# 834FX-A

**MG Chemicals UK Limited**

Chemwatch Hazard Alert Code: **2**

Version No: **2.5**
Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date: **29/06/2017**
Print Date: **18/08/2017**
L.REACH.GBR.EN

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

### 1.1. Product Identifier

| | |
|---|---|
| **Product name** | 834FX-A |
| **Synonyms** | SDS Code: 834FX-Part A, 834FX-450ML, 834FX-1.7L, 834FX-7.4L, 834FX-40L, 834FX-40L |
| **Proper shipping name** | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid and zinc borate hydrate) |
| **Other means of identification** | Not Available |

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

| | |
|---|---|
| **Relevant identified uses** | epoxy resin |
| **Uses advised against** | Not Applicable |

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

| | | |
|---|---|---|
| **Registered company name** | MG Chemicals UK Limited | MG Chemicals (Head office) |
| **Address** | Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom | 9347 - 193 Street Surrey V4N 4E7 British Columbia Canada |
| **Telephone** | +(44) 1663 362888 | +(1) 800-201-8822 |
| **Fax** | Not Available | +(1) 800-708-9888 |
| **Website** | Not Available | www.mgchemicals.com |
| **Email** | sales@mgchemicals.com | Info@mgchemicals.com |

### 1.4. Emergency telephone number

| | | |
|---|---|---|
| **Association / Organisation** | CHEMTREC | Not Available |
| **Emergency telephone numbers** | +(44) 870-8200418 | Not Available |
| **Other emergency telephone numbers** | +(1) 703-527-3887 | Not Available |

## SECTION 2 HAZARDS IDENTIFICATION

### 2.1. Classification of the substance or mixture

| | |
|---|---|
| **Classification according to regulation (EC) No 1272/2008 [CLP] [1]** | H315 - Skin Corrosion/Irritation Category 2, H319 - Eye Irritation Category 2, H302 - Acute Toxicity (Oral) Category 4, H317 - Skin Sensitizer Category 1, H411 - Chronic Aquatic Hazard Category 2, H361 - Reproductive Toxicity Category 2 |
| ***Legend:*** | *1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI* |

### 2.2. Label elements

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

### Hazard statement(s)

| | |
|---|---|
| **H315** | Causes skin irritation. |
| **H319** | Causes serious eye irritation. |
| **H302** | Harmful if swallowed. |
| **H317** | May cause an allergic skin reaction. |

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| | |
|---|---|
| **H411** | Toxic to aquatic life with long lasting effects. |
| **H361** | Suspected of damaging fertility or the unborn child. |

### Supplementary statement(s)

Not Applicable

### Precautionary statement(s) Prevention

| | |
|---|---|
| **P201** | Obtain special instructions before use. |
| **P280** | Wear protective gloves/protective clothing/eye protection/face protection. |
| **P261** | Avoid breathing mist/vapours/spray. |
| **P270** | Do not eat, drink or smoke when using this product. |
| **P273** | Avoid release to the environment. |
| **P272** | Contaminated work clothing should not be allowed out of the workplace. |

### Precautionary statement(s) Response

| | |
|---|---|
| **P308+P313** | IF exposed or concerned: Get medical advice/ attention. |
| **P302+P352** | IF ON SKIN: Wash with plenty of water and soap. |
| **P305+P351+P338** | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| **P333+P313** | If skin irritation or rash occurs: Get medical advice/attention. |
| **P337+P313** | If eye irritation persists: Get medical advice/attention. |
| **P362+P364** | Take off contaminated clothing and wash it before reuse. |
| **P391** | Collect spillage. |
| **P301+P312** | IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell. |
| **P330** | Rinse mouth. |

### Precautionary statement(s) Storage

| | |
|---|---|
| **P405** | Store locked up. |

### Precautionary statement(s) Disposal

| | |
|---|---|
| **P501** | Dispose of contents/container in accordance with local regulations. |

### 2.3. Other hazards

Inhalation may produce health damage*.

Cumulative effects may result following exposure*.

Limited evidence of a carcinogenic effect*.

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

## SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

### 3.1.Substances

See 'Composition on ingredients' in Section 3.2

### 3.2.Mixtures

| 1.CAS No<br>2.EC No<br>3.Index No<br>4.REACH No | %[weight] | Name | Classification according to regulation (EC) No 1272/2008 [CLP] |
|---|---|---|---|
| 1.25085-99-8<br>2.500-033-5<br>3.603-074-00-8<br>4.01-2119456619-26-XXXX | 22 | bisphenol A/ diglycidyl ether resin, liquid | Eye Irritation Category 2, Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2; H319, H315, H317, H411 [3] |
| 1.21645-51-2<br>2.244-492-7<br>3.Not Available<br>4.01-2119529246-39-XXXX | 22 | alumina hydrate | Not Applicable |
| 1.68333-79-9<br>2.269-789-9<br>3.Not Available<br>4.Not Available | 19 | ammonium polyphosphate | Chronic Aquatic Hazard Category 4; H413 [1] |
| 1.1344-28-1.<br>2.215-691-6<br>3.Not Available<br>4.01-2119529248-35-XXXX\|01-2119817795-27-XXXX | 14 | aluminium oxide | Not Applicable |

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| | | | |
|---|---|---|---|
| 1.41638-13-5<br>2.Not Available<br>3.Not Available<br>4.Not Available | 8 | dipropylene glycol diglycidyl ether | Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1; H315, H317, EUH019 [1] |
| 1.68609-97-2<br>2.271-846-8<br>3.603-103-00-4<br>4.01-2119485289-22-XXXX | 7 | (C12-14)alkylglycidyl ether | Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1; H315, H317 [3] |
| 1.138265-88-0<br>2.Not Available<br>3.Not Available<br>4.Not Available | 5 | zinc borate hydrate | Reproductive Toxicity Category 1B, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H360, H410 [1] |
| 1.25068-38-6<br>2.216-823-5<br>3.603-073-00-2\|603-074-00-8<br>4.01-2119456619-26-XXXX | 1 | bisphenol A diglycidyl ether | Eye Irritation Category 2, Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1; H319, H315, H317 [3] |
| 1.68037-01-4<br>2.500-183-1<br>3.Not Available<br>4.01-2119486452-34-XXXX | 0.6 | 1-decene homopolymer, hydrogenated | Chronic Aquatic Hazard Category 4; H413 [1] |
| 1.64741-65-7.<br>2.Not Available<br>3.649-275-00-4<br>4.Not Available | 0.4 | naphtha petroleum, heavy alkylate | Flammable Liquid Category 3, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1; H226, H336, H304 [1] |
| 1.1333-86-4<br>2.215-609-9<br>3.Not Available<br>4.01-2119384822-32-XXXX\|01-2119489801-30-XXXX\|01-2119475601-40-XXXX | 0.4 | carbon black | Carcinogenicity Category 2; H351 [1] |
| ***Legend:*** | *1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI 4. Classification drawn from C&L* | | |

## SECTION 4 FIRST AID MEASURES

### 4.1. Description of first aid measures

| | |
|---|---|
| **Eye Contact** | If this product comes in contact with the eyes:<br>▸ Wash out immediately with fresh running water.<br>▸ 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.<br>▸ Seek medical attention without delay; if pain persists or recurs seek medical attention.<br>▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| **Skin Contact** | If skin contact occurs:<br>▸ Immediately remove all contaminated clothing, including footwear.<br>▸ Flush skin and hair with running water (and soap if available).<br>▸ Seek medical attention in event of irritation. |
| **Inhalation** | ▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area.<br>▸ Other measures are usually unnecessary. |
| **Ingestion** | ▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.**<br>▸ For advice, contact a Poisons Information Centre or a doctor.<br>▸ Urgent hospital treatment is likely to be needed.<br>▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition.<br>▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist.<br>▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS.<br><br>**Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:**<br>▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.<br>**NOTE:** Wear a protective glove when inducing vomiting by mechanical means. |

### 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.

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

**Continued...**

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

[Ellenhorn and Barceloux: Medical Toxicology]

## SECTION 5 FIREFIGHTING MEASURES

### 5.1. Extinguishing media

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

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

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

### 5.3. Advice for firefighters

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

## SECTION 6 ACCIDENTAL RELEASE MEASURES

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

See section 8

### 6.2. Environmental precautions

See section 12

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

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

### 6.4. Reference to other sections

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

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## SECTION 7 HANDLING AND STORAGE

### 7.1. Precautions for safe handling

| | |
|---|---|
| **Safe handling** | ▸ Avoid all personal contact, including inhalation.<br>▸ Wear protective clothing when risk of exposure occurs.<br>▸ Use in a well-ventilated area.<br>▸ Prevent concentration in hollows and sumps.<br>▸ **DO NOT** enter confined spaces until atmosphere has been checked.<br>▸ Avoid smoking, naked lights or ignition sources.<br>▸ Avoid contact with incompatible materials.<br>▸ When handling, **DO NOT** eat, drink or smoke.<br>▸ Keep containers securely sealed when not in use.<br>▸ Avoid physical damage to containers.<br>▸ Always wash hands with soap and water after handling.<br>▸ Work clothes should be laundered separately.<br>▸ Use good occupational work practice.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.<br>▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.<br>▸ **DO NOT** allow clothing wet with material to stay in contact with skin |
| **Fire and explosion protection** | See section 5 |
| **Other information** | ▸ Store in original containers.<br>▸ Keep containers securely sealed.<br>▸ Store in a cool, dry, well-ventilated area.<br>▸ Store away from incompatible materials and foodstuff containers.<br>▸ Protect containers against physical damage and check regularly for leaks.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS. |

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

| | |
|---|---|
| **Suitable container** | ▸ Metal can or drum<br>▸ Packaging as recommended by manufacturer.<br>▸ Check all containers are clearly labelled and free from leaks. |
| **Storage incompatibility** | For aluminas (aluminium oxide):<br>Incompatible with hot chlorinated rubber.<br>In the presence of chlorine trifluoride may react violently and ignite.<br>-May initiate explosive polymerisation of olefin oxides including ethylene oxide.<br>-Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.<br>-Produces exothermic reaction with oxygen difluoride.<br>-May form explosive mixture with oxygen difluoride.<br>-Forms explosive mixtures with sodium nitrate.<br>-Reacts vigorously with vinyl acetate.<br>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.<br>▸ Avoid reaction with amines, mercaptans, strong acids and oxidising agents<br>Glycidyl ethers:<br>▸ 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<br>▸ may polymerise in contact with heat, organic and inorganic free radical producing initiators<br>▸ may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines<br>▸ react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide<br>▸ attack some forms of plastics, coatings, and rubber |

### 7.3. Specific end use(s)

See section 1.2

## SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

### 8.1. Control parameters

**DERIVED NO EFFECT LEVEL (DNEL)**

Not Available

**PREDICTED NO EFFECT LEVEL (PNEC)**

Not Available

**OCCUPATIONAL EXPOSURE LIMITS (OEL)**

**INGREDIENT DATA**

| Source | Ingredient | Material name | TWA | STEL | Peak | Notes |
|---|---|---|---|---|---|---|
| UK Workplace Exposure Limits (WELs) | carbon black | Carbon black | 3.5 mg/m3 | 7 mg/m3 | Not Available | Not Available |

**EMERGENCY LIMITS**

| Ingredient | Material name | TEEL-1 | TEEL-2 | TEEL-3 |
|---|---|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | Epoxy resin includes EPON 1001, 1007, 820, ERL-2795 | 90 mg/m3 | 990 mg/m3 | 5,900 mg/m3 |

