



Kit Revision Date: 07/03/2022

## 8349TFM THERMAL ADHESIVE KIT

### MG Chemicals Multipart Product Kit

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

#### **Kit Content**

<i>Part</i>	<i>Product Name</i>	<i>Product Use</i>
A	8349TFM-A	Thermally conductive adhesive resin
B	8349TFM-B	Thermally conductive adhesive hardener

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

#### **Transportation Instruction**

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



## 8349TFM-A Thermal Adhesive

### MG Chemicals UK Limited

Version No: A-2.00

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

Issue Date: 07/03/2022

Revision Date: 07/03/2022

L.REACH.GB.EN

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

### 1.1. Product Identifier

Product name	8349TFM-A
Synonyms	SDS Code: 8349TFM-Part A; 8349TFM-25ML, 8349TFM-50ML   UFI:TEQ0-Y0SS-6008-17J8
Other means of identification	Thermal Adhesive

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

Relevant identified uses	Thermally conductive adhesive resin
Uses advised against	Not Applicable

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

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

### 1.4. Emergency telephone number

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

## SECTION 2 Hazards identification

### 2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H411 - Hazardous to the Aquatic Environment Long-Term Hazard Category 2, 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 statement(s)

EUH205	Contains epoxy constituents. May produce an allergic reaction.
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## 8349TFM-A Thermal Adhesive

## Precautionary statement(s) Prevention

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

## Precautionary statement(s) Response

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

## Precautionary statement(s) Storage

Not Applicable

## Precautionary statement(s) Disposal

<b>P501</b>	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

Cumulative effects may result following exposure\*.

May produce discomfort of the respiratory system\*.

Limited evidence of a carcinogenic effect\*.

Possible respiratory sensitizer\*.

<b>bisphenol F diglycidyl ether copolymer</b>	Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors
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## 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
1.21645-51-2 2.244-492-7 3.Not Available 4.Not Available	50	<u>aluminium hydroxide</u>	Serious Eye Damage/Eye Irritation Category 2; H319 [1]	Not Available	Not Available
1.28064-14-4 2.Not Available 3.Not Available 4.Not Available	30	<u>bisphenol E diglycidyl ether copolymer [e]</u>	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, EUH205 [1]	Not Available	Not Available
1.1344-28-1. 2.215-691-6 3.Not Available 4.Not Available	7	<u>aluminium oxide</u>	Not Applicable	Not Available	Not Available
1.68333-79-9 2.269-789-9 3.Not Available 4.Not Available	7	<u>ammonium polyphosphate</u>	Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H413 [1]	Not Available	Not Available
1.17557-23-2 2.241-536-7 3.603-094-00-7 4.Not Available	3	<u>neopentyl glycol diglycidyl ether</u>	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1; H315, H317 [2]	Not Available	Not Available
1.70700-21-9 2.Not Available 3.Not Available 4.Not Available	1	<u>monomethyl phosphate ethoxylated</u>	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H315, H318, H413 [1]	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

**Legend:**

1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L; \* EU IOELVs available; [e] Substance identified as having endocrine disrupting properties

## 8349TFM-A Thermal Adhesive

## 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.

Treat symptomatically.

## SECTION 5 Firefighting measures

## 5.1. Extinguishing media

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

## 5.2. Special hazards arising from the substrate or mixture

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

## SECTION 6 Accidental release measures

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

Continued...

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See section 8

**6.2. Environmental precautions**

See section 12

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

<b>Minor Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ In the event of a spill of a reactive diluent, the focus is on containing the spill to prevent contamination of soil and surface or ground water.</li> <li>▶ If irritating vapors are present, an approved air-purifying respirator with organic vapor canister is recommended for cleaning up spills and leaks.</li> <li>▶ For small spills, reactive diluents should be absorbed with sand.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>▶ Wipe up.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> </ul>																																																																											
<b>Major Spills</b>	<p>Environmental hazard - contain spillage. Chemical Class: phenols and cresols For release onto land: recommended sorbents listed in order of priority.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="text-align: left;">SORBENT TYPE</th> <th style="text-align: left;">RANK</th> <th style="text-align: left;">APPLICATION</th> <th style="text-align: left;">COLLECTION</th> <th style="text-align: left;">LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5"><b>LAND SPILL - SMALL</b></td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>shovel</td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>wood fiber - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>foamed glass - pillow</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R, W, P, DGC</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>wood fibre - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, W, P, DGC</td> </tr> <tr> <td colspan="5"><b>LAND SPILL - MEDIUM</b></td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>blower</td> <td>skid loader</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>2</td> <td>throw</td> <td>skid loader</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skid loader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skid loader</td> <td>R, SS, DGC</td> </tr> <tr> <td>wood fiber - particulate</td> <td>4</td> <td>blower</td> <td>skid loader</td> <td>R, W, P, DGC</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skid loader</td> <td>R, I, W, P, DGC</td> </tr> </tbody> </table> <p>Legend DGC: Not effective where ground cover is dense R: Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT: Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988 Industrial spills or releases of reactive diluents are infrequent and generally contained. If a large spill does occur, the material should be captured, collected, and reprocessed or disposed of according to applicable governmental requirements. An approved air-purifying respirator with organic-vapor canister is recommended for emergency work. Moderate hazard.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Absorb remaining product with sand, earth or vermiculite.</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	<b>LAND SPILL - SMALL</b>					cross-linked polymer - particulate	1	shovel	shovel	R, W, SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	wood fiber - pillow	1	throw	pitchfork	R, P, DGC, RT	foamed glass - pillow	2	shovel	shovel	R, W, P, DGC	sorbent clay - particulate	2	shovel	shovel	R, I, P	wood fibre - particulate	3	shovel	shovel	R, W, P, DGC	<b>LAND SPILL - MEDIUM</b>					cross-linked polymer - particulate	1	blower	skid loader	R, W, SS	cross-linked polymer - pillow	2	throw	skid loader	R, DGC, RT	sorbent clay - particulate	3	blower	skid loader	R, I, P	polypropylene - particulate	3	blower	skid loader	R, SS, DGC	wood fiber - particulate	4	blower	skid loader	R, W, P, DGC	expanded mineral - particulate	4	blower	skid loader	R, I, W, P, DGC
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**6.4. Reference to other sections**

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

**SECTION 7 Handling and storage****7.1. Precautions for safe handling**

<b>Safe handling</b>	▶ Avoid all personal contact, including inhalation.
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Continued...

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

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

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Metal can or drum</li> <li>▶ Packaging as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
<b>Storage incompatibility</b>	<p>For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate.</p> <p>Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.</p> <p>Epoxides:</p> <ul style="list-style-type: none"> <li>▶ are highly reactive with acids, bases, and oxidising and reducing agents.</li> <li>▶ react, possibly violently, with anhydrous metal chlorides, ammonia, amines and group 1 metals.</li> <li>▶ may polymerise in the presence of peroxides or heat - polymerisation may be violent</li> <li>▶ may react, possibly violently, with water in the presence of acids and other catalysts.</li> <li>▶ Phenols are incompatible with strong reducing substances such as hydrides, nitrides, alkali metals, and sulfides.</li> <li>▶ Avoid use of aluminium, copper and brass alloys in storage and process equipment.</li> <li>▶ Heat is generated by the acid-base reaction between phenols and bases.</li> <li>▶ Phenols are sulfonated very readily (for example, by concentrated sulfuric acid at room temperature), these reactions generate heat.</li> <li>▶ Phenols are nitrated very rapidly, even by dilute nitric acid.</li> <li>▶ Nitrated phenols often explode when heated. Many of them form metal salts that tend toward detonation by rather mild shock.</li> </ul> <p>Glycidyl ethers:</p> <ul style="list-style-type: none"> <li>▶ may form unstable peroxides on storage in air, light, sunlight, UV light or other ionising radiation, trace metals - inhibitor should be maintained at adequate levels</li> <li>▶ may polymerise in contact with heat, organic and inorganic free radical producing initiators</li> <li>▶ may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines</li> <li>▶ react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide</li> <li>▶ attack some forms of plastics, coatings, and rubber</li> </ul> <p>Reactive diluents are stable under recommended storage conditions, but can decompose at elevated temperatures. In some cases, decomposition can cause pressure build-up in closed systems.</p> <ul style="list-style-type: none"> <li>▶ Avoid cross contamination between the two liquid parts of product (kit).</li> <li>▶ If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur.</li> <li>▶ This excess heat may generate toxic vapour</li> <li>▶ Avoid reaction with amines, mercaptans, strong acids and oxidising agents</li> </ul>

## 7.3. Specific end use(s)

See section 1.2

## SECTION 8 Exposure controls / personal protection

## 8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
aluminium hydroxide	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
aluminium oxide	Dermal 0.84 mg/kg bw/day (Systemic, Chronic) Inhalation 3 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 3 mg/m <sup>3</sup> (Local, Chronic)	74.9 µg/L (Water (Fresh)) 20 mg/L (STP)

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Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
	<i>Dermal 0.3 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 0.75 mg/m<sup>3</sup> (Systemic, Chronic) *</i> <i>Oral 1.32 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 0.75 mg/m<sup>3</sup> (Local, Chronic) *</i>	
ammonium polyphosphate	Inhalation 18.06 mg/m <sup>3</sup> (Systemic, Chronic) <i>Inhalation 4.45 mg/m<sup>3</sup> (Systemic, Chronic) *</i> <i>Oral 1.28 mg/kg bw/day (Systemic, Chronic) *</i>	Not Available
carbon black	Inhalation 1 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 0.5 mg/m <sup>3</sup> (Local, Chronic) <i>Inhalation 0.06 mg/m<sup>3</sup> (Systemic, Chronic) *</i>	1 mg/L (Water (Fresh)) 0.1 mg/L (Water - Intermittent release) 10 mg/L (Water (Marine))

