &gt;4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.</p> <p>Irritation and Sensitization</p> <p>Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.</p> <p>Repeated Dose Toxicity</p> <p>Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m<sup>3</sup>). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOEL for systemic and/or neurotoxicity was 6,500 mg/m<sup>3</sup>, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m<sup>3</sup>) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.</p> <p>The NOEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m<sup>3</sup>. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m<sup>3</sup>). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m<sup>3</sup> (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m<sup>3</sup> for respiratory irritation and 250 ppm or 1230 mg/m<sup>3</sup> for systemic effects.</p> <p>Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.</p> <p>Mutagenicity</p> <p>In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with <i>Salmonella typhimurium</i> and <i>Escherichia coli</i> bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m<sup>3</sup>) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category</p> <p>Reproductive and Developmental Toxicity</p> <p>Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m<sup>3</sup>, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex /group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex /group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.</p> <p>Systemic Effects on Parental Generations:</p> <p>The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m<sup>3</sup>).</p> <p>Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m<sup>3</sup>). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation,, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m<sup>3</sup>), which excludes analysis of the highest concentration due to excessive mortality.</p> <p>Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m<sup>3</sup>) based on the body weights reductions observed in the F3 offspring.</p> <p>Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was</p>

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	observed at a concentration that was also associated with maternal toxicity.
<b>XYLENE</b>	<p>Reproductive effector in rats</p> <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>The substance is classified by IARC as Group 3:</p> <p><b>NOT</b> classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
<b>834BLV Flame Retardant Epoxy (Part A) &amp; BISPHENOL A DIGLYCIDYL ETHER RESIN, SOLID</b>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>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>In vitro cell models were used to evaluate the ability of 22 bisphenols (BPs) to induce or inhibit estrogenic and androgenic activity. BPA, Bisphenol AF (BPAF), bisphenol Z (BPZ), bisphenol C (BPC), tetramethyl bisphenol A (TMBPA), bisphenol S (BPS), bisphenol E (BPE), 4,4-bisphenol F (4,4-BPF), bisphenol AP (BPAP), bisphenol B (BPB), tetrachlorobisphenol A (TCBPA), and benzylparaben (PHBB) induced estrogen receptor (ER)alpha and/or ERbeta-mediated activity. With the exception of BPS, TCBPA, and PHBB, these same BPs were also androgen receptor (AR) antagonists. Only 3 BPs were found to be ER antagonists. Bisphenol P (BPP) selectively inhibited ERbeta-mediated activity and 4-(4-phenylmethoxyphenyl)sulfonylphenol (BPS-MPE) and 2,4-bisphenol S (2,4-BPS) selectively inhibited ERalpha-mediated activity. None of the BPs induced AR-mediated activity.</p>
<b>834BLV Flame Retardant Epoxy (Part A) &amp; NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT</b>	<p>For trimethylbenzenes:</p> <p>Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption. 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid. The major routes of excretion of 1,2,4-trimethylbenzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.</p> <p><b>Acute Toxicity</b> Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis. High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness. The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1).</p> <p>2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes. Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg). Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels. No effects were reported for rats exposed to a mixture of trimethylbenzenes at 1700 ppm for 10 to 21 days.</p> <p><b>Neurotoxicity</b> 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes.</p> <p>Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.</p> <p><b>Subchronic/Chronic Toxicity</b> Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene.</p> <p>Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia.</p> <p><b>Genotoxicity:</b> Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella typhimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation.</p> <p><b>Developmental/Reproductive Toxicity:</b> A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established. Developmental toxicity, including possible developmental neurotoxicity, was evident in rats in a 3-generation reproductive study.</p> <p>No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethylbenzenes, 4-6 hours/day, 5 days/week over one generation.</p>
<b>834BLV Flame Retardant Epoxy (Part A) &amp; PROPYLENE GLYCOL MONOMETHYL</b>	<p>for propylene glycol ethers (PGEs):</p> <p>Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).</p>