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| | | | | |
|---|---|---|---|---|
| alumina hydrate | Aluminum hydroxide | 8.7 mg/m3 | 73 mg/m3 | 440 mg/m3 |
| aluminium oxide | Aluminum oxide; (Alumina) | 5.7 mg/m3 | 15 mg/m3 | 25 mg/m3 |
| bisphenol A diglycidyl ether | Bisphenol A diglycidyl ether | 39 mg/m3 | 430 mg/m3 | 2,600 mg/m3 |
| bisphenol A diglycidyl ether | Epoxy resin includes EPON 1001, 1007, 820, ERL-2795 | 90 mg/m3 | 990 mg/m3 | 5,900 mg/m3 |
| 1-decene homopolymer, hydrogenated | Decene, 1-, homopolymer, hydrogenated | 30 mg/m3 | 330 mg/m3 | 2,000 mg/m3 |
| carbon black | Carbon black | 9 mg/m3 | 99 mg/m3 | 590 mg/m3 |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | Not Available | Not Available |
| alumina hydrate | Not Available | Not Available |
| ammonium polyphosphate | Not Available | Not Available |
| aluminium oxide | Not Available | Not Available |
| dipropylene glycol diglycidyl ether | Not Available | Not Available |
| (C12-14)alkylglycidyl ether | Not Available | Not Available |
| zinc borate hydrate | Not Available | Not Available |
| bisphenol A diglycidyl ether | Not Available | Not Available |
| 1-decene homopolymer, hydrogenated | Not Available | Not Available |
| naphtha petroleum, heavy alkylate | Not Available | Not Available |
| carbon black | N.E. mg/m3 / N.E. ppm | 1,750 mg/m3 |

**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/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA.
The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For 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
NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP
NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

## 8.2. Exposure controls

**8.2.1. Appropriate engineering controls**

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal operating conditions. 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.

| Type of Contaminant: | Air Speed: |
|---|---|
| solvent, vapours, degreasing etc., evaporating from tank (in still air). | 0.25-0.5 m/s (50-100 f/min) |
| aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation) | 0.5-1 m/s (100-200 f/min.) |
| direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 f/min.) |
| grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion). | 2.5-10 m/s (500-2000 f/min.) |

Within each range the appropriate value depends on:

| Lower end of the range | Upper end of the range |
|---|---|
| 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents |
| 2: Contaminants of low toxicity or of nuisance value only. | 2: Contaminants of high toxicity |

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| | |
|---|---|
| | 3: Intermittent, low production. \| 3: High production, heavy use<br>4: Large hood or large air mass in motion \| 4: Small hood-local control only<br><br>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used. |
| **8.2.2. Personal protection** |  |
| **Eye and face protection** | ▸ Safety glasses with side shields.<br>▸ Chemical goggles.<br>▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] |
| **Skin protection** | See Hand protection below |
| **Hands/feet protection** | **NOTE:**<br>▸ 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.<br>▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.<br>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.<br>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.<br>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 moisturizer is recommended.<br>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:<br>· frequency and duration of contact,<br>· chemical resistance of glove material,<br>· glove thickness and<br>· dexterity<br>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).<br>· 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.<br>· 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.<br>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.<br>· Contaminated gloves should be replaced.<br>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.<br>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.<br>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.<br>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:<br>· 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.<br>· 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<br>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.<br>▸ When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butatoluene rubber), boots and aprons.<br>▸ **DO NOT** use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber or polyethylene gloves (which absorb the resin).<br>▸ **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. |
| **Body protection** | See Other protection below |
| **Other protection** | ▸ Overalls.<br>▸ P.V.C. apron.<br>▸ Barrier cream.<br>▸ Skin cleansing cream.<br>▸ Eye wash unit. |
| **Thermal hazards** | Not Available |

### Respiratory protection

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

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

**Continued...**

| | | | |
|---|---|---|---|
| up to 100 | 10000 | - | A-3 |
| 100+ | | - | Airline** |

* - Continuous Flow
** - Continuous-flow or positive pressure demand.
A(All classes) = Organic vapours, B AUS or B1 = Acid gases, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 deg C)

### 8.2.3. Environmental exposure controls

See section 12

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### 9.1. Information on basic physical and chemical properties

| | |
|---|---|
| **Appearance** | Black |

| | | | |
|---|---|---|---|
| **Physical state** | Liquid | **Relative density (Water = 1)** | 1.63 |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | Not Available |
| **pH (as supplied)** | Not Available | **Decomposition temperature** | Not Available |
| **Melting point / freezing point (°C)** | Not Available | **Viscosity (cSt)** | 2800 |
| **Initial boiling point and boiling range (°C)** | >218 | **Molecular weight (g/mol)** | Not Available |
| **Flash point (°C)** | >150 | **Taste** | Not Available |
| **Evaporation rate** | Not Available | **Explosive properties** | Not Available |
| **Flammability** | Not Applicable | **Oxidising properties** | Not Available |
| **Upper Explosive Limit (%)** | Not Available | **Surface Tension (dyn/cm or mN/m)** | Not Available |
| **Lower Explosive Limit (%)** | Not Available | **Volatile Component (%vol)** | Not Available |
| **Vapour pressure (kPa)** | Not Available | **Gas group** | Not Available |
| **Solubility in water (g/L)** | Immiscible | **pH as a solution (1%)** | Not Available |
| **Vapour density (Air = 1)** | Not Available | **VOC g/L** | Not Available |

### 9.2. Other information

Not Available

## SECTION 10 STABILITY AND REACTIVITY

| | |
|---|---|
| **10.1.Reactivity** | See section 7.2 |
| **10.2. Chemical stability** | ▸ Unstable in the presence of incompatible materials.<br>▸ Product is considered stable.<br>▸ Hazardous polymerisation will not occur. |
| **10.3. Possibility of hazardous reactions** | See section 7.2 |
| **10.4. Conditions to avoid** | See section 7.2 |
| **10.5. Incompatible materials** | See section 7.2 |
| **10.6. Hazardous decomposition products** | See section 5.3 |

## SECTION 11 TOXICOLOGICAL INFORMATION

### 11.1. Information on toxicological effects

| | |
|---|---|
| **Inhaled** | 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.<br><br>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. |
| **Ingestion** | Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.<br>Acute toxic responses to aluminium are confined to the more soluble forms.<br>Male rats exposed to a single oral dose of bisphenol A diglycidyl ether (BADGE) at 750, 1000, and 2000 mg/kg/day showed a significantly increase in the number of immature and maturing sperm on the testis. There were no significant differences with respect to sperm head count, sperm motility, and sperm abnormality in the BADGE treatment groups |

**Continued...**

| | |
|---|---|
| | Inorganic polyphosphates are used extensively in domestic and industrial products. Rats fed 10% sodium trimetaphosphate for a month exhibited transient tubular necrosis;<br>those given 10% sodium metaphosphate exhibited growth retardation; 10% sodium hexametaphosphate produced pale and swollen kidneys.<br>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. |
| **Skin Contact** | 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.<br>The material may accentuate any pre-existing dermatitis condition<br>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.<br>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.<br>Open cuts, abraded or irritated skin should not be exposed to this material<br>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** | 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.<br>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. |
| **Chronic** | 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.<br>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.<br>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.<br><br>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.<br>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. 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.<br>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.<br>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.<br>There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.<br>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.<br>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 osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.<br>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.<br>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.<br>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.<br>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 |

**Continued...**

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*]
All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.
Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether.
A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not *n*-butyl glycidyl ether, induced morphological transformation in mammalian cells *in vitro*. *n*-Butyl glycidyl ether induced micronuclei in mice *in vivo* following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations *in vivo* or chromosomal aberrations in animal cells *in vitro*. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in *Drosophila*. The glycidyl ethers were generally mutagenic to bacteria
Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone.. Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review.
A 2009 study on Chinese workers in bisphenol A factories found that workers were four times more likely to report erectile dysfunction, reduced sexual desire and overall dissatisfaction with their sex life than workers with no heightened bisphenol A exposure. Bisphenol A workers were also seven times more likely to have ejaculation difficulties. They were also more likely to report reduced sexual function within one year of beginning employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties.
Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades'
One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials'
One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood.
A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions.
Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells.[whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes.
Bisphenol A is the isopropyl adduct of 4,4'-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called 'cytostatic hormones'. Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown to be oestrogenic in vitro; such sealants may represent an additional source of xenoestrogens in humans and may be the cause of additional concerns in children.
Concerns have been raised about the possible developmental effects on the foetus/embryo or neonate resulting from the leaching of bisphenol A from epoxy linings in metal cans which come in contact with food-stuffs.
Many drugs, including naproxen, salicylic acid, carbamazepine and mefenamic acid can, in vitro, significantly inhibit bisphenol A glucuronidation (detoxification).

**834FX Black Flexible Epoxy, Thermally Conductive–Flame Retardant, Encapsulating and Potting Compound (Part A)**

| TOXICITY | IRRITATION |
|---|---|
| #51allergy#551badge#55bisphender#55badge#55bisphen#551oxintro#551oxirane[2] | Not Available |

**bisphenol A/ diglycidyl ether resin, liquid**

| TOXICITY | IRRITATION |
|---|---|
| dermal (rat) LD50: >1200 mg/kg[2] | Eye (rabbit): 100mg - Mild |
| Oral (rat) LD50: >1000 mg/kg[2] | |

**alumina hydrate**

| TOXICITY | IRRITATION |
|---|---|
| Oral (rat) LD50: >2000 mg/kg[1] | Not Available |

**ammonium polyphosphate**

| TOXICITY | IRRITATION |
|---|---|
| Dermal (rabbit) LD50: >3160 mg/kg*o[2] | Not Available |
| Oral (rat) LD50: 5625 mg/kg*d[2] | |

**Continued...**

| | |
|---|---|
| **aluminium oxide** | |

| TOXICITY | IRRITATION |
|---|---|
| Oral (rat) LD50: >2000 mg/kg[1] | Not Available |

| | |
|---|---|
| **dipropylene glycol diglycidyl ether** | |

| TOXICITY | IRRITATION |
|---|---|
| Dermal (rabbit) LD50: >2000 mg/kg*[2] | Not Available |
| Oral (rat) LD50: >2000 mg/kg*][2] | |

| | |
|---|---|
| **(C12-14)alkylglycidyl ether** | |

| TOXICITY | IRRITATION |
|---|---|
| Oral (rat) LD50: >10000 mg/kgt[2] | Eye (rabbit): mild [Ciba] |
| | Skin (guinea pig): sensitiser |
| | Skin (human): Irritant |
| | Skin (human): non- sensitiser |
| | Skin (rabbit): moderate |
| | Skin : Moderate |

| | |
|---|---|
| **zinc borate hydrate** | |

| TOXICITY | IRRITATION |
|---|---|
| Not Available | Not Available |

| | |
|---|---|
| **bisphenol A diglycidyl ether** | |

| TOXICITY | IRRITATION |
|---|---|
| Dermal (rabbit) LD50: 20000 mg/kgd[2] | Eye (rabbit): 2 mg/24h - SEVERE |
| Oral (rat) LD50: 11000 mg/kgE[2] | Skin (rabbit): 500 mg - mild |

| | |
|---|---|
| **1-decene homopolymer, hydrogenated** | |

| TOXICITY | IRRITATION |
|---|---|
| dermal (rat) LD50: >2000 mg/kg[1] | Eye*(rabbit):0-4/110.0-nonirritant |
| Inhalation (rat) LC50: 0.9 mg/l/4hr[1] | Skin**(rabbit)-0.5/8.0-nonirritant |
| Inhalation (rat) LC50: 1.17 mg/l/1ht[2] | |
| Inhalation (rat) LC50: 1.4 mg/l/4hr[1] | |
| Oral (rat) LD50: >2000 mg/kg[1] | |

| | |
|---|---|
| **naphtha petroleum, heavy alkylate** | |

| TOXICITY | IRRITATION |
|---|---|
| Not Available | Not Available |

| | |
|---|---|
| **carbon black** | |

| TOXICITY | IRRITATION |
|---|---|
| Dermal (rabbit) LD50: >3000 mg/kg[2] | Not Available |
| Oral (rat) LD50: >10000 mg/kg[1] | |

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

| | |
|---|---|
| **BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID** | Foetoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity; NOEL (maternal 60 mg/kg |
| **DIPROPYLENE GLYCOL DIGLYCIDYL ETHER** | The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.<br>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.<br>for 1,2-butylene oxide (ethyloxirane):<br>Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic<br>**NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.<br>MUTAGENICITY: In vitro genetic toxicity studies were positive. * Dow Chemical SDS |