\* Values for General Population

## Occupational Exposure Limits (OEL)

## INGREDIENT DATA

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

## Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
aluminium hydroxide	8.7 mg/m <sup>3</sup>	73 mg/m <sup>3</sup>	440 mg/m <sup>3</sup>
bisphenol F diglycidyl ether copolymer	30 mg/m <sup>3</sup>	330 mg/m <sup>3</sup>	2,000 mg/m <sup>3</sup>
aluminium oxide	15 mg/m <sup>3</sup>	170 mg/m <sup>3</sup>	990 mg/m <sup>3</sup>
carbon black	9 mg/m <sup>3</sup>	99 mg/m <sup>3</sup>	590 mg/m <sup>3</sup>

Ingredient	Original IDLH	Revised IDLH
aluminium hydroxide	Not Available	Not Available
bisphenol F diglycidyl ether copolymer	Not Available	Not Available
aluminium oxide	Not Available	Not Available
ammonium polyphosphate	Not Available	Not Available
neopentyl glycol diglycidyl ether	Not Available	Not Available
monomethyl phosphate ethoxylated	Not Available	Not Available
carbon black	1,750 mg/m <sup>3</sup>	Not Available

## Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
aluminium hydroxide	E	≤ 0.01 mg/m <sup>3</sup>
bisphenol F diglycidyl ether copolymer	E	≤ 0.1 ppm
neopentyl glycol diglycidyl ether	E	≤ 0.1 ppm
monomethyl phosphate ethoxylated	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

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m<sup>3</sup> over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

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

For aluminium oxide:

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

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

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

Continued...

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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 epichlorohydrin

Odour Threshold Value: 0.08 ppm

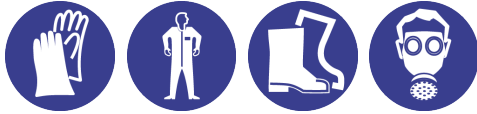
NOTE: Detector tubes for epichlorohydrin, measuring in excess of 5 ppm, are commercially available.

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

Odour Safety Factor (OSF)

OSF=0.54 (EPICHLOROHYDRIN)

## 8.2. Exposure controls

<p><b>8.2.1. Appropriate engineering controls</b></p>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.</p> <table border="1" data-bbox="391 896 1484 1153"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="391 1209 1085 1377"> <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. Personal protection</b></p>																					
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> </ul>																				
<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>																				



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

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

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

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

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

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

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

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

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

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

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

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

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

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

The performance, based on breakthrough times, of:

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

As defined in ASTM F-739-96

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

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

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

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

**Body protection** See Other protection below

**Other protection**

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

### Respiratory protection

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

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 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

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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

<b>Appearance</b>	Dark		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	1.73
<b>Odour</b>	Slight	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Available	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	>20.5
<b>Initial boiling point and boiling range (°C)</b>	>150	<b>Molecular weight (g/mol)</b>	Not Available
<b>Flash point (°C)</b>	150	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Applicable	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (Not Available%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available
<b>Nanoform Solubility</b>	Not Available	<b>Nanoform Particle Characteristics</b>	Not Available
<b>Particle Size</b>	Not Available		

### 9.2. Other information

Not Available

## SECTION 10 Stability and reactivity

<b>10.1.Reactivity</b>	See section 7.2
<b>10.2. Chemical stability</b>	<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>
<b>10.3. Possibility of hazardous reactions</b>	See section 7.2
<b>10.4. Conditions to avoid</b>	See section 7.2
<b>10.5. Incompatible materials</b>	See section 7.2
<b>10.6. Hazardous decomposition products</b>	See section 5.3

## SECTION 11 Toxicological information

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## 11.1. Information on toxicological effects

Inhaled	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>In animal testing, exposure to aerosols of some reactive diluents (notably o-cresol glycidyl ether, CAS RN: 2210-79-9) has been reported to affect the adrenal gland, central nervous system, kidney, liver, ovaries, spleen, testes, thymus, and respiratory tract.</p> <p>Inhalation hazard is increased at higher temperatures.</p>
Ingestion	<p>Reactive diluents exhibit a range of ingestion hazards. Small amounts swallowed incidental to normal handling operations are not likely to cause injury. However, swallowing larger amounts may cause injury.</p> <p>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>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>High molecular weight material; on single acute exposure would be expected to pass through gastrointestinal tract with little change / absorption. Occasionally accumulation of the solid material within the alimentary tract may result in formation of a bezoar (concretion), producing discomfort.</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p> <p>Skin contact with reactive diluents may cause slight to moderate irritation with local redness. Repeated or prolonged skin contact may cause burns.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
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.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury.</p>
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>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>The polymer contained in this product has reactive groups (aldehydes and phenolics) generally considered to be of moderate concern (US EPA). In general, aldehydes are reactive. Due to their water solubility and severe irritant properties, the lower aldehydes attack exposed moist tissue, particularly the eyes and mucous membranes of the upper respiratory tract. Aldehydes can also be skin and respiratory sensitisers, e.g. formaldehyde and glutaraldehyde. Lower solubility aldehydes can penetrate further into the lungs. Skin sensitisation reactions have been noted after exposure to urea-formaldehyde resins.</p> <p>Phenolic groups with ortho and para positions free from substitution are reactive; this is because the ortho and para positions on the aromatic ring are highly activated by the phenolic hydroxyl group and are therefore readily substituted.</p> <p>The acute toxicity of polymers of the group with a molecular weight above 1000 is expected to be lower. Whilst it is generally accepted that polymers with a molecular weight exceeding 1000 are unlikely to pass through biological membranes, oligomers with lower molecular weight and specifically, those with a molecular weight below 500, may. Estimations based on a 'highly' dispersed polymer population suggest that a polymer of approximate molecular weight 1000 could contain no more than one reactive group of moderate concern for it to be regulated as a polymer of low concern (a so-called PLC) 2500). Polymers with a molecular weight above 10000 are generally considered to be PLCs because these are not expected to be absorbed by biological systems. The choice of 10000 as a cut-off value is thought to provide a safety factor of 100, regarded as reasonable in light of limited data, duration of studies, dose levels at which effects are seen, and extrapolation from animals to humans.</p> <p>All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.</p> <p>Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity..</p>

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

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

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

Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.

Very fine 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.

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

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

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

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

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.

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.

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

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

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

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 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]

## 8349TFM-A Thermal Adhesive

In chronic animal studies inorganic polyphosphates produced growth inhibition, increased kidney weights (with calcium deposition and desquamation), bone decalcification, parathyroid hypertrophy and hyperplasia, inorganic phosphaturia, hepatic focal necrosis and alterations to the size of muscle fibres.

Inorganic phosphates are not genotoxic in bacterial systems nor are they carcinogenic in rats. No reproductive or developmental toxicity was seen in studies using rats exposed to sodium hexametaphosphate or sodium trimetaphosphate.

For some reactive diluents, prolonged or repeated skin contact may result in absorption of potentially harmful amounts or allergic skin reactions. Exposure to some reactive diluents (notably neopentylglycol diglycidyl ether, CAS RN:17557-23-2) has caused cancer in some animal testing.

There are reports of lung damage due to excessive inhalation of alumina dust. Ingestion of large amounts of aluminium hydroxide for prolonged periods may cause phosphate depletion, especially if phosphate intake is low. This may cause loss of appetite, muscle weakness, muscular disease and even softening of the bones. These effects have not been reported in people occupationally exposed to aluminium hydroxide.

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

Bisphenol F was orally administered at doses 0, 20, 100 and 500 mg/kg per day for at least 28 days, but no clear endocrine-mediated changes were detected, and it was concluded to have no endocrine-mediated effects in young adult rats. On the other hand, the main effect of bisphenol F was concluded to be liver toxicity based on clinical biochemical parameters and liver weight, but without histopathological changes. The no-observed-effect level for bisphenol F is concluded to be under 20 mg/kg per day since decreased body weight accompanied by decreased serum total cholesterol, glucose, and albumin values were observed in the female rats given 20 mg/kg per day or higher doses of bisphenol F. Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. The presence of the p-hydroxy group on the benzene rings is 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 potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes.

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

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

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

BPA belongs to the list of compounds having this property as the rodent models have shown that BPA exposure is linked with increased body 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.

8349TFM-A Thermal Adhesive	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
aluminium hydroxide	<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>
bisphenol F diglycidyl ether copolymer	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: 4000 mg/kg <sup>[2]</sup>	Eyes * (-) (-) Slight irritant

## 8349TFM-A Thermal Adhesive

	Oral (Rat) LD50; 4000 mg/kg <sup>[2]</sup>	Skin * (-) (-) Slight irritant
aluminium oxide	<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>	
neopentyl glycol diglycidyl ether	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 2150 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50; 4500 mg/kg <sup>[2]</sup>	Skin (human): Sensitiser [Shell] Skin: adverse effect observed (irritating) <sup>[1]</sup>
monomethyl phosphate ethoxylated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
carbon black	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >3000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50; >8000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>Legend:</b>	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

8349TFM-A Thermal Adhesive	<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). 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. 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</p>
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## 8349TFM-A Thermal Adhesive

rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate. This study was used by JECFA as key study to derive the PTWL.

## 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. The various members of the bisphenol family produce hormone-like effects, seemingly as a result of binding to estrogen receptor-related receptors (ERRs; not to be confused with estrogen receptors).