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ETHER - MIXTURE OF ISOMERS	<p>Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.</p> <p>Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).</p> <p>This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.</p> <p>Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.</p> <p>As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.</p> <p>As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from &gt;3,000 mg/kg (PnB) to &gt;5,000 mg/kg (DPMA). Dermal LD50s are all &gt; 2,000 mg/kg (PnB, &amp; DPnB; where no deaths occurred), and ranging up to &gt;15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is &gt;2,040 mg/m3. For PnB, the 4-hour LC50 was &gt;651 ppm (&gt;3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating.</p> <p>None are skin sensitizers.</p> <p>In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).</p> <p>Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.</p> <p>One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.</p> <p>In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.</p> <p>The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. <i>In vitro</i>, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic <i>in vivo</i>. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.</p>
BISPHENOL A DIGLYCIDYL ETHER RESIN, SOLID & PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
BISPHENOL A DIGLYCIDYL ETHER RESIN, SOLID & ALUMINA HYDRATE & PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS & CARBON BLACK	No significant acute toxicological data identified in literature search.
PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

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

Continued...

## 834BLV Flame Retardant Epoxy (Part A)

✔ – Data available to make classification

## 11.2 Information on other hazards

## 11.2.1. Endocrine disrupting properties

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

## 11.2.2. Other information

See Section 11.1

## SECTION 12 Ecological information

## 12.1. Toxicity

834BLV Flame Retardant Epoxy (Part A)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
bisphenol A diglycidyl ether resin, solid	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	24h	Crustacea	3mg/l	Not Available
	LC50	96h	Fish	2.4mg/l	Not Available
	EC50	48h	Crustacea	~2mg/l	2
alumina hydrate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
	EC50	72h	Algae or other aquatic plants	0.0169mg/l	2
	EC50	96h	Algae or other aquatic plants	0.0054mg/l	2
	LC50	96h	Fish	0.57mg/l	2
	EC50	48h	Crustacea	>0.065mg/l	4
ammonium polyphosphate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	3.57mg/l	2
	LC50	96h	Fish	70mg/l	4
	EC50	72h	Algae or other aquatic plants	>97.1mg/l	2
	EC50	48h	Crustacea	90.89mg/l	4
propylene glycol monomethyl ether - mixture of isomers	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	100mg/l	1
	EC50	72h	Algae or other aquatic plants	>1000mg/l	2
	EC50	48h	Crustacea	373mg/l	2
	NOEC(ECx)	336h	Fish	47.5mg/l	2
	EC50	96h	Algae or other aquatic plants	>1000mg/l	2
carbon black	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	EC50	48h	Crustacea	33.076-41.968mg/l	4
	NOEC(ECx)	24h	Crustacea	3200mg/l	1
Stoddard Solvent	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	3072h	Fish	1mg/l	1
	LC50	96h	Fish	2.2mg/l	4
	NOEC(ECx)	720h	Fish	0.02mg/l	2
	EC50	96h	Algae or other aquatic plants	0.277mg/l	2
	LC50	96h	Fish	0.14mg/l	2
caprolactone, ethoxylated polyphosphates	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...



## 834BLV Flame Retardant Epoxy (Part A)

C14-20 aliphatics (<=2% aromatics)	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	<0.03mg/l	1
	NOEC(ECx)	3072h	Fish	1mg/l	1
	LC50	96h	Fish	2.2mg/l	4
naphtha petroleum, light aromatic solvent	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
	EC50	72h	Algae or other aquatic plants	19mg/l	1
	EC50	96h	Algae or other aquatic plants	64mg/l	2
xylene	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	2.6mg/l	2
	EC50	72h	Algae or other aquatic plants	4.6mg/l	2
	EC50	48h	Crustacea	1.8mg/l	2
Legend:	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
	LC50	96h	Fish	2.6mg/l	2
	EC50	72h	Algae or other aquatic plants	4.6mg/l	2
	EC50	48h	Crustacea	1.8mg/l	2
	NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2

**Legend:** Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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.

For 1,2,4 - Trimethylbenzene:

Half-life (hr) air: 0.48-16;

Half-life (hr) H<sub>2</sub>O surface water: 0.24 -672;

Half-life (hr) H<sub>2</sub>O ground: 336-1344;

Half-life (hr) soil: 168-672;

Henry's Pa m<sup>3</sup>/mol: 385 -627;

Bioaccumulation: not significant. 1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance.