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| **BISPHENOL A DIGLYCIDYL ETHER** | The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.<br>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. |
| **1-DECENE HOMOPOLYMER, HYDROGENATED** | for poly-alpha-olefins (PAOs):<br>PAOs are highly branched isoparaffinic chemicals produced by oligomerisation of 1-octene, 1-decene, and/or 1-dodecene. The crude polyalphaolefin mixture is then distilled into appropriate product fractions to meet specific viscosity specifications and hydrogenated.<br>Read across data exist for health effects endpoints from the following similar *hydrogenated* long chain branched alkanes derived from a C8, C10, and/or C12 alpha olefins:<br>▸ Decene homopolymer<br>▸ Decene/dodecene copolymer<br>▸ Octene/decene/dodecene copolymer<br>▸ Dodecene trimer<br>The data for these structural analogs demonstrated no evidence of health effects. In addition, there is evidence in the literature that alkanes with 30 or more carbon atoms are unlikely to be absorbed when administered orally. The physicochemical data suggest that it is unlikely that significant absorption will occur. If a substance of the size and structure of a typical PAO is absorbed, then the principal mechanisms of absorption after oral administration are likely to be passive diffusion and absorption by way of the lymphatic system. The former requires both good lipid solubility and good water solubility as the substance has to partition from an aqueous environment through a lipophilic membrane into another aqueous environment during absorption. Absorption by way of the lymphatics occurs by mechanisms analogous to those that absorb fatty acids and is limited by the size of the molecule. Lipophilicity generally enhances the ability of chemicals to cross biological membranes. Biotransformation by mixed function oxidases often increases the water solubility of a substance; however, existing data suggest that these substances will not undergo oxidation to more hydrophilic metabolites. Finally, a chemical must have an active functional group that can interact chemically or physically with the target cell or receptor upon reaching it; there are no moieties in PAOs that represent a functional group that may have biological activity. The water solubilities of a C10 dimer PAO and a C12 trimer PAO were determined to be <1 ppb and < 1 ppt respectively. The partition coefficient for a C12 trimer PAO was determined to be log Kow of >7 . Given the very low water solubility it is extremely unlikely that PAOs will be absorbed by passive diffusion following oral administration, and the size of the molecules suggest that the extent of lymphatic absorption is likely to be very low. Although PAOs are relatively large lipophilic compounds, and molecular size may be a critical limiting determinant for absorption, there is some evidence that these substances are absorbed. However, the lack of observed toxicity in the studies with PAOs suggests that these products are absorbed poorly, if at all.<br>Furthermore, a review of the literature regarding the absorption and metabolism of long chain alkanes indicates that alkanes with 30+ carbon atoms are unlikely to be absorbed. For example the absorption of squalane, an analogous C30 product, administered orally to male CD rats was examined - essentially all of the squalane was recovered unchanged in the faeces. At the same time, the hydrophobic properties of PAOs suggest that, should they be absorbed, they would undergo limited distribution in the aqueous systemic circulation and reach potential target organs in limited concentrations.<br>In addition to the general considerations discussed above, the low volatility of PAOs indicates that, under normal conditions of use or transportation, exposure by the inhalation route is unlikely. In particular, the high viscosity of these substances suggests that it would be difficult to generate a high concentration of respirable particles in the air.<br>**Acute toxicity:** PAOs (decene/dodecene copolymer, octene/decene/dodecene homo-polymer, and dodecene trimer) have been adequately tested for acute oral toxicity. There were no deaths when the test materials were administered at doses of 5,000 mg/kg (decene/dodecene copolymer and dodecene trimer) and at 2,000 mg/kg (octene/decene/dodecene copolymer) in rats. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.<br>PAOs (decene/dodecene copolymer, octene/decene/dodecene copolymer, and dodecene trimer) have been tested for acute dermal toxicity. No mortality was observed for any substance when administered at the limit dose of 2000 or 5000 mg/kg. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.<br>1-Decene, homopolymer, is absorbed (unexpectedly for a high molecular weight polymer) to a moderate degree in rat skin<br>and is eliminated slowly<br>PAOs (decene homopolymer, decene/dodecene copolymer, and decene trimer) have been tested for acute inhalation toxicity. Rats were exposed to aerosols of the substances at nominal atmospheric concentrations of 2.5, 5.0, and 5.06 mg/L, respectively, for four hours. These levels were the maximum attainable concentrations under the conditions of the tests, due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. The lack of mortality at concentrations at or above the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for these substances.<br>**Repeat dose toxicity:** Eight repeated-dose toxicity studies using two different animal species, rats and mice, and oral and dermal routes of administration have been conducted with three structural analogs. These data suggest that the structural analogs exhibit a low order of toxicity following repeated applications, due to their similarity in chemical structures and physicochemical properties.<br>One 28-day oral toxicity study in rats, one 90-day dermal and two 90-day dietary studies in rats, and a dermal carcinogenicity study in mice exist for decene homopolymer. A rat oral combined reproductive toxicity and 91-day systemic toxicity study was also conducted with decene homopolymer. In addition, 28-day rat oral toxicity studies exist for two structurally analogous substances (dodecene trimer and octene/decene/dodecene copolymer); and a 90-day rat dermal toxicity study exists for octene/decene/dodecene copolymer. Results from these studies show a low order of repeated dose toxicity. The dermal NOAEL for systemic toxicity studies was equal to or greater than 2000 mg/kg/day.<br>The oral NOAEL for 1-decene homopolymer is between 5,000 and 20,000 mg/kg/day in Sprague-Dawley rats.<br>Rats exposed repeatedly by dermal exposure at doses of 2000 mg/kg decene/dodecene copolymer showed increased incidences of hyperplasia of the sebaceous glands, hyperplasia/hyperkeratosis of the epidermis and dermal inflammation. These symptoms generally subsided within 2 weeks. Males showed decreased body weight gain and altered serum chemistry.<br>In a 90-day feeding study rats receiving 20000 ppm of 1-decene, homopolymer, hydrogenated did not exhibit any clinical signs of systemic toxicity. Marginal effects on clinical chemistry (glucose and ALT in males; sodium, phosphorus and calcium in females) were seen.<br>**Reproductive toxicity:** Data are available for decene homopolymer. Results from these studies show a low order of reproductive/ developmental toxicity. The NOAEL for reproductive toxicity was 1000 mg/kg/day, the highest concentration tested. The lack of effects on fertility in this study or effects on reproductive organs in this or other subchronic studies with closely related chemicals indicates that PAOs are unlikely to exert effects on reproduction.<br>**Developmental toxicity:** Decene homopolymer (with 10 ppm of an antioxidant) was administered once daily on gestation days 0-19 via dermal application to presumed-pregnant rats at doses of 0, 800, and 2000 mg/kg/day. Dermal administration of the test material did not adversely affect parameters of reproductive performance during gestation, nor did it adversely affect *in utero* survival and development of the offspring. The NOAEL in this study for developmental parameters was 2000 mg/kg/day.<br>**Genotoxicity:** Information for the following PAOs (decene homopolymer, octene/decene/dodecene copolymer, dodecene trimer; and decene/dodecene copolymer [*prepared from 10% C12 and 90% C10 alpha olefins; approx. 33% trimer and 51% tetramer, 16% pentamer and higher*]) is available. Either bacterial or mammalian gene mutation assays, *in vitro* chromosomal aberration assays, or *in vivo* chromosomal aberration assays have been conducted for these substances. Neither mutagenicity nor clastogenicity were exhibited by any of these substances in the referenced *in vivo* or *in vitro* tests, with or without metabolic activation.<br>**Carcinogenicity:** While alpha-olefin polymers have similar properties to mineral oils, they do not contain polycyclic aromatic hydrocarbons, or other known possible carcinogens.<br>Decene homopolymer produced no treatment-related tumors in C3H mice treated with a 50 ul/application twice weekly for 104 weeks. In addition, survival (56%) was greater than in any other group, including the untreated control.<br><br>(estimated) * Evidence of conjunctival changes ** No evidence of tissue damage [Inland Vacuum Industries] ^ US EPA HPV Challenge program October 2002 |

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| **NAPHTHA PETROLEUM, HEAVY ALKYLATE** | Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins.<br>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the 'hydrocarbon continuum hypothesis', and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.<br>**for petroleum:**<br>This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.<br>This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.<br>This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents<br>**Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.<br>**Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.<br>**Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.<br>**Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.<br><br>Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans. |
| **CARBON BLACK** | **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.<br>Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported |
| **BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & DIPROPYLENE GLYCOL DIGLYCIDYL ETHER & (C12-14)ALKYLGLYCIDYL ETHER & BISPHENOL A DIGLYCIDYL ETHER** | The following information refers to contact allergens as a group and may not be specific to this product.<br>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. |
| **BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & BISPHENOL A DIGLYCIDYL ETHER** | 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<br>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.<br>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. |
| **BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & BISPHENOL A DIGLYCIDYL ETHER** | The substance is classified by IARC as Group 3:<br>**NOT** classifiable as to its carcinogenicity to humans.<br>Evidence of carcinogenicity may be inadequate or limited in animal testing. |
| **BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & BISPHENOL A DIGLYCIDYL ETHER** | In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg).<br>**Reproductive and Developmental Toxicity**: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg.<br>**Carcinogenicity:** IARC concluded that 'there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals.' Its overall evaluation was 'Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3).<br>In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997).<br>**Genotoxicity**: In S. typhimurium strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg).<br>**Immunotoxicity:** Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs<br>-<br>**Consumer exposure** to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by |

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| | negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/ kg/body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weigh/day (male rats) from the 2-year carcinogenicity study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs. |
| **ALUMINA HYDRATE & ALUMINIUM OXIDE & ZINC BORATE HYDRATE & CARBON BLACK** | No significant acute toxicological data identified in literature search. |

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| **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 available but does not fill the criteria for classification*
✔ *– Data available to make classification*
⊘ *– Data Not Available to make classification*

## SECTION 12 ECOLOGICAL INFORMATION

### 12.1. Toxicity

**834FX Black Flexible Epoxy, Thermally Conductive–Flame Retardant, Encapsulating and Potting Compound (Part A)**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**bisphenol A/ diglycidyl ether resin, liquid**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 1.2mg/L | 2 |
| EC50 | 72 | Algae or other aquatic plants | 9.4mg/L | 2 |
| NOEC | 72 | Algae or other aquatic plants | 2.4mg/L | 2 |

**alumina hydrate**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 0.2262mg/L | 2 |
| EC50 | 48 | Crustacea | 0.7364mg/L | 2 |
| EC50 | 96 | Algae or other aquatic plants | 0.0054mg/L | 2 |
| NOEC | 72 | Algae or other aquatic plants | >=0.004mg/L | 2 |

**ammonium polyphosphate**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 70mg/L | 4 |
| EC50 | 48 | Crustacea | 813mg/L | 4 |

**aluminium oxide**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 0.0029mg/L | 2 |
| EC50 | 48 | Crustacea | 0.7364mg/L | 2 |
| EC50 | 96 | Algae or other aquatic plants | 0.0054mg/L | 2 |
| NOEC | 72 | Algae or other aquatic plants | >=0.004mg/L | 2 |

**dipropylene glycol diglycidyl ether**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**(C12-14)alkylglycidyl ether**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**zinc borate hydrate**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

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|---|---|---|---|---|---|
| **bisphenol A diglycidyl ether** | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
| | LC50 | 96 | Fish | 1.2mg/L | 2 |
| | EC50 | 72 | Algae or other aquatic plants | 9.4mg/L | 2 |
| | NOEC | 72 | Algae or other aquatic plants | 2.4mg/L | 2 |

| | | | | | |
|---|---|---|---|---|---|
| **1-decene homopolymer, hydrogenated** | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
| | Not Available | Not Available | Not Available | Not Available | Not Available |

| | | | | | |
|---|---|---|---|---|---|
| **naphtha petroleum, heavy alkylate** | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
| | EC50 | 72 | Algae or other aquatic plants | =13mg/L | 1 |
| | NOEC | 72 | Algae or other aquatic plants | =0.1mg/L | 1 |

| | | | | | |
|---|---|---|---|---|---|
| **carbon black** | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
| | LC50 | 96 | Fish | =1000mg/L | 1 |
| | NOEC | 96 | Fish | =1000mg/L | 1 |

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

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
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 Fe3+ ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.