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

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

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

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

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

- ERR-beta is a nuclear receptor. Its function is unknown; however, a similar protein in mouse plays an essential role in placental development.

- ERR-gamma is a nuclear receptor that behaves as a constitutive activator of transcription. There is evidence that bisphenol A functions as an endocrine disruptor by binding strongly to ERRgamma. BPA as well as its nitrated and chlorinated metabolites seems to bind 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.

**NEOPENTYL GLYCOL  
DIGLYCIDYL ETHER**

## \* Anchor SDS]

for 1,2-butylene oxide (ethyloxirane):

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

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	<p>(propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic</p> <p>for alkyl alcohol alkoxyphosphate (AAPD) surfactants (alkyl or alcohol ether phosphates):</p> <p><b>Acute toxicity:</b> This group of surfactants exhibits similar effects to the alcohol ether sulfates (AAASDs) (typically sodium lauryl ether sulfate - SLES - CAS RN 68891-38-3).</p> <p>They are likely to be skin/ eye irritants (R36/38) in their undiluted forms but not acutely toxic. The reported oral LD50 values were higher than 1600 mg/kg for the alkyl ether phosphates family described by CAS RN: 9046-01-9. No effects were found at any concentration tested dermally. Commercial products may contain excess phosphoric acid and may produce serious eye irritation (R41) or may even be classified as corrosive, acidic substances.</p> <p><b>Subchronic toxicity:</b> Data for sulfate derivatives has been identified in the public domain. Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2(CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction. SLES was evaluated for effects on the reproduction and prenatal/postnatal development of the rat when administered orally via the drinking water through two successive generations. Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives</p> <p><b>Genotoxicity:</b> Alcohol ether phosphates are unlikely to be genotoxic by analogy with their alcohol ether sulfate equivalents.</p> <p><b>Carcinogenicity:</b> Chronic dietary studies conducted with rats on sulfate derivatives showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day.)</p> <p><b>Reproductive and developmental toxicity:</b> Studies with sulfate derivatives showed little to no toxicity in dams or pups with the NOEL in a developmental toxicity study in rats with SLES at the limit dose of 1000 mg/kg/day and a reproductive NOAEL of 0.3% in drinking water (equivalent to 300 mg/kg/day), the highest dose tested in a two-generation reproduction study.</p> <p>In studies with phosphate derivatives, the reproductive/ developmental NOAEL for an OECD 422 study with CAS 681340-47-2 was 800 mg/kg/day, the highest dose tested, and for CAS RN 78330-24-2 the NOEL was 200 mg/kg/day.</p> <p>An NOAEL of 200 mg/kg/day was selected as the toxicological endpoint for the chronic risk assessment for phosphate derivatives by the US EPA. Both alcohol ether sulfates and phosphates have been evaluated in acute, subchronic, developmental and reproductive studies capable of detecting effects on endocrine mediated events. The results of these studies did not give any indication of a treatment-related effect on the oestrogen receptor or endocrine system.</p> <p><b>Metabolic fate:</b> For compounds of comparable C16 carbon chain, the metabolites of the lower molecular weight ethoxylated (POE n=3) alcohol ether sulfate surfactants are readily absorbed and excreted primarily in the urine whereas the C16 surfactants with increased ethoxylation (POE n=9) are poorly absorbed and excreted primarily in the faeces. There was also no evidence of hydrolysis of the sulfate group from C16 POE n=3 and C16 POE n=9 or of metabolism of the ethoxylate portion of the molecule. With C11 POE n=3 and C12 POE n=3 metabolic studies in rats confirmed that the alkyl chain is extensively metabolised by beta- or omega oxidation leaving the ethoxysulfate, which is excreted directly.</p> <p>By analogy alcohol ether phosphate esters may initially undergo metabolism to generate the corresponding alkyl alcohol alkoxyphosphate and POE (or POE/POP - polyoxypropylene) phosphate glycol; the dephosphorylated metabolite should be hydrolysed to the POE (or POE/POP) polyalkoxyglycols and linear branched saturated and unsaturated alkyl alcohol metabolites. The resultant alkyl alcohol metabolites would be oxidised in fatty acid oxidation pathways. The polyalkoxyglycols may either be conjugated and excreted unchanged or hydrolysed/ oxidised to various degraded metabolites before being conjugated and excreted</p> <p><b>Sensitising potential:</b> 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 autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.</p> <p>Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. 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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. 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>
CARBON BLACK	<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>



## 8349TFM-A Thermal Adhesive

<b>8349TFM-A Thermal Adhesive &amp; BISPHENOL F DIGLYCIDYL ETHER COPOLYMER &amp; NEOPENTYL GLYCOL DIGLYCIDYL ETHER</b>	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative.
<b>8349TFM-A Thermal Adhesive &amp; BISPHENOL F DIGLYCIDYL ETHER COPOLYMER</b>	The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics. Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities. Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor. In vitro cell models were used to evaluate the ability of 22 bisphenols (BPs) to induce or inhibit estrogenic and androgenic activity. BPA, Bisphenol AF (BPAF), bisphenol Z (BPZ), bisphenol C (BPC), tetramethyl bisphenol A (TMBPA), bisphenol S (BPS), bisphenol E (BPE), 4,4-bisphenol F (4,4-BPF), bisphenol AP (BPAP), bisphenol B (BPB), tetrachlorobisphenol A (TCBPA), and benzylparaben (PHBB) induced estrogen receptor (ER)alpha and/or ERbeta-mediated activity. With the exception of BPS, TCBPA, and PHBB, these same BPs were also androgen receptor (AR) antagonists. Only 3 BPs were found to be ER antagonists. Bisphenol P (BPP) selectively inhibited ERbeta-mediated activity and 4-(4-phenylmethoxyphenyl)sulfonylphenol (BPS-MPE) and 2,4-bisphenol S (2,4-BPS) selectively inhibited ERalpha-mediated activity. None of the BPs induced AR-mediated activity.
<b>ALUMINIUM HYDROXIDE &amp; ALUMINIUM OXIDE &amp; CARBON BLACK</b>	No significant acute toxicological data identified in literature search.

<b>Acute Toxicity</b>	✗	<b>Carcinogenicity</b>	✗
<b>Skin Irritation/Corrosion</b>	✓	<b>Reproductivity</b>	✗
<b>Serious Eye Damage/Irritation</b>	✓	<b>STOT - Single Exposure</b>	✗
<b>Respiratory or Skin sensitisation</b>	✓	<b>STOT - Repeated Exposure</b>	✗
<b>Mutagenicity</b>	✗	<b>Aspiration Hazard</b>	✗

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

## 11.2.1. Endocrine Disruption Properties

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

## SECTION 12 Ecological information

## 12.1. Toxicity

<b>8349TFM-A Thermal Adhesive</b>	<table border="1"> <thead> <tr> <th>Endpoint</th> <th>Test Duration (hr)</th> <th>Species</th> <th>Value</th> <th>Source</th> </tr> </thead> <tbody> <tr> <td>Not Available</td> <td>Not Available</td> <td>Not Available</td> <td>Not Available</td> <td>Not Available</td> </tr> </tbody> </table>	Endpoint	Test Duration (hr)	Species	Value	Source	Not Available	Not Available	Not Available	Not Available	Not Available																				
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ammonium polyphosphate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	3.57mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>97.1mg/l	2
	EC50	48h	Crustacea	>100mg/l	2

neopentyl glycol diglycidyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

monomethyl phosphate ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

carbon black	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	24h	Crustacea	3200mg/l	1
	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

**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 high molecular weight synthetic polymers: (according to the Sustainable Futures (SF) program (U.S. EPA 2005b; U.S. EPA 2012c) polymer assessment guidance.)

High MW polymers are expected:

- to have low vapour pressure and are not expected to undergo volatilization .
- to adsorb strongly to soil and sediment
- to be non-biodegradable (not anticipated to be assimilated by microorganisms.- therefore, biodegradation is not expected to be an important removal process. However many exceptions exist

High MW polymers are not expected to undergo removal by other degradative processes under environmental conditions

For bisphenol A and related bisphenols:

Environmental fate:

Biodegradability (28 d) 89% - Easily biodegradable

Bioconcentration factor (BCF) 7.8 mg/l

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

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

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

Ecotoxicity:

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

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

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

Freshwater algae (96 h): 2.73 mg/l

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

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

In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms.

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

A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations.

A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.

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

Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the 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.

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

They would not be expected to persist in the environment.

Most reactive diluents should be considered slightly to moderately toxic to aquatic organisms on an acute basis while some might also be considered harmful to the environment.

Environmental toxicity is a function of the n-octanol/water partition coefficient (log Pow, log Kow). Compounds with log Pow >5 act as neutral organics, but at a lower log Pow, the toxicity of epoxide-containing polymers is greater than that predicted for simple narcotics.

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

for 1,2-butylene oxide (ethyloxirane):

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

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

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

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

**Ecotoxicity:**

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

\* Persistence and Bioaccumulation Regulations (Canada 2000).

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

Environmental toxicity is a function of the n-octanol/ water partition coefficient (log Pow, log Kow). Phenols with log Pow >7.4 are expected to exhibit low toxicity to aquatic organisms.

However the toxicity of phenols with a lower log Pow is variable, ranging from low toxicity (LC50 values >100 mg/l) to highly toxic (LC50 values <1 mg/l) dependent on log Pow, molecular weight and substitutions on the aromatic ring. Dinitrophenols are more toxic than predicted from QSAR estimates. Hazard information for these groups is not generally available.