Atmospheric Fate: 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs. Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals. Reaction also occurs with ozone but very slowly (half life 8820 days).

Aquatic Fate: 1,2,4-Trimethylbenzene volatilizes rapidly from surface waters with volatilization half-life from a model river calculated to be 3.4 hours. Biodegradation of 1,2,4-trimethylbenzene has been noted in both seawater and ground water. Various strains of *Pseudomonas* can biodegrade 1,2,4-trimethylbenzene.

Terrestrial Fate: 1,2,4-Trimethylbenzene also volatilizes from soils however; moderate adsorption to soils and sediments may occur. Volatilization is the major route of removal of 1,2,4-trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.

Ecotoxicity: No significant bioaccumulation has been noted. 1,2,4-Trimethylbenzene is moderately toxic to fathead minnow and slightly toxic to dungeness crab. 1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms. No stress was observed in rainbow trout, sea lamprey and *Daphnia magna* water fleas. The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.

For Aromatic Substances Series:

Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.

Atmospheric Fate: PAHs are "semi-volatile substances" which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.

Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes > naphthalenes. Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.

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

## 834BLV Flame Retardant Epoxy (Part A)

slight acute toxicity, no mutagenicity, and weak oestrogenic activity as well as BPA. Some of the BPs showed considerably higher oestrogenic activity than BPA, and others exhibited much lower activity. Bisphenol S (bis(4-hydroxydiphenyl)sulfone) and bis(4-hydroxyphenyl)sulfide showed oestrogenic activity.

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

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

For 1,2-Butylene oxide (Ethyloxirane):

log Kow values of 0.68 and 0.86. BAF and BCF : 1 to 17 L/kg.

Aquatic 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. Volatilization of ethyloxirane from water surfaces would be expected. 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. Models have predicted a biodegradation half-life in water of 15 days.

Terrestrial Fate: When released to soil, ethyloxirane is expected to have low adsorption and thus very high mobility. Volatilization from moist soil and dry soil surfaces is expected.

Ethyloxirane is not expected to be persistent in soil.

Atmospheric Fate: It is expected that ethyloxirane exists solely as a vapor in ambient atmosphere. Ethyloxirane may also be removed from the atmosphere by wet deposition processes. 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).

Ecotoxicity - The potential for bioaccumulation of ethyloxirane in organisms is likely to be low and has low to moderate toxicity to aquatic organisms. Ethyloxirane is acutely toxic to water fleas and toxicity values for bacteria are close to 5000 mg/L. For algae, toxicity values exceed 500 mg/L.

For Xylenes:

log Koc : 2.05-3.08; Koc : 25.4-204; Half-life (hr) air : 0.24-42; Half-life (hr) H<sub>2</sub>O surface water : 24-672; Half-life (hr) H<sub>2</sub>O ground : 336-8640; Half-life (hr) soil : 52-672; Henry's Pa m<sup>3</sup>/mol : 637-879; Henry's atm m<sup>3</sup>/mol - 7.68E-03; BOD 5 if unstated - 1.4,1%; COD - 2.56,13% ThOD - 3.125 : BCF : 23; log BCF : 1.17-2.41.

Environmental Fate: Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. Soil - Xylenes are expected to have moderate mobility in soil evaporating rapidly from soil surfaces. The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. Xylene can remain below the soil surface for several days and may travel through the soil profile and enter groundwater. Soil and water microbes may transform it into other, less harmful compounds, although this happens slowly. It is not clear how long xylene remains trapped deep underground in soil or groundwater, but it may be months or years.