Significant environmental findings are limited. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit common characteristics with respect to environmental fate and ecotoxicology. One such oxirane is ethyloxirane and data presented here may be taken as representative.
for 1,2-butylene oxide (ethyloxirane):
**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

**Continued...**

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).
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(H2O)6]3+, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H2O)5(OH)]2+, [Al(H2O)4(OH)2]+). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)2+ and Al(OH)2+, while the solid Al(OH)3 is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)4- is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)3, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.
Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.
Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.
The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.
Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.
Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.
Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.
The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic.
Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.
Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.
**Ecotoxicity:**
***Freshwater species pH >6.5***
Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar)* - 106 mg/L; Chronic NOEC (8-28 d): 7 spp,NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.
Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L
Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L
Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L
***Freshwater species pH <6.5 (all between pH 4.5 and 6.0)***
Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L
Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)
Alga: 1 sp NOEC growth 2.0 mg/L
Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.
The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4.5.4, with a maximum toxicity occurring around pH 5.0.5.2. The inorganic single unit aluminium species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.
Drinking Water Standards:
aluminium: 200 ug/l (UK max.)
200 ug/l (WHO guideline)
chloride: 400 mg/l (UK max.)
250 mg/l (WHO guideline)
fluoride: 1.5 mg/l (UK max.)
1.5 mg/l (WHO guideline)
nitrate: 50 mg/l (UK max.)
50 mg/l (WHO guideline)
sulfate: 250 mg/l (UK max.)
Soil Guideline: none available.
Air Quality Standards: none available.

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

**Continued...**

drinking purposes, on fisheries, and on the use of lakes for recreational purposes.
**DO NOT** discharge into sewer or waterways.

### 12.2. Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | HIGH | HIGH |
| bisphenol A diglycidyl ether | HIGH | HIGH |
| 1-decene homopolymer, hydrogenated | LOW | LOW |

### 12.3. Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | LOW (LogKOW = 2.6835) |
| bisphenol A diglycidyl ether | MEDIUM (LogKOW = 3.8446) |
| 1-decene homopolymer, hydrogenated | HIGH (LogKOW = 5.116) |

### 12.4. Mobility in soil

| Ingredient | Mobility |
|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | LOW (KOC = 51.43) |
| bisphenol A diglycidyl ether | LOW (KOC = 1767) |
| 1-decene homopolymer, hydrogenated | LOW (KOC = 1724) |

### 12.5.Results of PBT and vPvB assessment

| | P | B | T |
|---|---|---|---|
| Relevant available data | Not Available | Not Available | Not Available |
| PBT Criteria fulfilled? | Not Available | Not Available | Not Available |

### 12.6. Other adverse effects

No data available

## SECTION 13 DISPOSAL CONSIDERATIONS

### 13.1. Waste treatment methods

| | |
|---|---|
| **Product / Packaging disposal** | ▸ Containers may still present a chemical hazard/ danger when empty.<br>▸ Return to supplier for reuse/ recycling if possible.<br>Otherwise:<br>▸ 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.<br>▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product.<br>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.<br>A Hierarchy of Controls seems to be common - the user should investigate:<br>▸ Reduction<br>▸ Reuse<br>▸ Recycling<br>▸ Disposal (if all else fails)<br>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.<br>Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.<br>▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains.<br>▸ It may be necessary to collect all wash water for treatment before disposal.<br>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.<br>▸ Where in doubt contact the responsible authority.<br>▸ Recycle wherever possible or consult manufacturer for recycling options.<br>▸ Consult State Land Waste Authority for disposal.<br>▸ Bury or incinerate residue at an approved site.<br>▸ Recycle containers if possible, or dispose of in an authorised landfill. |
| **Waste treatment options** | Not Available |
| **Sewage disposal options** | Not Available |

## SECTION 14 TRANSPORT INFORMATION

### Labels Required

**Continued...**

| | |
|---|---|
| |  Limited Quantity: (For 834FX-450ML, 834FX-1.7L kits ship as per Part B) |

### Land transport (ADR)

| | |
|---|---|
| **14.1.UN number** | 3082 |
| **14.2.UN proper shipping name** | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid and zinc borate hydrate) |
| **14.3. Transport hazard class(es)** | Class: 9<br>Subrisk: Not Applicable |
| **14.4.Packing group** | III |
| **14.5.Environmental hazard** | Environmentally hazardous |
| **14.6. Special precautions for user** | Hazard identification (Kemler): 90<br>Classification code: M6<br>Hazard Label: 9<br>Special provisions: 274 335 375 601<br>Limited quantity: 5 L |

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

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

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

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

**Continued...**

### Inland waterways transport (ADN)

| | |
|---|---|
| **14.1. UN number** | 3082 |
| **14.2. UN proper shipping name** | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid and zinc borate hydrate) |
| **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<br>Special provisions: 274; 335; 375; 601<br>Limited quantity: 5 L<br>Equipment required: PP<br>Fire cones number: 0 |

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

Not Applicable

## SECTION 15 REGULATORY INFORMATION

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

**BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID(25085-99-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

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

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

European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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

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

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

European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
UK Workplace Exposure Limits (WELs)

**DIPROPYLENE GLYCOL DIGLYCIDYL ETHER(41638-13-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Not Applicable

**(C12-14)ALKYLGLYCIDYL ETHER(68609-97-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
European Customs Inventory of Chemical Substances ECICS (English)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

**ZINC BORATE HYDRATE(138265-88-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Not Applicable

**BISPHENOL A DIGLYCIDYL ETHER(25068-38-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
European Customs Inventory of Chemical Substances ECICS (English)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
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

**1-DECENE HOMOPOLYMER, HYDROGENATED(68037-01-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

European Customs Inventory of Chemical Substances ECICS (English)

**NAPHTHA PETROLEUM, HEAVY ALKYLATE(64741-65-7.) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

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
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 2) Carcinogens: category 1B (Table 3.1)/category 2 (Table 3.2)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 4) Mutagens: category 1B (Table 3.1)/category 2 (Table 3.2)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

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

**Continued...**

| | |
|---|---|
| EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances | European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English) |
| European Customs Inventory of Chemical Substances ECICS (English) | International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs |
| European List of Notified Chemical Substances (ELINCS) | UK Workplace Exposure Limits (WELs) |
| European Trade Union Confederation (ETUC) Priority List for REACH Authorisation | |

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments

### 15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

### ECHA SUMMARY

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | 25085-99-8 | 603-074-00-8 | 01-2119456619-26-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2 | GHS09, GHS07, Wng | H315, H317, H319, H411 |
| 2 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Skin Corr. 1A, Acute Tox. 4, Aquatic Chronic 3, Aquatic Acute 1, Aquatic Chronic 4, Aquatic Chronic 1, STOT SE 3 | GHS09, Dgr, GHS08 | H315, H317, H319, H410, H372, H335, H400 |
| 1 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2 | GHS09, GHS07, Wng | H315, H317, H319, H411 |
| 2 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2 | GHS09, GHS07, Wng | H315, H317, H319, H411 |
| 1 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2 | GHS09, GHS07, Wng | H315, H317, H319, H411 |
| 2 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Skin Sens. 1B, Skin Sens. 1A | GHS09, GHS07, Wng | H315, H317, H319, H411 |
| 1 | Skin Irrit. 2, Skin Sens. 1A, Aquatic Chronic 2 | GHS09, GHS07, Wng | H315, H317, H411 |
| 2 | Skin Irrit. 2, Skin Sens. 1A, Aquatic Chronic 2 | GHS09, GHS07, Wng | H315, H317, H411 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| alumina hydrate | 21645-51-2 | Not Available | 01-2119529246-39-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | Skin Irrit. 2, Eye Irrit. 2, STOT SE 3, Aquatic Acute 1, Aquatic Chronic 1 | GHS07, Wng, GHS09 | H315, H319, H335, H410 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| ammonium polyphosphate | 68333-79-9 | Not Available | Not Available |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | Acute Tox. 4, Eye Irrit. 2 | GHS07, Wng | H302, H319 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| aluminium oxide | 1344-28-1. | Not Available | 01-2119529248-35-XXXX, 01-2119817795-27-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | STOT SE 3, STOT RE 1, Skin Sens. 1, Muta. 2, Carc. 1B, Repr. 2, Aquatic Chronic 3, Acute Tox. 4, Skin Irrit. 2, Eye Irrit. 2, STOT RE 2, Flam. Liq. 2 | GHS08, Dgr, GHS09, GHS02 | H370, H335, H372, H317, H341, H350, H361, H412, H302, H332, H315, H319, H220, H225 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| dipropylene glycol diglycidyl ether | 41638-13-5 | Not Available | Not Available |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, STOT SE 3, Aquatic Chronic 3 | GHS07, Wng | H315, H317, H319, H335, H412 |
| 2 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, STOT SE 3, Aquatic Chronic 3, Aquatic Chronic 2 | GHS07, Wng, GHS09 | H315, H317, H319, H335, H411 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| (C12-14)alkylglycidyl ether | 68609-97-2 | 603-103-00-4 | 01-2119485289-22-XXXX |

| Harmonisation (C&L | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|

**Continued...**

| Inventory) | | | |
|---|---|---|---|
| 1 | Skin Irrit. 2, Skin Sens. 1 | GHS07, Wng | H315, H317 |
| 2 | Skin Irrit. 2, Skin Sens. 1, Aquatic Chronic 2, Acute Tox. 4, Eye Irrit. 2 | GHS07, Wng, GHS09 | H315, H317, H411 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| zinc borate hydrate | 138265-88-0 | Not Available | Not Available |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | Aquatic Acute 1, Aquatic Chronic 1, Eye Irrit. 2, Repr. 2, Aquatic Chronic 2 | GHS09, Wng, GHS08 | H400, H410, H319, H361fd |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| bisphenol A diglycidyl ether | 25068-38-6 | 603-073-00-2, 603-074-00-8 | 01-2119456619-26-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2 | GHS07, Wng | H315, H317, H319 |
| 2 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, STOT RE 2, Aquatic Acute 1, Aquatic Chronic 3 | Wng, GHS09, GHS08 | H315, H317, H319, H410, H400 |
| 1 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2 | GHS09, GHS07, Wng | H315, H317, H319, H411 |
| 2 | Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Skin Corr. 1A, Acute Tox. 4, Aquatic Chronic 3, Aquatic Acute 1, Aquatic Chronic 4, Aquatic Chronic 1, STOT SE 3 | GHS09, Dgr, GHS08 | H315, H317, H319, H410, H372, H335, H400 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| 1-decene homopolymer, hydrogenated | 68037-01-4 | Not Available | 01-2119486452-34-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Asp. Tox. 1 | GHS08, Dgr | H304 |
| 2 | Asp. Tox. 1, Aquatic Chronic 3, Acute Tox. 4, Eye Irrit. 2, STOT SE 3, STOT RE 2, Flam. Liq. 3 | GHS08, Dgr, GHS09, GHS02 | H304, H412, H332, H319, H335, H373, H226 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| naphtha petroleum, heavy alkylate | 64741-65-7. | 649-275-00-4 | Not Available |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Flam. Liq. 3, Asp. Tox. 1 | GHS02, GHS08, Dgr | H226, H304 |
| 2 | Flam. Liq. 3, Asp. Tox. 1, Muta. 1B, Carc. 1B, Aquatic Chronic 4, Aquatic Chronic 2, Skin Irrit. 2, Aquatic Acute 1, Aquatic Chronic 1, Acute Tox. 3, Flam. Liq. 1, STOT SE 3, Repr. 2, Flam. Liq. 2, Aquatic Chronic 3 | GHS02, GHS08, Dgr, GHS09, GHS06 | H304, H340, H350, H315, H410, H331, H224, H336, H361 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| carbon black | 1333-86-4 | Not Available | 01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | Carc. 2, STOT SE 3, Eye Irrit. 2, STOT RE 2, STOT RE 1, Aquatic Chronic 4, Self-heat. 1, Self-heat. 2, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Flam. Sol. 2, Acute Tox. 4 | GHS08, Dgr, GHS06, GHS02, GHS09 | H351, H335, H319, H372, H251, H228, H315, H370, H410, H332 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| National Inventory | Status |
|---|---|
| Australia - AICS | Y |
| Canada - DSL | N (zinc borate hydrate) |
| Canada - NDSL | N (dipropylene glycol diglycidyl ether; 1-decene homopolymer, hydrogenated; (C12-14)alkylglycidyl ether; zinc borate hydrate; bisphenol A/ diglycidyl ether resin, liquid; bisphenol A diglycidyl ether; aluminium oxide; alumina hydrate; naphtha petroleum, heavy alkylate; carbon black; ammonium polyphosphate) |
| China - IECSC | Y |
| Europe - EINEC / ELINCS / NLP | N (dipropylene glycol diglycidyl ether; zinc borate hydrate) |
| Japan - ENCS | N (dipropylene glycol diglycidyl ether; (C12-14)alkylglycidyl ether; zinc borate hydrate; bisphenol A/ diglycidyl ether resin, liquid; aluminium oxide; naphtha petroleum, heavy alkylate) |
| Korea - KECI | N (zinc borate hydrate) |