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,  $[Al(H_2O)_6]^{3+}$ , undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g.,  $[Al(H_2O)_5(OH)]^{2+}$ ,  $[Al(H_2O)_4(OH)_2]^+$ ). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are  $Al(OH)_2^+$  and  $Al(OH)_2^+$ , while the solid  $Al(OH)_3$  is most prevalent between pH 5.2 and 8.8. The soluble species  $Al(OH)_4^-$  is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous  $Al(OH)_3$ , which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.

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

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

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

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

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

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

The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.

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

Bioconcentration of aluminum has also been reported for aquatic insects.

**Ecotoxicity:**

**Freshwater species pH >6.5**

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

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

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

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

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

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

Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 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.

## 8349TFM-A Thermal Adhesive

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 µg/l (UK max.)

200 µg/l (WHO guideline)

chloride: 400 mg/l (UK max.)

250 mg/l (WHO guideline)

fluoride: 1.5 mg/l (UK max.)

1.5 mg/l (WHO guideline)

nitrate: 50 mg/l (UK max.)

50 mg/l (WHO guideline)

sulfate: 250 mg/l (UK max.)

Soil Guideline: none available.

Air Quality Standards: none available.

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

## 12.2. Persistence and degradability

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

## 12.3. Bioaccumulative potential

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

## 12.4. Mobility in soil

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

## 12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗
PBT Criteria fulfilled?	No		
vPvB	No		

## 12.6. Endocrine Disruption Properties

The evidence linking adverse effects to endocrine disruptors is more compelling in the environment than it is in humans. Endocrine 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

Not Available

## SECTION 13 Disposal considerations

### 13.1. Waste treatment methods

<b>Product / Packaging disposal</b>	<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>
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## 8349TFM-A Thermal Adhesive

	<ul style="list-style-type: none"> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Authority for disposal.</li> <li>▶ Bury or incinerate residue at an approved site.</li> <li>▶ Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>
<b>Waste treatment options</b>	Not Available
<b>Sewage disposal options</b>	Not Available

**SECTION 14 Transport information****Labels Required**

	<p>NOT REGULATED by Ground ADR Special Provision 375  NOT REGULATED by Air IATA Special Provision A197  NOT REGULATED by Sea IMDG per 2.10.2.7  NOT REGULATED by ADN Special Provision 274 (The provision of 3.1.2.8 apply)</p>
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**Land transport (ADR-RID)**

14.1. UN number	3082												
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol F diglycidyl ether copolymer)												
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>Class</td> <td>9</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	9	Subrisk	Not Applicable								
Class	9												
Subrisk	Not Applicable												
14.4. Packing group	III												
14.5. Environmental hazard	Environmentally hazardous												
14.6. Special precautions for user	<table border="1"> <tr> <td>Hazard identification (Kemler)</td> <td>90</td> </tr> <tr> <td>Classification code</td> <td>M6</td> </tr> <tr> <td>Hazard Label</td> <td>9</td> </tr> <tr> <td>Special provisions</td> <td>274 335 375 601</td> </tr> <tr> <td>Limited quantity</td> <td>5 L</td> </tr> <tr> <td>Tunnel Restriction Code</td> <td>3 (-)</td> </tr> </table>	Hazard identification (Kemler)	90	Classification code	M6	Hazard Label	9	Special provisions	274 335 375 601	Limited quantity	5 L	Tunnel Restriction Code	3 (-)
Hazard identification (Kemler)	90												
Classification code	M6												
Hazard Label	9												
Special provisions	274 335 375 601												
Limited quantity	5 L												
Tunnel Restriction Code	3 (-)												

**Air transport (ICAO-IATA / DGR)**

14.1. UN number	3082														
14.2. UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains bisphenol F diglycidyl ether copolymer)														
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>ICAO/IATA Class</td> <td>9</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>9L</td> </tr> </table>	ICAO/IATA Class	9	ICAO / IATA Subrisk	Not Applicable	ERG Code	9L								
ICAO/IATA Class	9														
ICAO / IATA Subrisk	Not Applicable														
ERG Code	9L														
14.4. Packing group	III														
14.5. Environmental hazard	Environmentally hazardous														
14.6. Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>A97 A158 A197 A215</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>964</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>450 L</td> </tr> <tr> <td>Passenger and Cargo Packing Instructions</td> <td>964</td> </tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td> <td>450 L</td> </tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td> <td>Y964</td> </tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td> <td>30 kg G</td> </tr> </table>	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
Special provisions	A97 A158 A197 A215														
Cargo Only Packing Instructions	964														
Cargo Only Maximum Qty / Pack	450 L														
Passenger and Cargo Packing Instructions	964														
Passenger and Cargo Maximum Qty / Pack	450 L														
Passenger and Cargo Limited Quantity Packing Instructions	Y964														
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G														

**Sea transport (IMDG-Code / GGVSee)**

14.1. UN number	3082				
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol F diglycidyl ether copolymer)				
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>IMDG Class</td> <td>9</td> </tr> <tr> <td>IMDG Subrisk</td> <td>Not Applicable</td> </tr> </table>	IMDG Class	9	IMDG Subrisk	Not Applicable
IMDG Class	9				
IMDG Subrisk	Not Applicable				
14.4. Packing group	III				
14.5. Environmental hazard	Marine Pollutant				

## 8349TFM-A Thermal Adhesive

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 F diglycidyl ether copolymer)	
14.3. Transport hazard class(es)	9   Not Applicable	
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Classification code	M6
	Special provisions	274; 335; 375; 601
	Limited quantity	5 L
	Equipment required	PP
	Fire cones number	0

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

Not Applicable

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

Product name	Group
aluminium hydroxide	Not Available
bisphenol F diglycidyl ether copolymer	Not Available
aluminium oxide	Not Available
ammonium polyphosphate	Not Available
neopentyl glycol diglycidyl ether	Not Available
monomethyl phosphate ethoxylated	Not Available
carbon black	Not Available

## 14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
aluminium hydroxide	Not Available
bisphenol F diglycidyl ether copolymer	Not Available
aluminium oxide	Not Available
ammonium polyphosphate	Not Available
neopentyl glycol diglycidyl ether	Not Available
monomethyl phosphate ethoxylated	Not Available
carbon black	Not Available

## SECTION 15 Regulatory information

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

## aluminium hydroxide is found on the following regulatory lists

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

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

## bisphenol F diglycidyl ether copolymer is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

## aluminium oxide is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List  
Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)  
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

## ammonium polyphosphate is found on the following regulatory lists

Europe EC Inventory

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

## neopentyl glycol diglycidyl ether is found on the following regulatory lists

Continued...

## 8349TFM-A Thermal Adhesive

Chemical Footprint Project - Chemicals of High Concern List  
Europe EC Inventory

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

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

#### monomethyl phosphate ethoxylated is found on the following regulatory lists

Not Applicable

#### carbon black is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

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

Europe EC Inventory

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

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)

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

## 15.2. Chemical safety assessment

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

### National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (monomethyl phosphate ethoxylated)
Canada - DSL	Yes
Canada - NDSL	No (aluminium hydroxide; bisphenol F diglycidyl ether copolymer; aluminium oxide; ammonium polyphosphate; neopentyl glycol diglycidyl ether; monomethyl phosphate ethoxylated; carbon black)
China - IECSC	No (monomethyl phosphate ethoxylated)
Europe - EINEC / ELINCS / NLP	No (bisphenol F diglycidyl ether copolymer; monomethyl phosphate ethoxylated)
Japan - ENCS	No (ammonium polyphosphate; monomethyl phosphate ethoxylated)
Korea - KECI	No (monomethyl phosphate ethoxylated)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (monomethyl phosphate ethoxylated)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (bisphenol F diglycidyl ether copolymer; ammonium polyphosphate; neopentyl glycol diglycidyl ether; monomethyl phosphate ethoxylated)
Vietnam - NCI	Yes
Russia - FBEPH	No (neopentyl glycol diglycidyl ether; monomethyl phosphate ethoxylated)
<b>Legend:</b>	<i>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.</i>

## SECTION 16 Other information

<b>Revision Date</b>	07/03/2022
<b>Initial Date</b>	08/03/2022

### Full text Risk and Hazard codes

<b>H318</b>	Causes serious eye damage.
<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

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

Continued...

**8349TFM-A Thermal Adhesive**

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  
AIIIC: Australian Inventory of Industrial Chemicals  
DSL: Domestic Substances List  
NDSL: Non-Domestic Substances List  
IECSC: Inventory of Existing Chemical Substance in China  
EINECS: European INventory of Existing Commercial chemical Substances  
ELINCS: European List of Notified Chemical Substances  
NLP: No-Longer Polymers  
ENCS: Existing and New Chemical Substances Inventory  
KECI: Korea Existing Chemicals Inventory  
NZIoC: New Zealand Inventory of Chemicals  
PICCS: Philippine Inventory of Chemicals and Chemical Substances  
TSCA: Toxic Substances Control Act  
TCSI: Taiwan Chemical Substance Inventory  
INSQ: Inventario Nacional de Sustancias Químicas  
NCI: National Chemical Inventory  
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

**Reason For Change**

A-2.00 - Modification to the safety data sheet





## 8349TFM-B Thermal Adhesive

### MG Chemicals UK Limited

Version No: A-2.00

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

Issue Date: 07/03/2022

Revision Date: 07/03/2022

L.REACH.GB.EN

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

### 1.1. Product Identifier

Product name	8349TFM-B
Synonyms	SDS Code: 8349TFM-Part B; 8349TFM-25ML, 8349TFM-50ML   UFI:3GQ0-G0G5-G00R-QK4A
Other means of identification	Thermal Adhesive

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

Relevant identified uses	Thermally conductive adhesive hardener
Uses advised against	Not Applicable

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

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

### 1.4. Emergency telephone number

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

## SECTION 2 Hazards identification

### 2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H318 - Serious Eye Damage/Eye Irritation Category 1, H315 - Skin Corrosion/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	Danger

### Hazard statement(s)

H318	Causes serious eye damage.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.