Atmospheric Fate: Xylene evaporates quickly into the air from surface soil and water and can remain in the air for several days until it is broken down by sunlight into other less harmful chemicals. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylene may contribute to photochemical smog formation. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzyl nitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

Aquatic Fate: p-xylene may adsorb to suspended solids and sediment in water and is expected to volatilise from water surfaces. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. Measurements taken from goldfish, eels and clams indicate that bioconcentration in aquatic organisms is low. Photo-oxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. p-Xylene is biodegradable and has been observed to degrade in pond water however; it is unclear if it degrades in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high. Ecotoxicity: Xylenes are slightly toxic to fathead minnow, rainbow trout and bluegill and not acutely toxic to water fleas. For Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/L. and Gammarus lacustris LC50 (48 h): 0.6 mg/L.

For Ammonia:

Atmospheric Fate: Ammonia reacts rapidly with available acids (mainly sulfuric, nitric, and sometimes hydrochloric acid) to form the corresponding salts. Ammonia is persistent in the air.

Aquatic Fate: Biodegrades rapidly to nitrate, producing a high oxygen demand. Non-persistent in water (half-life 2 days).

Ecotoxicity: Moderately toxic to fish under normal temperature and pH conditions and harmful to aquatic life at low concentrations. Does not concentrate in food chain.

For Phosphate: 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.

Aquatic Fate: Lakes overloaded with phosphates is the primary catalyst for the 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 an anoxic condition 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, aluminum cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminum in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminum can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminum in the environment will depend on the ligands present and the pH.

The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion, [Al(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 aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)<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 aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)<sub>3</sub>, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.

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

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

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

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

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

Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their



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

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

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

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

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

Crustaceans LC50 (48 h): 1 sp 2.3-36.9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

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

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

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

Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 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 resin, solid	HIGH	HIGH
propylene glycol monomethyl ether - mixture of isomers	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)

**12.3. Bioaccumulative potential**

Ingredient	Bioaccumulation
bisphenol A diglycidyl ether resin, solid	LOW (LogKOW = 2.6835)
propylene glycol monomethyl ether - mixture of isomers	LOW (BCF = 2)
Stoddard Solvent	LOW (BCF = 159)
C14-20 aliphatics (<=2% aromatics)	LOW (BCF = 159)
xylene	MEDIUM (BCF = 740)

**12.4. Mobility in soil**

Ingredient	Mobility
bisphenol A diglycidyl ether resin, solid	LOW (KOC = 51.43)
propylene glycol monomethyl ether - mixture of isomers	HIGH (KOC = 1)

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

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

PBT Criteria fulfilled?

No

Continued...

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vPvB

No

## 12.6. Endocrine disrupting properties

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

## 12.7. Other adverse effects

No evidence of ozone depleting properties were found in the current literature.




## SECTION 13 Disposal considerations

## 13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> <li>Containers may still present a chemical hazard/ danger when empty.</li> <li>Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.</li> <li>Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> </ul> <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>Reduction</li> <li>Reuse</li> <li>Recycling</li> <li>Disposal (if all else fails)</li> </ul> <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> <li><b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>It may be necessary to collect all wash water for treatment before disposal.</li> <li>In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>Where in doubt contact the responsible authority.</li> </ul> <p>Removal of bisphenol A (BPA) from aqueous solutions was accomplished by adsorption of enzymatically generated quinone derivatives on chitosan beads. The use of chitosan in the form of beads was found to be more effective because heterogeneous removal of BPA with chitosan beads was much faster than homogeneous removal of BPA with chitosan solutions, and the removal efficiency was enhanced by increasing the amount of chitosan beads dispersed in the BPA solutions and BPA was completely removed by quinone adsorption in the presence of chitosan beads more than 0.10 cm<sup>3</sup>/cm<sup>3</sup>. In addition, a variety of bisphenol derivatives were completely or effectively removed by the procedure constructed in this study, although the enzyme dose or the amount of chitosan beads was further increased as necessary for some of the bisphenol derivatives used.</p> <p>M. Suzuki, and E Musashi J Appl Polym Sci, 118(2):721 - 732; October 2010</p> <ul style="list-style-type: none"> <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