**Continued...**

| | |
|---|---|
| New Zealand - NZIoC | Y |
| Philippines - PICCS | N (zinc borate hydrate) |
| USA - TSCA | N (zinc borate hydrate) |
| ***Legend:*** | *Y = All ingredients are on the inventory*<br>*N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)* |

## SECTION 16 OTHER INFORMATION

### Full text Risk and Hazard codes

| | |
|---|---|
| **H220** | Extremely flammable gas. |
| **H224** | Extremely flammable liquid and vapour. |
| **H225** | Highly flammable liquid and vapour. |
| **H226** | Flammable liquid and vapour. |
| **H228** | Flammable solid. |
| **H251** | Self-heating: may catch fire. |
| **H304** | May be fatal if swallowed and enters airways. |
| **H331** | Toxic if inhaled. |
| **H332** | Harmful if inhaled. |
| **H335** | May cause respiratory irritation. |
| **H336** | May cause drowsiness or dizziness. |
| **H340** | May cause genetic defects. |
| **H341** | Suspected of causing genetic defects. |
| **H350** | May cause cancer. |
| **H351** | Suspected of causing cancer. |
| **H360** | May damage fertility or the unborn child. |
| **H361fd** | Suspected of damaging fertility. Suspected of damaging the unborn child. |
| **H370** | Causes damage to organs. |
| **H372** | Causes damage to organs through prolonged or repeated exposure. |
| **H373** | May cause damage to organs through prolonged or repeated exposure. |
| **H400** | Very toxic to aquatic life. |
| **H410** | Very toxic to aquatic life with long lasting effects. |
| **H412** | Harmful to aquatic life with long lasting effects. |
| **H413** | May cause long lasting harmful effects to aquatic life. |

### Other information

### Ingredients with multiple cas numbers

| Name | CAS No |
|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | 25068-38-6, 25085-99-8 |
| alumina hydrate | 14762-49-3, 21645-51-2 |
| bisphenol A diglycidyl ether | 1675-54-3, 116161-20-7, 170962-54-6, 47424-12-4, 85101-00-4, 25068-38-6 |

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

**Continued...**

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
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

# 834FX-B

**MG Chemicals UK Limited**

Chemwatch Hazard Alert Code: **3**

Version No: **1.4**
Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date: **29/06/2017**
Print Date: **17/08/2017**
L.REACH.GBR.EN

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

### 1.1. Product Identifier

| | |
|---|---|
| **Product name** | 834FX-B |
| **Synonyms** | SDS Code: 834FX-Part B, 834FX-450ML, 834FX-1.7L, 834FX-7.4L, 834FX-40L |
| **Proper shipping name** | AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains polypropylene glycol bis(2-aminopropyl ether), trimethylhexamethylene diamine and cocoamine) |
| **Other means of identification** | Not Available |

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

| | |
|---|---|
| **Relevant identified uses** | epoxy hardener |
| **Uses advised against** | Not Applicable |

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

| | | |
|---|---|---|
| **Registered company name** | MG Chemicals UK Limited | MG Chemicals (Head office) |
| **Address** | Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom | 9347 - 193 Street Surrey V4N 4E7 British Columbia Canada |
| **Telephone** | +(44) 1663 362888 | +(1) 800-201-8822 |
| **Fax** | Not Available | +(1) 800-708-9888 |
| **Website** | Not Available | www.mgchemicals.com |
| **Email** | sales@mgchemicals.com | Info@mgchemicals.com |

### 1.4. Emergency telephone number

| | | |
|---|---|---|
| **Association / Organisation** | CHEMTREC | Not Available |
| **Emergency telephone numbers** | +(44) 870-8200418 | Not Available |
| **Other emergency telephone numbers** | +(1) 703-527-3887 | Not Available |

## SECTION 2 HAZARDS IDENTIFICATION

### 2.1. Classification of the substance or mixture

| | |
|---|---|
| **Classification according to regulation (EC) No 1272/2008 [CLP] [1]** | H302 - Acute Toxicity (Oral) Category 4, H314 - Skin Corrosion/Irritation Category 1A, H317 - Skin Sensitizer Category 1, H373 - Specific target organ toxicity - repeated exposure Category 2, H361 - Reproductive Toxicity Category 2, H410 - Chronic Aquatic Hazard Category 1 |
| ***Legend:*** | *1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI* |

### 2.2. Label elements

| | |
|---|---|
| **Hazard pictogram(s)** |  |
| **SIGNAL WORD** | **DANGER** |

### Hazard statement(s)

| | |
|---|---|
| **H302** | Harmful if swallowed. |
| **H314** | Causes severe skin burns and eye damage. |
| **H317** | May cause an allergic skin reaction. |
| **H373** | May cause damage to organs through prolonged or repeated exposure. |

**Continued...**

| | |
|---|---|
| **H361** | Suspected of damaging fertility or the unborn child. |
| **H410** | Very toxic to aquatic life with long lasting effects. |

### Supplementary statement(s)

Not Applicable

### Precautionary statement(s) Prevention

| | |
|---|---|
| **P201** | Obtain special instructions before use. |
| **P260** | Do not breathe dust/fume/gas/mist/vapours/spray. |
| **P280** | Wear protective gloves/protective clothing/eye protection/face protection. |
| **P270** | Do not eat, drink or smoke when using this product. |
| **P273** | Avoid release to the environment. |
| **P272** | Contaminated work clothing should not be allowed out of the workplace. |

### Precautionary statement(s) Response

| | |
|---|---|
| **P301+P330+P331** | IF SWALLOWED: Rinse mouth. Do NOT induce vomiting. |
| **P303+P361+P353** | IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower. |
| **P305+P351+P338** | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| **P308+P313** | IF exposed or concerned: Get medical advice/ attention. |
| **P310** | Immediately call a POISON CENTER/doctor/physician/first aider. |
| **P302+P352** | IF ON SKIN: Wash with plenty of water and soap. |
| **P363** | Wash contaminated clothing before reuse. |
| **P333+P313** | If skin irritation or rash occurs: Get medical advice/attention. |
| **P362+P364** | Take off contaminated clothing and wash it before reuse. |
| **P391** | Collect spillage. |
| **P301+P312** | IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell. |
| **P304+P340** | IF INHALED: Remove person to fresh air and keep comfortable for breathing. |

### Precautionary statement(s) Storage

| | |
|---|---|
| **P405** | Store locked up. |

### Precautionary statement(s) Disposal

| | |
|---|---|
| **P501** | Dispose of contents/container in accordance with local regulations. |

### 2.3. Other hazards

Inhalation may produce health damage*.

Limited evidence of a carcinogenic effect*.

Possible respiratory sensitizer*.

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

## SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

### 3.1.Substances

See 'Composition on ingredients' in Section 3.2

### 3.2.Mixtures

| 1.CAS No<br>2.EC No<br>3.Index No<br>4.REACH No | %[weight] | Name | Classification according to regulation (EC) No 1272/2008 [CLP] |
|---|---|---|---|
| 1.21645-51-2<br>2.244-492-7<br>3.Not Available<br>4.01-2119529246-39-XXXX | 26 | alumina hydrate | Not Applicable |
| 1.9046-10-0<br>2.Not Available<br>3.Not Available<br>4.01-2119557899-12-XXXX | 19 | polypropylene glycol bis(2-aminopropyl ether) | Metal Corrosion Category 1, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1A, Serious Eye Damage Category 1, Chronic Aquatic Hazard Category 3; H290, H302, H312, H314, H412 [1] |
| 1.68333-79-9<br>2.269-789-9<br>3.Not Available<br>4.Not Available | 19 | ammonium polyphosphate | Chronic Aquatic Hazard Category 4; H413 [1] |

**Continued...**

| | | | |
|---|---|---|---|
| 1.1344-28-1.<br>2.215-691-6<br>3.Not Available<br>4.01-2119529248-35-XXXX\|01-2119817795-27-XXXX | 16 | aluminium oxide | Not Applicable |
| 1.61788-44-1<br>2.262-975-0<br>3.Not Available<br>4.01-2119557886-19-XXXX\|01-2119979575-18-XXXX\|01-2119980970-27-XXXX | 6 | phenol, styrenated | Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Germ cell mutagenicity Category 2, Chronic Aquatic Hazard Category 2; H315, H319, H341, H411 [1] |
| 1.138265-88-0<br>2.Not Available<br>3.Not Available<br>4.Not Available | 5 | zinc borate hydrate | Reproductive Toxicity Category 1B, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H360, H410 [1] |
| 1.61788-46-3<br>2.262-977-1<br>3.612-285-00-4<br>4.01-2119971069-29-XXXX\|01-2119473798-17-XXXX | 3 | cocoamine | Acute Toxicity (Oral) Category 4, Aspiration Hazard Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - repeated exposure Category 2 (gastro-intestinal tract, liver, immune system), Skin Corrosion/Irritation Category 1B, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H302, H304, H335, H373, H314, H410 [3] |
| 1.25620-58-0<br>2.247-134-8<br>3.Not Available<br>4.01-2119560598-25-XXXX | 3 | trimethylhexamethylene diamine | Metal Corrosion Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1B, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 3; H290, H302, H314, H317, H412 [1] |
| 1.1333-86-4<br>2.215-609-9<br>3.Not Available<br>4.01-2119384822-32-XXXX\|01-2119489801-30-XXXX\|01-2119475601-40-XXXX | 0.5 | carbon black | Carcinogenicity Category 2; H351 [1] |
| ***Legend:*** | *1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI 4. Classification drawn from C&L* | | |

## SECTION 4 FIRST AID MEASURES

### 4.1. Description of first aid measures

| | |
|---|---|
| **Eye Contact** | If this product comes in contact with the eyes:<br>▸ Immediately hold eyelids apart and flush the eye continuously with running water.<br>▸ 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.<br>▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.<br>▸ Transport to hospital or doctor without delay.<br>▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.<br>For amines:<br>▸ If liquid amines come in contact with the eyes, irrigate immediately and continuously with low pressure flowing water, preferably from an eye wash fountain, for 15 to 30 minutes.<br>▸ For more effective flushing of the eyes, use the fingers to spread apart and hold open the eyelids. The eyes should then be “rolled” or moved in all directions.<br>▸ Seek immediate medical attention, preferably from an ophthalmologist. |
| **Skin Contact** | If skin or hair contact occurs:<br>▸ Immediately flush body and clothes with large amounts of water, using safety shower if available.<br>▸ Quickly remove all contaminated clothing, including footwear.<br>▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.<br>▸ Transport to hospital, or doctor.<br>For amines:<br>▸ In case of major exposure to liquid amine, promptly remove any contaminated clothing, including rings, watches, and shoe, preferably under a safety shower.<br>▸ Wash skin for 15 to 30 minutes with plenty of water and soap. Call a physician immediately.<br>▸ Remove and dry-clean or launder clothing soaked or soiled with this material before reuse. Dry cleaning of contaminated clothing may be more effective than normal laundering.<br>▸ Inform individuals responsible for cleaning of potential hazards associated with handling contaminated clothing.<br>▸ Discard contaminated leather articles such as shoes, belts, and watchbands.<br>▸ Note to Physician: Treat any skin burns as thermal burns. After decontamination, consider the use of cold packs and topical antibiotics. |
| **Inhalation** | ▸ If fumes or combustion products are inhaled remove from contaminated area.<br>▸ Lay patient down. Keep warm and rested.<br>▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.<br>▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.<br>▸ Transport to hospital, or doctor, without delay.<br>▸ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema.<br>▸ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).<br>▸ As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested.<br>▸ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered.<br>**This must definitely be left to a doctor or person authorised by him/her.**<br>(ICSC13719)<br>For amines:<br>▸ All employees working in areas where contact with amine catalysts is possible should be thoroughly trained in the administration of appropriate first aid procedures.<br>▸ Experience has demonstrated that prompt administration of such aid can minimize the effects of accidental exposure.<br>▸ Promptly move the affected person away from the contaminated area to an area of fresh air.<br>▸ Keep the affected person calm and warm, but not hot.<br>▸ If breathing is difficult, oxygen may be administered by a qualified person.<br>▸ If breathing stops, give artificial respiration. Call a physician at once. |

**Continued...**

| | |
|---|---|
| **Ingestion** | ▸ For advice, contact a Poisons Information Centre or a doctor at once.<br>▸ Urgent hospital treatment is likely to be needed.<br>▸ If swallowed do **NOT** induce vomiting.<br>▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.<br>▸ Observe the patient carefully.<br>▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.<br>▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.<br>▸ Transport to hospital or doctor without delay.<br>For amines:<br>▸ If liquid amine are ingested, have the affected person drink several glasses of water or milk.<br>▸ Do not induce vomiting.<br>▸ Immediately transport to a medical facility and inform medical personnel about the nature of the exposure. The decision of whether to induce vomiting should be made by an attending physician. |

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

See Section 11

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

Treat symptomatically.