### Supplementary statement(s)

Not Applicable

### Precautionary statement(s) Prevention

## 8349TFM-B Thermal Adhesive

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

## Precautionary statement(s) Response

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

## 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

Ingestion may produce health damage\*.

Cumulative effects may result following exposure\*.

Eye contact may produce serious damage\*.

Possible respiratory sensitizer\*.

## 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
1.21645-51-2 2.244-492-7 3.Not Available 4.Not Available	53	<u>aluminium hydroxide</u>	Serious Eye Damage/Eye Irritation Category 2; H319 [1]	Not Available	Not Available
1.1344-28-1. 2.215-691-6 3.Not Available 4.Not Available	15	<u>aluminium oxide</u>	Not Applicable	Not Available	Not Available
1.100-51-6 2.202-859-9 3.603-057-00-5 4.Not Available	3	<u>benzyl alcohol</u>	Acute Toxicity (Oral) Category 4, Acute Toxicity (Inhalation) Category 4; H302, H332 [2]	Not Available	Not Available
1.135108-88-2 2.Not Available 3.Not Available 4.Not Available	3	<u>formaldehyde/benzenamine, hydrogenated</u>	Corrosive to Metals Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1B, Serious Eye Damage/Eye Irritation Category 1; H290, H302, H314, H318 [1]	Not Available	Not Available
1.109-55-7 2.203-680-9 3.612-061-00-6 4.Not Available	2	<u>3-dimethylaminopropylamine</u>	Flammable Liquids Category 3, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1; H226, H302, H314, H317 [2]	Not Available	Not Available
1.70700-21-9 2.Not Available 3.Not Available 4.Not Available	1	<u>monomethyl phosphate ethoxylated</u>	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H315, H318, H413 [1]	Not Available	Not Available
1.1761-71-3 2.217-168-8 3.Not Available 4.Not Available	0.2	<u>4,4'-methylenbis(cyclohexylamine)</u>	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1A, Serious Eye Damage/Eye Irritation Category 1, Sensitisation (Skin) Category 1, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H302, H314, H318, H317, H373, H411 [1]	Not Available	Not Available
1.108-95-2 2.203-632-7 3.604-001-00-2	0.2	<u>phenol</u> *	Acute Toxicity (Oral) Category 3, Acute Toxicity (Dermal) Category 3, Acute Toxicity (Inhalation) Category 3, Skin	* Skin Corr. 1B; H314: C ≥ 3 %   Skin Irrit. 2;	Not Available

Continued...

## 8349TFM-B Thermal Adhesive

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
4.Not Available			Corrosion/Irritation Category 1B, Germ Cell Mutagenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2; H301, H311, H331, H314, H341, H373 [2]	H315: 1 % ≤ C < 3 %   Eye Irrit. 2; H319: 1 % ≤ C < 3 %	
<p><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&amp;L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties</p>					

## 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>▶ Immediately hold eyelids apart and flush the eye continuously with 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>▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area.</li> <li>▶ Other measures are usually unnecessary.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> </ul>

## 4.2 Most important symptoms and effects, both acute and delayed

See Section 11

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

Treat symptomatically.

## SECTION 5 Firefighting measures

## 5.1. Extinguishing media

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

## 5.2. Special hazards arising from the substrate or mixture

<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>) metal oxides other pyrolysis products typical of burning organic material.</p>

Continued...

## 8349TFM-B Thermal Adhesive

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

## SECTION 6 Accidental release measures

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

See section 8

## 6.2. Environmental precautions

See section 12

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

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

## 6.4. Reference to other sections

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

## SECTION 7 Handling and storage

## 7.1. Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ When handling, <b>DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> </ul>
<b>Fire and explosion protection</b>	See section 5
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ No smoking, naked lights or ignition sources.</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

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Metal can or drum</li> <li>▶ Packaging as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
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## 8349TFM-B Thermal Adhesive

<b>Storage incompatibility</b>	<p>For aluminas (aluminium oxide):            Incompatible with hot chlorinated rubber.            In the presence of chlorine trifluoride may react violently and ignite.            -May initiate explosive polymerisation of olefin oxides including ethylene oxide.            -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.            -Produces exothermic reaction with oxygen difluoride.            -May form explosive mixture with oxygen difluoride.            -Forms explosive mixtures with sodium nitrate.            -Reacts vigorously with vinyl acetate.</p> <p>Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.</p> <p>‣ Avoid reaction with oxidising agents</p>
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**7.3. Specific end use(s)**

See section 1.2

**SECTION 8 Exposure controls / personal protection****8.1. Control parameters**

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
aluminium hydroxide	Inhalation 10.76 mg/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
aluminium oxide	Dermal 0.84 mg/kg bw/day (Systemic, Chronic) Inhalation 3 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 3 mg/m <sup>3</sup> (Local, Chronic) Dermal 0.3 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.75 mg/m <sup>3</sup> (Systemic, Chronic) * Oral 1.32 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.75 mg/m <sup>3</sup> (Local, Chronic) *	74.9 µg/L (Water (Fresh)) 20 mg/L (STP)
benzyl alcohol	Dermal 8 mg/kg bw/day (Systemic, Chronic) Inhalation 22 mg/m <sup>3</sup> (Systemic, Chronic) Dermal 40 mg/kg bw/day (Systemic, Acute) Inhalation 110 mg/m <sup>3</sup> (Systemic, Acute) Dermal 4 mg/kg bw/day (Systemic, Chronic) * Inhalation 5.4 mg/m <sup>3</sup> (Systemic, Chronic) * Oral 4 mg/kg bw/day (Systemic, Chronic) * Dermal 20 mg/kg bw/day (Systemic, Acute) * Inhalation 27 mg/m <sup>3</sup> (Systemic, Acute) * Oral 20 mg/kg bw/day (Systemic, Acute) *	1 mg/L (Water (Fresh)) 0.1 mg/L (Water - Intermittent release) 2.3 mg/L (Water (Marine)) 5.27 mg/kg sediment dw (Sediment (Fresh Water)) 0.527 mg/kg sediment dw (Sediment (Marine)) 0.456 mg/kg soil dw (Soil) 39 mg/L (STP)
formaldehyde/ benzenamine, hydrogenated	Dermal 2 mg/kg bw/day (Systemic, Chronic) Inhalation 0.2 mg/m <sup>3</sup> (Systemic, Chronic) Dermal 6 mg/kg bw/day (Systemic, Acute) Inhalation 2 mg/m <sup>3</sup> (Systemic, Acute)	0.015 mg/L (Water (Fresh)) 0.002 mg/L (Water - Intermittent release) 0.15 mg/L (Water (Marine)) 15 mg/kg sediment dw (Sediment (Fresh Water)) 1.5 mg/kg sediment dw (Sediment (Marine)) 1.8 mg/kg soil dw (Soil) 1.9 mg/L (STP)
3-dimethylaminopropylamine	Inhalation 1.2 mg/m <sup>3</sup> (Systemic, Chronic)	0.073 mg/L (Water (Fresh)) 0.007 mg/L (Water - Intermittent release) 0.34 mg/L (Water (Marine)) 0.735 mg/kg sediment dw (Sediment (Fresh Water)) 0.073 mg/kg sediment dw (Sediment (Marine)) 0.104 mg/kg soil dw (Soil) 10 mg/L (STP)
4,4'-methylenebis(cyclohexylamine)	Dermal 0.1 mg/kg bw/day (Systemic, Chronic) Inhalation 0.9 mg/m <sup>3</sup> (Systemic, Chronic) Dermal 0.06 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.21 mg/m <sup>3</sup> (Systemic, Chronic) * Oral 0.06 mg/kg bw/day (Systemic, Chronic) *	0.08 mg/L (Water (Fresh)) 0.008 mg/L (Water - Intermittent release) 0.08 mg/L (Water (Marine)) 14.6 mg/kg sediment dw (Sediment (Fresh Water)) 1.46 mg/kg sediment dw (Sediment (Marine)) 4.56 mg/kg soil dw (Soil) 3.2 mg/L (STP) 0.556 mg/kg food (Oral)
phenol	Dermal 1.23 mg/kg bw/day (Systemic, Chronic) Inhalation 8 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 16 mg/m <sup>3</sup> (Local, Acute) Dermal 0.4 mg/kg bw/day (Systemic, Chronic) * Inhalation 1.32 mg/m <sup>3</sup> (Systemic, Chronic) * Oral 0.4 mg/kg bw/day (Systemic, Chronic) *	0.008 mg/L (Water (Fresh)) 0.001 mg/L (Water - Intermittent release) 0.031 mg/L (Water (Marine)) 0.091 mg/kg sediment dw (Sediment (Fresh Water)) 0.009 mg/kg sediment dw (Sediment (Marine)) 0.136 mg/kg soil dw (Soil) 2.1 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)	aluminium oxide	Aluminium oxides: respirable dust	4 mg/m <sup>3</sup>	Not Available	Not Available	Not Available

Continued...