	 Limited Quantity: 834BLV-450ML, 834BLV-3L
	  Sizes greater than 5L: 834BLV-60L

## Land transport (ADR-RID)

14.1. UN number or ID number	3082				
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A diglycidyl ether resin, solid)				
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>Class</td><td>9</td></tr> <tr> <td>Subsidiary risk</td><td>Not Applicable</td></tr> </table>	Class	9	Subsidiary risk	Not Applicable
Class	9				
Subsidiary risk	Not Applicable				
14.4. Packing group	III				

Continued...

## 834BLV Flame Retardant Epoxy (Part A)

14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Hazard identification (Kemler)	90
	Classification code	M6
	Hazard Label	9
	Special provisions	274 335 375 601
	Limited quantity	5 L
	Tunnel Restriction Code	3 (-)

## Air transport (ICAO-IATA / DGR)

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

## Sea transport (IMDG-Code / GGVSee)

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

## Inland waterways transport (ADN)

14.1. UN number	3082	
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A diglycidyl ether resin, solid)	
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. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
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Continued...

## 834BLV Flame Retardant Epoxy (Part A)

Product name	Group
bisphenol A diglycidyl ether resin, solid	Not Available
alumina hydrate	Not Available
ammonium polyphosphate	Not Available
propylene glycol monomethyl ether - mixture of isomers	Not Available
carbon black	Not Available
Stoddard Solvent	Not Available
caprolactone, ethoxylated polyphosphates	Not Available
C14-20 aliphatics (<=2% aromatics)	Not Available
naphtha petroleum, light aromatic solvent	Not Available
xylene	Not Available

## 14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
bisphenol A diglycidyl ether resin, solid	Not Available
alumina hydrate	Not Available
ammonium polyphosphate	Not Available
propylene glycol monomethyl ether - mixture of isomers	Not Available
carbon black	Not Available
Stoddard Solvent	Not Available
caprolactone, ethoxylated polyphosphates	Not Available
C14-20 aliphatics (<=2% aromatics)	Not Available
naphtha petroleum, light aromatic solvent	Not Available
xylene	Not Available

## SECTION 15 Regulatory information

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

## bisphenol A diglycidyl ether resin, solid is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List  
Great Britain GB mandatory classification and labelling list (GB MCL)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

## alumina hydrate is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

## ammonium polyphosphate is found on the following regulatory lists

Not Applicable

## propylene glycol monomethyl ether - mixture of isomers is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List  
Great Britain GB mandatory classification and labelling list (GB MCL)

UK Workplace Exposure Limits (WELs).

## carbon black is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List  
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs  
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)  
UK Workplace Exposure Limits (WELs).

## Stoddard Solvent is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List  
Great Britain GB mandatory classification and labelling list (GB MCL)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

## caprolactone, ethoxylated polyphosphates is found on the following regulatory lists

Not Applicable

## C14-20 aliphatics (&lt;=2% aromatics) is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List  
Great Britain GB mandatory classification and labelling list (GB MCL)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

## naphtha petroleum, light aromatic solvent is found on the following regulatory lists

## 834BLV Flame Retardant Epoxy (Part A)

Chemical Footprint Project - Chemicals of High Concern List  
Great Britain GB mandatory classification and labelling list (GB MCL)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC  
Monographs - Not Classified as Carcinogenic

#### xylene is found on the following regulatory lists

Great Britain GB mandatory classification and labelling list (GB MCL)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC  
Monographs - Not Classified as Carcinogenic

UK Workplace Exposure Limits (WELs).