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

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

[Ellenhorn and Barceloux: Medical Toxicology]
For acute or short-term repeated exposures to highly alkaline materials:

- Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- Oxygen is given as indicated.
- The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.
INGESTION:

- Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

- Neutralising agents should never be given since exothermic heat reaction may compound injury.

* Catharsis and emesis are absolutely contra-indicated.
* Activated charcoal does not absorb alkali.
* Gastric lavage should not be used.

Supportive care involves the following:

- Withhold oral feedings initially.
- If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

- Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]
For amines:

- Certain amines may cause injury to the respiratory tract and lungs if aspirated. Also, such products may cause tissue destruction leading to stricture. If lavage is performed, endotracheal and/or esophagoscopic control is suggested.
- No specific antidote is known.
- Care should be supportive and treatment based on the judgment of the physician in response to the reaction of the patient.

Laboratory animal studies have shown that a few amines are suspected of causing depletion of certain white blood cells and their precursors in lymphoid tissue. These effects may be due to an immunosuppressive mechanism.
Some persons with hyperreactive airways (e.g., asthmatic persons) may experience wheezing attacks (bronchospasm) when exposed to airway irritants.
Lung injury may result following a single massive overexposure to high vapour concentrations or multiple exposures to lower concentrations of any pulmonary irritant material.
Health effects of amines, such as skin irritation and transient corneal edema (“blue haze,” “halo effect,” “glaucopsia”), are best prevented by means of formal worker education, industrial hygiene monitoring, and exposure control methods. Persons who are highly sensitive to the triggering effect of non-specific irritants should not be assigned to jobs in which such agents are used, handled, or manufactured.
**Medical surveillance programs** should consist of a pre-placement evaluation to determine if workers or applicants have any impairments (e.g., hyperreactive airways or bronchial asthma) that would limit their fitness for work in jobs with potential for exposure to amines. A clinical baseline can be established at the time of this evaluation.
Periodic medical evaluations can have significant value in the early detection of disease and in providing an opportunity for health counseling.
Medical personnel conducting medical surveillance of individuals potentially exposed to polyurethane amine catalysts should consider the following:

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

Persons who are concurrently exposed to isocyanates also should be kept under medical surveillance.
Pre-existing medical conditions generally aggravated by exposure include skin disorders and allergies, chronic respiratory disease (e.g. bronchitis, asthma, emphysema), liver disorders, kidney disease, and eye disease.
Broadly speaking, exposure to amines, as characterised by amine catalysts, may cause effects similar to those caused by exposure to ammonia. As such, amines should be considered potentially injurious to any tissue that is directly contacted.
Inhalation of aerosol mists or vapors, especially of heated product, can result in chemical pneumonitis, pulmonary edema, laryngeal edema, and delayed scarring of the airway or other affected organs. There is no specific treatment.

**Continued...**

Clinical management is based upon supportive treatment, similar to that for thermal burns.
Persons with major skin contact should be maintained under medical observation for at least 24 hours due to the possibility of delayed reactions.
**Polyurethene Amine Catalysts: Guidelines for Safe Handling and Disposal Technical Bulletin June 2000**
**Alliance for Polyurethanes Industry**

## SECTION 5 FIREFIGHTING MEASURES

### 5.1. Extinguishing media

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

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

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

### 5.3. Advice for firefighters

| | |
|---|---|
| **Fire Fighting** | ▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ Wear full body protective clothing with breathing apparatus.<br>▸ Prevent, by any means available, spillage from entering drains or water course.<br>▸ Use fire fighting procedures suitable for surrounding area.<br>▸ **Do not** approach containers suspected to be hot.<br>▸ Cool fire exposed containers with water spray from a protected location.<br>▸ If safe to do so, remove containers from path of fire.<br>▸ Equipment should be thoroughly decontaminated after use.<br>For amines:<br>▸ For firefighting, cleaning up large spills, and other emergency operations, workers must wear a self-contained breathing apparatus with full face-piece, operated in a pressure-demand mode.<br>▸ Airline and air purifying respirators should not be worn for firefighting or other emergency or upset conditions.<br>▸ Respirators should be used in conjunction with a respiratory protection program, which would include suitable fit testing and medical evaluation of the user. |
| **Fire/Explosion Hazard** | ▸ Combustible.<br>▸ Slight fire hazard when exposed to heat or flame.<br>▸ Heating may cause expansion or decomposition leading to violent rupture of containers.<br>▸ On combustion, may emit toxic fumes of carbon monoxide (CO).<br>▸ May emit acrid smoke.<br>▸ Mists containing combustible materials may be explosive.<br>Combustion products include:<br>carbon dioxide ($CO_2$)<br>nitrogen oxides (NOx)<br>phosphorus oxides (POx)<br>other pyrolysis products typical of burning organic material.<br>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.<br>May emit corrosive fumes. |

## SECTION 6 ACCIDENTAL RELEASE MEASURES

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

See section 8

### 6.2. Environmental precautions

See section 12

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

| | |
|---|---|
| **Minor Spills** | Environmental hazard - contain spillage.<br>▸ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.<br>▸ Check regularly for spills and leaks.<br>▸ Clean up all spills immediately.<br>▸ Avoid breathing vapours and contact with skin and eyes.<br>▸ Control personal contact with the substance, by using protective equipment.<br>▸ Contain and absorb spill with sand, earth, inert material or vermiculite.<br>▸ Wipe up.<br>▸ Place in a suitable, labelled container for waste disposal.<br>for amines:<br>▸ If possible (i.e., without risk of contact or exposure), stop the leak.<br>▸ Contain the spilled material by diking, then neutralize.<br>▸ Next, absorb the neutralized product with clay, sawdust, vermiculite, or other inert absorbent and shovel into containers.<br>▸ Store the containers outdoors.<br>▸ Brooms and mops should be disposed of, along with any remaining absorbent, in accordance with all applicable federal, state, and local regulations and requirements.<br>▸ Decontamination of floors and other hard surfaces after the spilled material has been removed may be accomplished by using a 5% solution of acetic acid, followed by very hot water<br>▸ Dispose of the material in full accordance with all federal, state, and local laws and regulations governing the disposal of chemical wastes.<br>▸ Waste materials from an amine catalyst spill or leak may be “hazardous wastes” that are regulated under various laws. |
| **Major Spills** | Environmental hazard - contain spillage. |

**Continued...**

Chemical Class: bases
For release onto land: recommended sorbents listed in order of priority.

| SORBENT TYPE | RANK | APPLICATION | COLLECTION | LIMITATIONS |
|---|---|---|---|---|
| LAND SPILL - SMALL | | | | |
| cross-linked polymer - particulate | 1 | shovel | shovel | R,W,SS |
| cross-linked polymer - pillow | 1 | throw | pitchfork | R, DGC, RT |
| sorbent clay - particulate | 2 | shovel | shovel | R, I, P |
| foamed glass - pillow | 2 | throw | pitchfork | R, P, DGC, RT |
| expanded minerals - particulate | 3 | shovel | shovel | R, I, W, P, DGC |
| foamed glass - particulate | 4 | shovel | shovel | R, W, P, DGC, |
| LAND SPILL - MEDIUM | | | | |
| cross-linked polymer -particulate | 1 | blower | skiploader | R,W, SS |
| sorbent clay - particulate | 2 | blower | skiploader | R, I, P |
| expanded mineral - particulate | 3 | blower | skiploader | R, I,W, P, DGC |
| cross-linked polymer - pillow | 3 | throw | skiploader | R, DGC, RT |
| foamed glass - particulate | 4 | blower | skiploader | R, W, P, DGC |
| foamed glass - pillow | 4 | throw | skiploader | R, P, DGC., RT |

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

- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear full body protective clothing with breathing apparatus.
- Prevent, by any means available, spillage from entering drains or water course.
- Consider evacuation (or protect in place).
- Stop leak if safe to do so.
- Contain spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Neutralise/decontaminate residue (see Section 13 for specific agent).
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- If contamination of drains or waterways occurs, advise emergency services.

For amines:

- First remove all ignition sources from the spill area.
- Have firefighting equipment nearby, and have firefighting personnel fully trained in the proper use of the equipment and in the procedures used in fighting a chemical fire.
- Spills and leaks of polyurethane amine catalysts should be contained by diking, if necessary, and cleaned up only by properly trained and equipped personnel. All others should promptly leave the contaminated area and stay upwind.
- Protective equipment for cleanup crews should include appropriate respiratory protective devices and impervious clothing, footwear, and gloves.
- All work areas should be equipped with safety showers and eyewash fountains in good working order.
- Any material spilled or splashed onto the skin should be quickly washed off.
- Spills or releases may need to be reported to federal, state, and local authorities. This reporting contingency should be a part of a site's emergency response plan.
- Protective equipment should be used during emergency situations whenever there is a likelihood of exposure to liquid amines or to excessive concentrations of amine vapor. "Emergency" may be defined as any occurrence, such as, but not limited to, equipment failure, rupture of containers, or failure of control equipment that results in an uncontrolled release of amine liquid or vapor.
- Emergency protective equipment should include:
- • Self-contained breathing apparatus, with full face-piece, operated in positive pressure or pressure-demand mode.
- • Rubber gloves
- • Long-sleeve coveralls or impervious full body suit
- • Head protection, such as a hood, made of material(s) providing protection against amine catalysts
- Firefighting personnel and other on-site Emergency Responders should be fully trained in Chemical Emergency Procedures. However back-up from local authorities should be sought

### 6.4. Reference to other sections

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

## SECTION 7 HANDLING AND STORAGE

### 7.1. Precautions for safe handling

**Safe handling**

- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- **WARNING:** To avoid violent reaction, **ALWAYS** add material to water and **NEVER** water to material.