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: inhalable dust	10 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	phenol	Phenol	2 ppm / 8 mg/m <sup>3</sup>	16 mg/m <sup>3</sup> / 4 ppm	Not Available	skin
UK Workplace Exposure Limits (WELs)	phenol	Phenol	2 ppm / 7.8 mg/m <sup>3</sup>	16 mg/m <sup>3</sup> / 4 ppm	Not Available	Sk

## Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
aluminium hydroxide	8.7 mg/m <sup>3</sup>	73 mg/m <sup>3</sup>	440 mg/m <sup>3</sup>
aluminium oxide	15 mg/m <sup>3</sup>	170 mg/m <sup>3</sup>	990 mg/m <sup>3</sup>
benzyl alcohol	30 ppm	52 ppm	740 ppm
3-dimethylaminopropylamine	1.2 ppm	13 ppm	89 ppm
phenol	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
aluminium hydroxide	Not Available	Not Available
aluminium oxide	Not Available	Not Available
benzyl alcohol	Not Available	Not Available
formaldehyde/ benzenamine, hydrogenated	Not Available	Not Available
3-dimethylaminopropylamine	Not Available	Not Available
monomethyl phosphate ethoxylated	Not Available	Not Available
4,4'-methylenebis(cyclohexylamine)	Not Available	Not Available
phenol	250 ppm	Not Available

## Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
aluminium hydroxide	E	≤ 0.01 mg/m <sup>3</sup>
benzyl alcohol	E	≤ 0.1 ppm
formaldehyde/ benzenamine, hydrogenated	E	≤ 0.1 ppm
3-dimethylaminopropylamine	E	≤ 0.1 ppm
monomethyl phosphate ethoxylated	E	≤ 0.1 ppm
4,4'-methylenebis(cyclohexylamine)	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

Fragrance substance with is an established contact allergen in humans.

Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m<sup>3</sup> over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

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

For aluminium oxide:

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

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

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)

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

Continued...

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- 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

Odour Threshold Value for phenol: 0.060 ppm (detection)

NOTE: Detector tubes for phenol, measuring in excess of 1 ppm, are commercially available.


Systemic absorption by all routes may induce convulsions with damage to the lungs and central nervous system.

Exposure at or below the recommended TLV-TWA is thought to protect the worker from respiratory, cardiovascular, hepatic, renal and neurological toxicity. Workers or volunteers exposed at or below 5.2 ppm phenol have experienced no ill-effects. Because phenol as a vapour, liquid or solid can penetrate the skin causing systemic effects, a skin notation is considered necessary. Although ACGIH has not recommended a STEL it is felt that ACGIH excursion limits (15 ppm limited to a total duration of 30 minutes with brief excursions limited to no more than 25 ppm) and NIOSH Ceiling values are sufficiently similar so as to provide the same margin of safety.

Odour Safety Factor(OSF)

OSF=25 (PHENOL)

## 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. 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>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.</p> <table border="1" data-bbox="389 853 1485 1111"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="389 1167 1091 1335"> <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. Personal protection</b></p>																					
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> </ul>																				
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>																				
<p><b>Hands/feet protection</b></p>	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <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>																				

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	<p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>· frequency and duration of contact,</li> <li>· chemical resistance of glove material,</li> <li>· glove thickness and</li> <li>· dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>· Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>· Excellent when breakthrough time &gt; 480 min</li> <li>· Good when breakthrough time &gt; 20 min</li> <li>· Fair when breakthrough time &lt; 20 min</li> <li>· Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.</li> <li>· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential</li> </ul> <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eye wash unit.</li> </ul>

## Recommended material(s)

## GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

**Forsberg Clothing Performance Index<sup>®</sup>**

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

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

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

## Respiratory protection

Type 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 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

Continued...



## 8349TFM-B Thermal Adhesive

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	Dark		
Physical state	Liquid	Relative density (Water = 1)	1.74
Odour	Slight	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	203
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	>20.5
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	96	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (Not Available%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

## 9.2. Other information

Not Available

## SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	<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

Continued...

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## 10.6. Hazardous decomposition products

See section 5.3

## SECTION 11 Toxicological information

## 11.1. Information on toxicological effects

Inhaled	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.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Acute toxic responses to aluminium are confined to the more soluble forms.
Skin Contact	The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives . Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. 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. 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>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of</p> <ul style="list-style-type: none"> <li>- appropriate animal studies,</li> <li>- other relevant information</li> </ul> <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>

Continued...

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

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

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

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 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]

There are reports of lung damage due to excessive inhalation of alumina dust. Ingestion of large amounts of aluminium hydroxide for prolonged periods may cause phosphate depletion, especially if phosphate intake is low. This may cause loss of appetite, muscle weakness, muscular disease and even softening of the bones. These effects have not been reported in people occupationally exposed to aluminium hydroxide. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

8349TFM-B Thermal Adhesive	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
aluminium hydroxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation(Rat) LC50; >2.3 mg/l4h <sup>[1]</sup> Oral (Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
aluminium oxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation(Rat) LC50; >2.3 mg/l4h <sup>[1]</sup> Oral (Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
benzyl alcohol	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 2000 mg/kg <sup>[2]</sup>	Eye (rabbit): 0.75 mg open SEVERE
	Inhalation(Rat) LC50; >4.178 mg/L4h <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50; 1230 mg/kg <sup>[2]</sup>	Skin (man): 16 mg/48h-mild Skin (rabbit):10 mg/24h open-mild Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
formaldehyde/ benzenamine, hydrogenated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1000 mg/kg <sup>[1]</sup> Oral (Rat) LD50; >50<300 mg/kg <sup>[1]</sup>	Skin: adverse effect observed (corrosive) <sup>[1]</sup>
3-dimethylaminopropylamine	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >400<2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 5 mg - moderate
	Inhalation(Rat) LC50; >4.31 mg/l4h <sup>[2]</sup>	Eye: adverse effect observed (irreversible damage) <sup>[1]</sup>
	Oral (Rat) LD50; 377.1 mg/kg <sup>[1]</sup>	Skin (rabbit): 0.1 mg/24h - open Skin: adverse effect observed (corrosive) <sup>[1]</sup> Skin: adverse effect observed (irritating) <sup>[1]</sup>

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monomethyl phosphate ethoxylated	TOXICITY	IRRITATION
	Not Available	Not Available

4,4'-methylenebis(cyclohexylamine)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1000 mg/kg <sup>[1]</sup>	Eye (rabbit): 10uL/24h SEVERE
	Inhalation(Mouse) LC50; 0.4 mg/4h <sup>[2]</sup>	Eye: adverse effect observed (irreversible damage) <sup>[1]</sup>
	Oral (Rat) LD50; 350 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): SEVERE Corrosive **
	Skin: adverse effect observed (corrosive) <sup>[1]</sup>	

phenol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 850 mg/kg <sup>[2]</sup>	Eye(rabbit): 100 mg rinse - mild
	Inhalation(Mouse) LC50; 0.177 mg/L4h <sup>[2]</sup>	Eye(rabbit): 5 mg - SEVERE
	Oral (Rat) LD50; 317 mg/kg <sup>[2]</sup>	Skin(rabbit): 500 mg open -SEVERE
	Skin(rabbit): 500 mg/24hr - SEVERE	

**Legend:** 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.\* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

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For aluminium compounds:

Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex. Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate).

Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability.

For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe.

Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium.

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

Systemic toxicity after repeated exposure

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

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

The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity.

Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent

Reproductive and developmental toxicity:

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

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

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	<p><b>Genotoxicity</b> Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation <i>in vitro</i>. Clastogenic effects were also observed <i>in vivo</i> 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 <i>in-vitro</i> and <i>in-vivo</i> mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.</p> <p><b>Carcinogenicity.</b> The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons.</p> <p><b>Neurodegenerative diseases.</b> Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. Some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.</p> <p><b>Contact sensitivity:</b> 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.</p> <p>Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported. Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste.</p>
BENZYL ALCOHOL	<p><b>For benzyl alkyl alcohols:</b> Unlike benzylic alcohols, the beta-hydroxyl group of the members of this cluster is unlikely to undergo phase II metabolic activation. Instead, the beta-hydroxyl group is expected to contribute to detoxification via oxidation to hydrophilic acid. Despite structural similarity to carcinogenic ethyl benzene, only a marginal concern has been assigned to phenethyl alcohol due to limited mechanistic analogy.</p> <p><b>For benzoates:</b> <b>Acute toxicity:</b> Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol.</p> <p>The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are &gt; 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds.</p> <p>Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye.</p> <p><b>Sensitisation:</b> The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers.</p> <p><b>Repeat dose toxicity:</b> For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values &gt; 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed. For benzyl alcohol the long-term studies indicate a NOAEL &gt; 400 mg/kg bw/d for rats and &gt; 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur.</p> <p><b>Mutagenicity:</b> All chemicals showed no mutagenic activity in <i>in vitro</i> Ames tests. Various results were obtained with other <i>in vitro</i> genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity <i>in vivo</i>. While some mixed and/or equivocal <i>in vitro</i> chromosomal/chromatid responses have been observed, no genotoxicity was observed in the <i>in vivo</i> cytogenetic, micronucleus, or other assays. The weight of the evidence of the <i>in vitro</i> and <i>in vivo</i> genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.</p> <p>In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL &gt;2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested</p>