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

#### Information according to 2012/18/EU (Seveso III):

Seveso Category	E2
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#### 15.2. Chemical safety assessment

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

#### National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (caprolactone, ethoxylated polyphosphates)
Canada - DSL	No (caprolactone, ethoxylated polyphosphates)
Canada - NDSL	No (bisphenol A diglycidyl ether resin, solid; alumina hydrate; ammonium polyphosphate; carbon black; Stoddard Solvent; caprolactone, ethoxylated polyphosphates; C14-20 aliphatics (<=2% aromatics); naphtha petroleum, light aromatic solvent; xylene)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (caprolactone, ethoxylated polyphosphates)
Japan - ENCS	No (ammonium polyphosphate; caprolactone, ethoxylated polyphosphates)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (caprolactone, ethoxylated polyphosphates)
USA - TSCA	No (caprolactone, ethoxylated polyphosphates)
Taiwan - TCSI	Yes
Mexico - INSQ	No (ammonium polyphosphate; caprolactone, ethoxylated polyphosphates)
Vietnam - NCI	Yes
Russia - FBEPH	No (caprolactone, ethoxylated polyphosphates)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

#### SECTION 16 Other information

Revision Date	13/06/2023
Initial Date	13/06/2023

#### Full text Risk and Hazard codes

<b>H226</b>	Flammable liquid and vapour.
<b>H304</b>	May be fatal if swallowed and enters airways.
<b>H312</b>	Harmful in contact with skin.
<b>H318</b>	Causes serious eye damage.
<b>H332</b>	Harmful if inhaled.
<b>H336</b>	May cause drowsiness or dizziness.
<b>H340</b>	May cause genetic defects.
<b>H350</b>	May cause cancer.
<b>H351</b>	Suspected of causing cancer.
<b>H413</b>	May cause long lasting harmful effects to aquatic life.

#### Other information

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

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

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

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

#### Definitions and abbreviations

PC - TWA: Permissible Concentration-Time Weighted Average

PC - STEL: Permissible Concentration-Short Term Exposure Limit

Continued...

## 834BLV Flame Retardant Epoxy (Part A)

IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit,  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 ES: Exposure Standard  
 OSF: Odour Safety Factor  
 NOAEL :No Observed Adverse Effect Level  
 LOAEL: Lowest Observed Adverse Effect Level  
 TLV: Threshold Limit Value  
 LOD: Limit Of Detection  
 OTV: Odour Threshold Value  
 BCF: BioConcentration Factors  
 BEI: Biological Exposure Index  
 AIIC: Australian Inventory of Industrial Chemicals  
 DSL: Domestic Substances List  
 NDSL: Non-Domestic Substances List  
 IECSC: Inventory of Existing Chemical Substance in China  
 EINECS: European INventory of Existing Commercial chemical Substances  
 ELINCS: European List of Notified Chemical Substances  
 NLP: No-Longer Polymers  
 ENCS: Existing and New Chemical Substances Inventory  
 KECI: Korea Existing Chemicals Inventory  
 NZIoC: New Zealand Inventory of Chemicals  
 PICCS: Philippine Inventory of Chemicals and Chemical Substances  
 TSCA: Toxic Substances Control Act  
 TCSI: Taiwan Chemical Substance Inventory  
 INSQ: Inventario Nacional de Sustancias Químicas  
 NCI: National Chemical Inventory  
 FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

**Classification and procedure used to derive the classification for mixtures according to Regulation (EC) 1272/2008 [CLP]**

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	Classification Procedure
Hazardous to the Aquatic Environment Long-Term Hazard Category 2, H411	Calculation method
Skin Corrosion/Irritation Category 2, H315	Calculation method
Serious Eye Damage/Eye Irritation Category 2, H319	Calculation method
Sensitisation (Skin) Category 1, H317	Calculation method
, EUH210	Expert judgement