**Continued...**

| | |
|---|---|
| | ▸ Avoid smoking, naked lights or ignition sources.<br>▸ Avoid contact with incompatible materials.<br>▸ When handling, **DO NOT** eat, drink or smoke.<br>▸ Keep containers securely sealed when not in use.<br>▸ Avoid physical damage to containers.<br>▸ Always wash hands with soap and water after handling.<br>▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use.<br>▸ Use good occupational work practice.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.<br>▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.<br>▸ **DO NOT** allow clothing wet with material to stay in contact with skin |
| **Fire and explosion protection** | See section 5 |
| **Other information** | ▸ Store in original containers.<br>▸ Keep containers securely sealed.<br>▸ Store in a cool, dry, well-ventilated area.<br>▸ Store away from incompatible materials and foodstuff containers.<br>▸ Protect containers against physical damage and check regularly for leaks.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.<br>▸ **DO NOT** store near acids, or oxidising agents<br>▸ No smoking, naked lights, heat or ignition sources. |

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

| | |
|---|---|
| **Suitable container** | ▸ Lined metal can, lined metal pail/ can.<br>▸ Plastic pail.<br>▸ Polyliner drum.<br>▸ Packing as recommended by manufacturer.<br>▸ Check all containers are clearly labelled and free from leaks.<br>For low viscosity materials<br>▸ Drums and jerricans must be of the non-removable head type.<br>▸ Where a can is to be used as an inner package, the can must have a screwed enclosure.<br>For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):<br>▸ Removable head packaging;<br>▸ Cans with friction closures and<br>▸ low pressure tubes and cartridges<br>may be used.<br>-<br>Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic. |
| **Storage incompatibility** | For aluminas (aluminium oxide):<br>Incompatible with hot chlorinated rubber.<br>In the presence of chlorine trifluoride may react violently and ignite.<br>-May initiate explosive polymerisation of olefin oxides including ethylene oxide.<br>-Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.<br>-Produces exothermic reaction with oxygen difluoride.<br>-May form explosive mixture with oxygen difluoride.<br>-Forms explosive mixtures with sodium nitrate.<br>-Reacts vigorously with vinyl acetate.<br>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.<br>▸ Avoid contact with copper, aluminium and their alloys.<br>▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.<br>▸ Avoid reaction with oxidising agents |

### 7.3. Specific end use(s)

See section 1.2

## SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

### 8.1. Control parameters

**DERIVED NO EFFECT LEVEL (DNEL)**

Not Available

**PREDICTED NO EFFECT LEVEL (PNEC)**

Not Available

**OCCUPATIONAL EXPOSURE LIMITS (OEL)**

**INGREDIENT DATA**

| Source | Ingredient | Material name | TWA | STEL | Peak | Notes |
|---|---|---|---|---|---|---|
| UK Workplace Exposure Limits (WELs) | carbon black | Carbon black | 3.5 mg/m3 | 7 mg/m3 | Not Available | Not Available |

**EMERGENCY LIMITS**

| Ingredient | Material name | TEEL-1 | TEEL-2 | TEEL-3 |
|---|---|---|---|---|
| alumina hydrate | Aluminum hydroxide | 8.7 mg/m3 | 73 mg/m3 | 440 mg/m3 |

**Continued...**

| | | | | |
|---|---|---|---|---|
| polypropylene glycol bis(2-aminopropyl ether) | Polyoxyalkyleneamine; (Poly(oxypropylene)diamine) | 0.73 mg/m3 | 8 mg/m3 | 48 mg/m3 |
| aluminium oxide | Aluminum oxide; (Alumina) | 5.7 mg/m3 | 15 mg/m3 | 25 mg/m3 |
| carbon black | Carbon black | 9 mg/m3 | 99 mg/m3 | 590 mg/m3 |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| alumina hydrate | Not Available | Not Available |
| polypropylene glycol bis(2-aminopropyl ether) | Not Available | Not Available |
| ammonium polyphosphate | Not Available | Not Available |
| aluminium oxide | Not Available | Not Available |
| phenol, styrenated | Not Available | Not Available |
| zinc borate hydrate | Not Available | Not Available |
| cocoamine | Not Available | Not Available |
| trimethylhexamethylene diamine | Not Available | Not Available |
| carbon black | N.E. mg/m3 / N.E. ppm | 1,750 mg/m3 |

**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/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA.
The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For 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

## 8.2. Exposure controls

**8.2.1. Appropriate engineering controls**

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection.
Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

| Type of Contaminant: | Air Speed: |
|---|---|
| solvent, vapours, degreasing etc., evaporating from tank (in still air). | 0.25-0.5 m/s (50-100 f/min.) |
| aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation) | 0.5-1 m/s (100-200 f/min.) |
| direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 f/min.) |
| grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion). | 2.5-10 m/s (500-2000 f/min.) |

Within each range the appropriate value depends on:

| Lower end of the range | Upper end of the range |
|---|---|
| 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents |
| 2: Contaminants of low toxicity or of nuisance value only. | 2: Contaminants of high toxicity |
| 3: Intermittent, low production. | 3: High production, heavy use |
| 4: Large hood or large air mass in motion | 4: Small hood-local control only |

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

**8.2.2. Personal protection**

**Continued...**

| | |
|---|---|
| **Eye and face protection** | ▸ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.<br>▸ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted.<br>▸ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.<br>▸ Alternatively a gas mask may replace splash goggles and face shields.<br>▸ 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]<br>For amines:<br>SPECIAL PRECAUTION:<br>▸ Because amines are alkaline materials that can cause rapid and severe tissue damage, wearing of contact lenses while working with amines is strongly discouraged. Wearing such lenses can prolong contact of the eye tissue with the amine, thereby causing more severe damage.<br>▸ Appropriate eye protection should be worn whenever amines are handled or whenever there is any possibility of direct contact with liquid products, vapors, or aerosol mists.<br>CAUTION:<br>▸ Ordinary safety glasses or face-shields will not prevent eye irritation from high concentrations of vapour.<br>▸ In operations where positive-pressure, air-supplied breathing apparatus is not required, all persons handling liquid amine catalysts or other polyurethane components in open containers should wear chemical workers safety goggles.<br>▸ Eyewash fountains should be installed, and kept in good working order, wherever amines are used. |
| **Skin protection** | See Hand protection below |
| **Hands/feet protection** | ▸ Elbow length PVC gloves<br>▸ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.<br>**NOTE:**<br>▸ 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.<br>▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.<br>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.<br>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.<br>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 moisturizer is recommended.<br>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:<br>· frequency and duration of contact,<br>· chemical resistance of glove material,<br>· glove thickness and<br>· dexterity<br>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).<br>· 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.<br>· 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.<br>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.<br>· Contaminated gloves should be replaced.<br>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.<br>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.<br>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.<br>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:<br>· 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.<br>· 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<br>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.<br>For amines:<br>▸ Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly.<br>▸ Application of a non-perfumed moisturiser is recommended<br>▸ Where there is a possibility of exposure to liquid amines skin protection should include: rubber gloves, (neoprene, nitrile, or butyl).<br>▸ DO NOT USE latex. |
| **Body protection** | See Other protection below |
| **Other protection** | ▸ Overalls.<br>▸ PVC Apron.<br>▸ PVC protective suit may be required if exposure severe.<br>▸ Eyewash unit.<br>▸ Ensure there is ready access to a safety shower. |
| **Thermal hazards** | Not Available |

### Respiratory protection

**Type EK-P 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 | EK-AUS P2 | - | EK-PAPR-AUS / Class 1 P2 |

**Continued...**

| | | | |
|---|---|---|---|
| up to 50 x ES | - | EK-AUS / Class 1 P2 | - |
| up to 100 x ES | - | EK-2 P2 | EK-PAPR-2 P2 ^ |

^ - 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(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

**Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.**
Where engineering controls are not feasible and work practices do not reduce airborne amine concentrations below recommended exposure limits, appropriate respiratory protection should be used. In such cases, air-purifying respirators equipped with cartridges designed to protect against amines are recommended.
76ak-p()

### 8.2.3. Environmental exposure controls

See section 12

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### 9.1. Information on basic physical and chemical properties

| | |
|---|---|
| **Appearance** | Black |

| | | | |
|---|---|---|---|
| **Physical state** | Liquid | **Relative density (Water = 1)** | 1.62 |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | Not Available |
| **pH (as supplied)** | Not Available | **Decomposition temperature** | Not Available |
| **Melting point / freezing point (°C)** | Not Available | **Viscosity (cSt)** | 2820 |
| **Initial boiling point and boiling range (°C)** | >200 | **Molecular weight (g/mol)** | Not Available |
| **Flash point (°C)** | >124 | **Taste** | Not Available |
| **Evaporation rate** | Not Available | **Explosive properties** | Not Available |
| **Flammability** | Not Applicable | **Oxidising properties** | Not Available |
| **Upper Explosive Limit (%)** | Not Available | **Surface Tension (dyn/cm or mN/m)** | Not Available |
| **Lower Explosive Limit (%)** | Not Available | **Volatile Component (%vol)** | Not Available |
| **Vapour pressure (kPa)** | 0.1 | **Gas group** | Not Available |
| **Solubility in water (g/L)** | Immiscible | **pH as a solution (1%)** | Not Available |
| **Vapour density (Air = 1)** | Not Available | **VOC g/L** | Not Available |

### 9.2. Other information

Not Available

## SECTION 10 STABILITY AND REACTIVITY

| | |
|---|---|
| **10.1.Reactivity** | See section 7.2 |
| **10.2. Chemical stability** | ▸ Unstable in the presence of incompatible materials.<br>▸ Product is considered stable.<br>▸ Hazardous polymerisation will not occur. |
| **10.3. Possibility of hazardous reactions** | See section 7.2 |
| **10.4. Conditions to avoid** | See section 7.2 |
| **10.5. Incompatible materials** | See section 7.2 |
| **10.6. Hazardous decomposition products** | See section 5.3 |

## SECTION 11 TOXICOLOGICAL INFORMATION

### 11.1. Information on toxicological effects

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| **Inhaled** | Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.<br>Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales. |

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| | Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.<br><br>Inhalation of amine vapours may cause irritation of the mucous membranes of the nose and throat and lung irritation with respiratory distress and cough. Single exposures to near lethal concentrations and repeated exposures to sublethal concentrations produces tracheitis, bronchitis, pneumonitis and pulmonary oedema. Aliphatic and alicyclic amines are generally well absorbed from the respiratory tract. Systemic effects include headache, nausea, faintness and anxiety. These effects are thought to be transient and are probably related to the pharmacodynamic action of the amines. Histamine release by aliphatic amines may produce bronchoconstriction and wheezing.<br><br>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. |
| **Ingestion** | Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.<br>Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the<br>effects of stricture formation.<br>Acute toxic responses to aluminium are confined to the more soluble forms.<br>Inorganic polyphosphates are used extensively in domestic and industrial products. Rats fed 10% sodium trimetaphosphate for a month exhibited transient tubular necrosis;<br>those given 10% sodium metaphosphate exhibited growth retardation; 10% sodium hexametaphosphate produced pale and swollen kidneys.<br>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.<br>Aliphatic and alicyclic amines are generally well absorbed from the gut. Corrosive action may cause tissue damage throughout the gastrointestinal tract. Detoxification is thought to occur in the liver, kidney and intestinal mucosa with the enzymes, monoamine oxidase and diamine oxidase (histaminase) having a significant role. |
| **Skin Contact** | The material can produce severe chemical burns following direct contact with the skin.<br><br>Skin contact is not thought to produce harmful health effects (as classified under EC Directives using animal models). Systemic harm, however, has been identified following exposure of animals by at least one other route and the material may still produce health damage following entry through wounds, lesions or abrasions. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.<br>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.<br>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.<br>Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep.<br>Volatile amine vapours produce primary skin irritation and dermatitis. Direct local contact, with the lower molecular weight liquids, may produce skin burns. Percutaneous absorption of simple aliphatic amines is known to produce lethal effects often the same as that for oral administration. Cutaneous sensitisation has been recorded chiefly due to ethyleneamines. Histamine release following exposure to many aliphatic amines may result in 'triple response' (white vasoconstriction, red flare and wheal) in human skin.<br>Open cuts, abraded or irritated skin should not be exposed to this material<br>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** | Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight.<br>Vapours of volatile amines cause eye irritation with lachrymation, conjunctivitis and minor transient corneal oedema which results in 'halos' around lights (glaucopsia, 'blue haze', or 'blue-grey haze'). Vision may become misty and halos may appear several hours after workers are exposed to the substance<br>This effect generally disappears spontaneously within a few hours of the end of exposure, and does not produce physiological after-effects. However oedema of the corneal epithelium, which is primarily responsible for vision disturbances, may take more than one or more days to clear, depending on the severity of exposure. Photophobia and discomfort from the roughness of the corneal surface also may occur after greater exposures.<br>Although no detriment to the eye occurs as such, glaucopsia predisposes an affected individual to physical accidents and reduces the ability to undertake skilled tasks such as driving a vehicle.<br>Direct local contact with the liquid may produce eye damage which may be permanent in the case of the lower molecular weight species. |
| **Chronic** | Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.<br>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.<br>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.<br>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.<br>Harmful: danger of serious damage to health by prolonged exposure through inhalation.<br>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.<br>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.<br><br>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.<br>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. 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.<br>The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental |

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animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.
Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.
There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.
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 osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.
After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.
At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.
Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.
Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 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*]
Inhalation of epoxy resin amine hardener vapours (including polyamines and amine adducts) may produce bronchospasm and coughing episodes lasting days after cessation of the exposure. Even faint traces of these vapours may trigger an intense reaction in individuals showing 'amine asthma'. The literature records several instances of systemic intoxications following the use of amines in epoxy resin systems.
Excessive exposure to the vapours of epoxy amine curing agents may cause both respiratory irritation and central nervous system depression. Signs and symptoms of central nervous system depression, in order of increasing exposure, are
headache, dizziness, drowsiness, and incoordination. In short, a single prolonged (measured in hours) or excessive inhalation exposure may cause serious adverse effects, including death.