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	<p>only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts.</p> <p><b>Developmental toxicity:</b> In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed.</p> <p>A member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption, metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances.</p> <p>All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals. The substances in this group:</p> <ul style="list-style-type: none"> <li>· contain a benzene ring substituted with a reactive primary oxygenated functional group or can be hydrolysed to such a functional group</li> <li>· the major pathway of metabolic detoxification involves hydrolysis and oxidation to yield the corresponding benzoic acid derivative which is excreted either as the free acid or the glycine conjugate</li> <li>· they show a consistent pattern of toxicity in both short- and long- term studies and</li> <li>· they exhibit no evidence of genotoxicity in standardised batteries of in vitro and in vivo assays.</li> </ul> <p>The benzyl derivatives are rapidly absorbed through the gut, metabolised primarily in the liver, and excreted in the urine as glycine conjugates of benzoic acid derivatives.</p> <p>In general, aromatic esters are hydrolysed in vivo through the catalytic activity of carboxylesterases, the most important of which are the A-esterases. Hydrolysis of benzyl and benzoate esters to yield corresponding alcohols and carboxylic acids and hydrolysis of acetals to yield benzaldehyde and simple alcohols have been reported in several experiments.</p> <p>The alcohols and aldehydes are rapidly oxidised to benzoic acid while benzoate esters are hydrolysed to benzoic acid.</p> <p>Flavor and Extract Manufacturers Association (FEMA)</p> <p>The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles.</p> <p>The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity.</p> <p>At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin.</p> <p>The potential for eye irritation is minimal.</p> <p>With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low.</p> <p>NOAELs for maternal and developmental toxicity are far in excess of current human exposure levels.</p> <p>No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity in vitro bacterial assays, and in vitro mammalian cell assays. All in vivo micronucleus assays were negative.</p> <p>It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients</p> <p>The Research Institute for Fragrance Materials (RIFM) Expert Panel</p>
<p><b>FORMALDEHYDE/ BENZENAMINE, HYDROGENATED</b></p>	<p>Amine adducts have much reduced volatility and are less irritating to the skin and eyes than amine hardeners. However commercial amine adducts may contain a percentage of unreacted amine and all unnecessary contact should be avoided.</p> <p>Amine adducts are prepared by reacting excess primary amines with epoxy resin.</p>
<p><b>3-DIMETHYLAMINOPROPYLAMINE</b></p>	<p>for 3-dimethylaminopropylamine (syn 3-aminopropyl dimethylamine, DMPA)</p> <p><b>Acute toxicity:</b> DMPA was been found to be harmful following oral administration to rats.</p> <p>In a field study workers showed impaired respiration (wheezy breath, constricted chest, irritation of mucosa of the eyes, nose and pharynx) as a result of occupational exposure to DMPA (2.34 – 5.87 mg/m<sup>3</sup>= 0.55 – 1.38 ppm).</p> <p>Based on the results of the sensitisation test on the skin DMPA has been classified as having a sensitising effect. DMPA showed strong irritating or corrosive effects.</p> <p><b>Repeat dose toxicity:</b> In a oral 28-day subchronic toxicity study with rats, the no-observed-adverse effect-level (NOAEL) was 50 mg /kg bw/day.</p> <p>In the oral reproduction/developmental toxicity screening test the no-observed-adverse effect-level (NOAEL) was 200 mg/kg bw/day.</p> <p><b>Genotoxicity:</b> DMPA was not mutagenic in the Ames Test and in a mouse micronucleus assay.</p>
<p><b>MONOMETHYL PHOSPHATE ETHOXYLATED</b></p>	<p>for alkyl alcohol alkoxylate phosphate (AAPD) surfactants (alkyl or alcohol ether phosphates):</p> <p><b>Acute toxicity:</b> This group of surfactants exhibits similar effects to the alcohol ether sulfates (AAASDs) (typically sodium lauryl ether sulfate - SLES - CAS RN 68891-38-3).</p> <p>They are likely to be skin/ eye irritants (R36/38) in their undiluted forms but not acutely toxic. The reported oral LD50 values were higher than 1600 mg/kg for the alkyl ether phosphates family described by CAS RN: 9046-01-9. No effects were found at any concentration tested dermally.</p> <p>Commercial products may contain excess phosphoric acid and may produce serious eye irritation (R41) or may even be classified as corrosive, acidic substances.</p> <p><b>Subchronic toxicity:</b> Data for sulfate derivatives has been identified in the public domain. Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity.</p> <p>No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2(CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction.</p> <p>SLES was evaluated for effects on the reproduction and prenatal/postnatal development of the rat when administered orally via the drinking water through two successive generations. Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives</p> <p><b>Genotoxicity:</b> Alcohol ether phosphates are unlikely to be genotoxic by analogy with their alcohol ether sulfate equivalents.</p> <p><b>Carcinogenicity:</b> Chronic dietary studies conducted with rats on sulfate derivatives showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).]</p> <p><b>Reproductive and developmental toxicity:</b> Studies with sulfate derivatives showed little to no toxicity in dams or pups with the NOEL in a developmental toxicity study in rats with SLES at the limit dose of 1000 mg/kg/day and a reproductive NOAEL of 0.3% in drinking water (equivalent to 300 mg/kg/day), the highest dose tested in a two-generation reproduction study.</p> <p>In studies with phosphate derivatives, the reproductive/ developmental NOAEL for an OECD 422 study with CAS 681340-47-2 was 800 mg/kg/day, the highest dose tested, and for CAS RN 78330-24-2 the NOEL was 200 mg/kg/day.</p> <p>An NOAEL of 200 mg/kg/day was selected as the toxicological endpoint for the chronic risk assessment for phosphate derivatives by the US EPA.</p>

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	<p>Both alcohol ether sulfates and phosphates have been evaluated in acute, subchronic, developmental and reproductive studies capable of detecting effects on endocrine mediated events. The results of these studies did not give any indication of a treatment-related effect on the oestrogen receptor or endocrine system.</p> <p><b>Metabolic fate:</b> For compounds of comparable C16 carbon chain, the metabolites of the lower molecular weight ethoxylated (POE n=3) alcohol ether sulfate surfactants are readily absorbed and excreted primarily in the urine whereas the C16 surfactants with increased ethoxylation (POE n=9) are poorly absorbed and excreted primarily in the faeces. There was also no evidence of hydrolysis of the sulfate group from C16 POE n= 3 and C16 POE n=9 or of metabolism of the ethoxylate portion of the molecule. With C11 POE n=3 and C12 POE n=3 metabolic studies in rats confirmed that the alkyl chain is extensively metabolised by beta- or omega oxidation leaving the ethoxysulfate, which is excreted directly.</p> <p>By analogy alcohol ether phosphate esters may initially undergo metabolism to generate the corresponding alkyl alcohol alkoxylate and POE (or POE/POP - polyoxypropylene) phosphate glycol; the dephosphorylated metabolite should be hydrolysed to the POE (or POE/POP) polyalkoxylate glycols and linear branched saturated and unsaturated alkyl alcohol metabolites. The resultant alkyl alcohol metabolites would be oxidised in fatty acid oxidation pathways. The polyalkoxylate glycols may either be conjugated and excreted unchanged or hydrolysed/ oxidised to various degraded metabolites before being conjugated and excreted.</p> <p><b>Sensitising potential:</b> 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>Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. 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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.</p> <p>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>
4,4'-METHYLENEBIS(CYCLOHEXYLAMINE)	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.</p> <p>Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).</p> <p>The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.</p>
PHENOL	<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 produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p> <p>The substance is classified by IARC as Group 3: <b>NOT</b> classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
8349TFM-B Thermal Adhesive & BENZYL ALCOHOL & 3-DIMETHYLAMINOPROPYLAMINE & 4,4'-METHYLENEBIS(CYCLOHEXYLAMINE)	<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</p>

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8349TFM-B Thermal Adhesive & BENZYL ALCOHOL	<p>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>Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and conubial contact dermatitis occur.</p> <p>Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to 'perfume mix'. The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.</p> <p>Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.</p> <p>Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.</p> <p>Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.</p> <p>Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question.</p> <p>Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease. Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.</p> <p><b>Hands:</b> Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.</p> <p><b>Axillae Bilateral axillary</b> (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.</p> <p><b>Face</b> Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.</p> <p><b>Irritant reactions (including contact urticaria):</b> Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.</p> <p><b>Pigmentary anomalies:</b> The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.</p> <p><b>Photo-reactions</b> Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.</p> <p><b>General/respiratory:</b> Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.</p> <p>Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prohaptens is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohaptens is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prohaptens or as a prohaptens, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.</p> <p><b>Prohaptens</b> Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens. In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Cross-reactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.</p>



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	<p>The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptenes can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity. <b>QSAR prediction:</b> The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptenes) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptenes. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation</p>
<p><b>ALUMINIUM HYDROXIDE &amp; ALUMINIUM OXIDE &amp; FORMALDEHYDE/ BENZENAMINE, HYDROGENATED</b></p>	<p>No significant acute toxicological data identified in literature search.</p>
<p><b>BENZYL ALCOHOL &amp; 4,4'-METHYLENEBIS(CYCLOHEXYLAMINE)</b></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><b>FORMALDEHYDE/ BENZENAMINE, HYDROGENATED &amp; 3-DIMETHYLAMINOPROPYLAMINE &amp; 4,4'-METHYLENEBIS(CYCLOHEXYLAMINE) &amp; PHENOL</b></p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>
<p><b>3-DIMETHYLAMINOPROPYLAMINE &amp; 4,4'-METHYLENEBIS(CYCLOHEXYLAMINE)</b></p>	<p>While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.</p> <ul style="list-style-type: none"> <li>▶ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis.</li> <li>▶ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient.</li> </ul> <p>Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.</p> <p><b>Inhalation:</b> Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies. While most polyurethane amine catalysts are not sensitisers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease. Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.</p> <p><b>Skin Contact:</b> Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis. Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.</p> <p><b>Eye Contact:</b> Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling. The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.</p>

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**Ingestion:**

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

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

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

Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract,

diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.

**Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000**

**Alliance for Polyurethanes Industry**

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

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

**11.2.1. Endocrine Disruption Properties**

Not Available

**SECTION 12 Ecological information****12.1. Toxicity**

8349TFM-B Thermal Adhesive	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

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

aluminium oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
	LC50	96h	Fish	0.078-0.108mg/l	2
	EC50	72h	Algae or other aquatic plants	0.2mg/l	2
	EC50	48h	Crustacea	1.5mg/l	2
EC50	96h	Algae or other aquatic plants	0.024mg/l	2	

benzyl alcohol	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	336h	Fish	5.1mg/l	2
	LC50	96h	Fish	10mg/l	2
	EC50	72h	Algae or other aquatic plants	500mg/l	2
	EC50	48h	Crustacea	230mg/l	2
EC50	96h	Algae or other aquatic plants	76.828mg/l	2	

formaldehyde/ benzenamine, hydrogenated	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	72h	Algae or other aquatic plants	1.2mg/l	2
	LC50	96h	Fish	63mg/l	2
	EC50	72h	Algae or other aquatic plants	43.94mg/l	2
EC50	48h	Crustacea	15.4mg/l	2	

3-dimethylaminopropylamine	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	528h	Crustacea	3.64mg/l	2
	EC50	72h	Algae or other aquatic plants	30mg/l	2
	LC50	96h	Fish	100mg/l	1
	EC50	48h	Crustacea	59.46mg/l	2
EC50	96h	Algae or other aquatic plants	57.5mg/l	1	

Continued...

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monomethyl phosphate ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

4,4'-methylenebis(cyclohexylamine)	Endpoint	Test Duration (hr)	Species	Value	Source
	EC0(ECx)	48h	Crustacea	2.5mg/l	2
	EC50	72h	Algae or other aquatic plants	140-200mg/l	2
	LC50	96h	Fish	68mg/l	2
	EC50	48h	Crustacea	6.84mg/l	2

phenol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	36h	Fish	0.008mg/L	4
	EC50	72h	Algae or other aquatic plants	48.937-57.407mg/L	4
	LC50	96h	Fish	2.809-5.554mg/L	4
	EC50	48h	Crustacea	3.1mg/l	1
EC50	96h	Algae or other aquatic plants	10.6mg/L	4	

**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*

Harmful to aquatic organisms.

Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms.

When released to dry soil most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. Environmental processes may also be important in changing solubilities.

Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects.

A metal ion is considered infinitely persistent because it cannot degrade further.

The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.

The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.

Environmental processes may enhance bioavailability.

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,  $[Al(H_2O)_6]^{3+}$ , undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g.,  $[Al(H_2O)_5(OH)]^{2+}$ ,  $[Al(H_2O)_4(OH)_2]^+$ ). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are  $Al(OH)_2^+$  and  $Al(OH)_3$ , while the solid  $Al(OH)_3$  is most prevalent between pH 5.2 and 8.8. The soluble species  $Al(OH)_4^-$  is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous  $Al(OH)_3$ , which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.

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

## 8349TFM-B Thermal Adhesive

**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 µg/l (UK max.)

200 µg/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
benzyl alcohol	LOW	LOW
3-dimethylaminopropylamine	HIGH	HIGH
4,4'-methylenebis(cyclohexylamine)	HIGH	HIGH
phenol	LOW (Half-life = 10 days)	LOW (Half-life = 0.95 days)

**12.3. Bioaccumulative potential**

Ingredient	Bioaccumulation
benzyl alcohol	LOW (LogKOW = 1.1)
3-dimethylaminopropylamine	LOW (LogKOW = -0.4502)
4,4'-methylenebis(cyclohexylamine)	LOW (LogKOW = 3.2649)
phenol	LOW (BCF = 17.5)

**12.4. Mobility in soil**

Ingredient	Mobility
benzyl alcohol	LOW (KOC = 15.66)
3-dimethylaminopropylamine	LOW (KOC = 73.36)
4,4'-methylenebis(cyclohexylamine)	LOW (KOC = 672.4)
phenol	LOW (KOC = 268)

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

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗
PBT Criteria fulfilled?	No		
vPvB	No		

**12.6. Endocrine Disruption Properties**

Not Available

**12.7. Other adverse effects**

Not Available

**SECTION 13 Disposal considerations****13.1. Waste treatment methods**

Continued...

## 8349TFM-B Thermal Adhesive

<b>Product / Packaging disposal</b>	<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> <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>
<b>Waste treatment options</b>	Not Available
<b>Sewage disposal options</b>	Not Available

## SECTION 14 Transport information

## Land transport (ADR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	Class	Not Applicable
	Subrisk	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Hazard identification (Kemler)	Not Applicable
	Classification code	Not Applicable
	Hazard Label	Not Applicable
	Special provisions	Not Applicable
	Limited quantity	Not Applicable
	Tunnel Restriction Code	Not Applicable

## Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	ICAO/IATA Class	Not Applicable
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	Not Applicable
	Cargo Only Packing Instructions	Not Applicable
	Cargo Only Maximum Qty / Pack	Not Applicable
	Passenger and Cargo Packing Instructions	Not Applicable
	Passenger and Cargo Maximum Qty / Pack	Not Applicable
	Passenger and Cargo Limited Quantity Packing Instructions	Not Applicable
	Passenger and Cargo Limited Maximum Qty / Pack	Not Applicable

## Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Applicable
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## 8349TFM-B Thermal Adhesive

14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	IMDG Class	Not Applicable
	IMDG Subrisk	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	Not Applicable
	Special provisions	Not Applicable
	Limited Quantities	Not Applicable

## Inland waterways transport (ADN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	Not Applicable	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Classification code	Not Applicable
	Special provisions	Not Applicable
	Limited quantity	Not Applicable
	Equipment required	Not Applicable
	Fire cones number	Not Applicable

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

Not Applicable

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

Product name	Group
aluminium hydroxide	Not Available
aluminium oxide	Not Available
benzyl alcohol	Not Available
formaldehyde/ benzenamine, hydrogenated	Not Available
3-dimethylaminopropylamine	Not Available
monomethyl phosphate ethoxylated	Not Available
4,4'-methylenebis(cyclohexylamine)	Not Available
phenol	Not Available

## 14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
aluminium hydroxide	Not Available
aluminium oxide	Not Available
benzyl alcohol	Not Available
formaldehyde/ benzenamine, hydrogenated	Not Available
3-dimethylaminopropylamine	Not Available
monomethyl phosphate ethoxylated	Not Available
4,4'-methylenebis(cyclohexylamine)	Not Available
phenol	Not Available

## SECTION 15 Regulatory information

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

## aluminium hydroxide is found on the following regulatory lists

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

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

## aluminium oxide is found on the following regulatory lists

## 8349TFM-B Thermal Adhesive

Chemical Footprint Project - Chemicals of High Concern List  
Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)  
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**benzyl alcohol is found on the following regulatory lists**

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

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

**formaldehyde/ benzenamine, hydrogenated is found on the following regulatory lists**

Not Applicable

**3-dimethylaminopropylamine is found on the following regulatory lists**

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances  
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles  
Europe EC Inventory

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

**monomethyl phosphate ethoxylated is found on the following regulatory lists**

Not Applicable

**4,4'-methylenebis(cyclohexylamine) is found on the following regulatory lists**

Europe EC Inventory

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

**phenol is found on the following regulatory lists**

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)  
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances  
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles  
Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)  
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI  
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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

**15.2. Chemical safety assessment**

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

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (monomethyl phosphate ethoxylated)
Canada - DSL	Yes
Canada - NDSL	No (aluminium hydroxide; aluminium oxide; benzyl alcohol; formaldehyde/ benzenamine, hydrogenated; 3-dimethylaminopropylamine; monomethyl phosphate ethoxylated; 4,4'-methylenebis(cyclohexylamine); phenol)
China - IECSC	No (monomethyl phosphate ethoxylated)
Europe - EINEC / ELINCS / NLP	No (formaldehyde/ benzenamine, hydrogenated; monomethyl phosphate ethoxylated)
Japan - ENCS	No (formaldehyde/ benzenamine, hydrogenated; monomethyl phosphate ethoxylated)
Korea - KECI	No (monomethyl phosphate ethoxylated)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (monomethyl phosphate ethoxylated)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (formaldehyde/ benzenamine, hydrogenated; monomethyl phosphate ethoxylated; 4,4'-methylenebis(cyclohexylamine))
Vietnam - NCI	Yes
Russia - FBEPH	No (formaldehyde/ benzenamine, hydrogenated; monomethyl phosphate ethoxylated)
<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**

<b>Revision Date</b>	07/03/2022
<b>Initial Date</b>	07/03/2022

**Full text Risk and Hazard codes**

<b>H226</b>	Flammable liquid and vapour.
<b>H290</b>	May be corrosive to metals.

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<b>H301</b>	Toxic if swallowed.
<b>H302</b>	Harmful if swallowed.
<b>H311</b>	Toxic in contact with skin.
<b>H314</b>	Causes severe skin burns and eye damage.
<b>H319</b>	Causes serious eye irritation.
<b>H331</b>	Toxic if inhaled.
<b>H332</b>	Harmful if inhaled.
<b>H341</b>	Suspected of causing genetic defects.
<b>H373</b>	May cause damage to organs through prolonged or repeated exposure.
<b>H411</b>	Toxic to aquatic life with long lasting effects.
<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

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit.

IDLH: Immediately Dangerous to Life or Health Concentrations

ES: Exposure Standard

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

AiIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory

NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act

TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

**Reason For Change**

A-2.00 - Modification to the safety data sheet