**834FX Black Flexible Epoxy, Thermally Conductive–Flame Retardant, Encapsulating and Potting Compound (Part B)**

| TOXICITY | IRRITATION |
|---|---|
| #55rads#51allergy#551aminepu[2] | Not Available |

**alumina hydrate**

| TOXICITY | IRRITATION |
|---|---|
| Oral (rat) LD50: >2000 mg/kg[1] | Not Available |

**polypropylene glycol bis(2-aminopropyl ether)**

| TOXICITY | IRRITATION |
|---|---|
| Dermal (rabbit) LD50: 250 mg/kg***[2] | Eye (rabbit): 100 mg - SEVERE |
| Oral (rat) LD50: 242 mg/kgE[2] | Eye (rabbit): SEVERE *** |
| | Skin (rabbit): SEVERE *** |

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| **ammonium polyphosphate** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: >3160 mg/kg*o[2] | Not Available |
| | Oral (rat) LD50: 5625 mg/kg*d[2] | |
| **aluminium oxide** | **TOXICITY** | **IRRITATION** |
| | Oral (rat) LD50: >2000 mg/kg[1] | Not Available |
| **phenol, styrenated** | **TOXICITY** | **IRRITATION** |
| | Oral (rat) LD50: 2500 mg/kg**[2] | Eye (rabbit): not irritating * |
| | | Skin (rabbit): slight * |
| **zinc borate hydrate** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |
| **cocoamine** | **TOXICITY** | **IRRITATION** |
| | Oral (rat) LD50: 1300 mg/kg][2] | Corrosive (Eye) |
| | | Corrosive (Skin) [ICI] |
| **trimethylhexamethylene diamine** | **TOXICITY** | **IRRITATION** |
| | Oral (rat) LD50: 910 mg/kg*[2] | Eye (rabbit): Corrosive * |
| | | Skin (rabbit): Corrosive * |
| **carbon black** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: >3000 mg/kg[2] | Not Available |
| | Oral (rat) LD50: >10000 mg/kg[1] | |

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| ***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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| **POLYPROPYLENE GLYCOL BIS(2-AMINOPROPYL ETHER)** | The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.<br>Convulsions, stomach ulceration, haemorrhage, respiratory tract changes, dermatitis after systemic administration recorded. * Reichard ** Bayer Inc. Canada *** Texaco ****Epoxylite |
| **PHENOL, STYRENATED** | for styrenated phenols:<br>**Acute toxicity**: Available acute oral and dermal toxicity data indicated members of this category are not acutely toxic.<br>**Repeated Dose Toxicity :** A 12-week feeding study has been conducted with styrenated phenol. In the study the thyroid was identified as a target organ and a NOAEL (50 mg/kg/day) and LOAEL (158 mg/kg/day) established.<br>**Genotoxicity.** Genotoxicity test indicate that the styrenated phenols do not have potential to cause mutations.<br>Bacterial Gene Mutation Assays. Bacterial gene mutations assays have been conducted with both substances in the category. Assays were done with and without metabolic activation and were negative.<br>Chromosome Aberration Studies. A chromosome aberration study in vivo has been conducted with isobutylenated methylstyrenated phenol and was negative. It would not be expected that styrenated phenol would give different results than<br>isobutylenated methylstyrenated phenol.<br>Other mutagenicity tests**.** An in vitro gene mutation assay with Mouse Lymphoma cells is available for isobutylenated methylstyrenated phenol and was negative. The only positive genotoxicity test was a bacterial DNA damage test with styrenated phenol.<br><br>For hindered phenols:<br>Available data shows that acute toxicity of these substances is low.<br>**Mutagenicity.** Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.<br>**In Vitro Chromosome Aberration Studies.** In vitro chromosome aberration studies are available for several members All except 2,6-di-tert-butyl-p-cresol were negative<br>**In Vivo Chromosome Aberration Studies.** In vivo studies evaluating chromosome damage are available for six of the hindered phenols. All in vivo evaluations were negative.<br>**Repeated Dose Toxicity**. Repeated dose toxicity data of approximately three months (90-day, 12- and 13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day<br>**Carcinogenicity:** Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats<br><br>**NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.<br>NOAEL 50 mg/kg * LOAEL 158 mg/kg* * IUCLID Database |

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| **COCOAMINE** | For Fatty Nitrogen-Derived ether amines and Fatty Nitrogen-derived amines (FND ether amines and FND amines):<br>FND ether amines and FND amines are very similar in structure and function. . The minimal difference among the alkyl substituents and the large database for the FND categories indicates that the structural differences in these large alkyl chains do not result in differences in toxicity or mutagenicity.<br>The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals<br>The available acute oral LD50 study for the propanamine derivative with the extensive data for the other supporting chemicals provides adequate evidence that the FND ether amines are only moderately to slightly toxic via this route and exposure period. Acute dermal studies for the supporting chemicals indicate these chemicals can be classified as minimally toxic. Acute inhalation studies did not result in deaths under normal exposure conditions for two chemicals. Repeated dose toxicity studies had similar NOAELs (12.5 to 50 mg/kg/day for rats and 3 or 13 mg/kg/day for dogs). Importantly because the highest exposure potential for some of the FND ether amines is via skin contact, a number of repeat dose dermal studies indicate the chemicals are highly irritating.<br>No clear organ-specific toxicity occurred in any of the repeat dose studies with the supporting chemicals in the FND ether amines category. In addition, available data indicate that the FND ether amines are unlikely to be mutagenic and that they are not reproductive or developmental toxins<br><br>In evaluating potential toxicity of the FND Amines chemicals, it is also useful to review the available data for the related FND Cationic and FND Amides Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (*in vitro* bacterial and mammalian cells as well as *in vivo* studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.<br>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. |
| **TRIMETHYLHEXAMETHYLENE DIAMINE** | The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. |
| **CARBON BLACK** | **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.<br>Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported |
| **ALUMINA HYDRATE & ALUMINIUM OXIDE & ZINC BORATE HYDRATE & CARBON BLACK** | No significant acute toxicological data identified in literature search. |
| **POLYPROPYLENE GLYCOL BIS(2-AMINOPROPYL ETHER) & COCOAMINE & TRIMETHYLHEXAMETHYLENE DIAMINE** | 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. |
| **COCOAMINE & TRIMETHYLHEXAMETHYLENE DIAMINE** | The following information refers to contact allergens as a group and may not be specific to this product.<br>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. |
| **COCOAMINE & TRIMETHYLHEXAMETHYLENE DIAMINE** | 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.<br>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).<br><br>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. |
| **COCOAMINE & TRIMETHYLHEXAMETHYLENE DIAMINE** | The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. |
| **COCOAMINE & TRIMETHYLHEXAMETHYLENE DIAMINE** | 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.<br>▸ 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.<br>▸ 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.<br>Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.<br>**Inhalation:**<br>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.<br>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.<br>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.<br>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 |

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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.
**Skin Contact:**
Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.
Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.
**Eye Contact:**
Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations.
Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.)
Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.
The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases.
Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.
**Ingestion:**
The oral toxicity of amine catalysts varies from moderately to very toxic.
Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus,and gastrointestinal tract.
Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.
Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.
**Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000**
**Alliance for Polyurethanes Industry**

| | | | |
|---|---|---|---|
| **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 available but does not fill the criteria for classification*
✔ *– Data available to make classification*
⊘ *– Data Not Available to make classification*

## SECTION 12 ECOLOGICAL INFORMATION

### 12.1. Toxicity

**834FX Black Flexible Epoxy, Thermally Conductive–Flame Retardant, Encapsulating and Potting Compound (Part B)**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**alumina hydrate**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 0.2262mg/L | 2 |
| EC50 | 48 | Crustacea | 0.7364mg/L | 2 |
| EC50 | 96 | Algae or other aquatic plants | 0.0054mg/L | 2 |
| NOEC | 72 | Algae or other aquatic plants | >=0.004mg/L | 2 |

**polypropylene glycol bis(2-aminopropyl ether)**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**ammonium polyphosphate**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 70mg/L | 4 |
| EC50 | 48 | Crustacea | 813mg/L | 4 |

**aluminium oxide**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 0.0029mg/L | 2 |
| EC50 | 48 | Crustacea | 0.7364mg/L | 2 |
| EC50 | 96 | Algae or other aquatic plants | 0.0054mg/L | 2 |
| NOEC | 72 | Algae or other aquatic plants | >=0.004mg/L | 2 |

**phenol, styrenated**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 1mg/L | 1 |
| EC50 | 48 | Crustacea | 4.6mg/L | 2 |

**Continued...**

| | | | | | |
|---|---|---|---|---|---|
| | EC50 | 72 | Algae or other aquatic plants | >10mg/L | 2 |
| | NOEC | 504 | Crustacea | 0.115mg/L | 2 |

**zinc borate hydrate**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**cocoamine**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | =0.1mg/L | 1 |
| EC50 | 48 | Crustacea | =0.045mg/L | 1 |
| EC50 | 96 | Algae or other aquatic plants | =0.0008mg/L | 1 |
| EC0 | 24 | Crustacea | =0.032mg/L | 1 |
| NOEC | 96 | Algae or other aquatic plants | =0.0002mg/L | 1 |

**trimethylhexamethylene diamine**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| EC50 | 72 | Algae or other aquatic plants | =29.5mg/L | 1 |
| EC10 | 72 | Algae or other aquatic plants | =16.3mg/L | 1 |

**carbon black**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | =1000mg/L | 1 |
| NOEC | 96 | Fish | =1000mg/L | 1 |

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

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
For 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(H2O)6]3+, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H2O)5(OH)]2+, [Al(H2O)4(OH)2]+). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)2+ and Al(OH)2+, while the solid Al(OH)3 is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)4- is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)3, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.
Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.
Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.
The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.
Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.
Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.
Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.
The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic.
Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.
Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.
**Ecotoxicity:**
***Freshwater species pH >6.5***
Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar)* - 106 mg/L; Chronic NOEC (8-28 d): 7 spp,NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for

**Continued...**

*Micropterus* sp.
Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L
Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L
Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L
***Freshwater species pH <6.5 (all between pH 4.5 and 6.0)***
Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L
Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)
Alga: 1 sp NOEC growth 2.0 mg/L
Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.
The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4.5.4, with a maximum toxicity occurring around pH 5.0.5.2. The inorganic single unit aluminium species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.
Drinking Water Standards:
aluminium: 200 ug/l (UK max.)
200 ug/l (WHO guideline)
chloride: 400 mg/l (UK max.)
250 mg/l (WHO guideline)
fluoride: 1.5 mg/l (UK max.)
1.5 mg/l (WHO guideline)
nitrate: 50 mg/l (UK max.)
50 mg/l (WHO guideline)
sulfate: 250 mg/l (UK max.)
Soil Guideline: none available.
Air Quality Standards: none available.

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

### 12.2. Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| phenol, styrenated | HIGH | HIGH |
| cocoamine | LOW | LOW |
| trimethylhexamethylene diamine | HIGH | HIGH |

### 12.3. Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| phenol, styrenated | LOW (LogKOW = 7.0554) |
| cocoamine | HIGH (LogKOW = 5.7458) |
| trimethylhexamethylene diamine | LOW (LogKOW = 1.6347) |

### 12.4. Mobility in soil

| Ingredient | Mobility |
|---|---|
| phenol, styrenated | LOW (KOC = 2622000) |
| cocoamine | LOW (KOC = 27640) |
| trimethylhexamethylene diamine | LOW (KOC = 1101) |

### 12.5.Results of PBT and vPvB assessment

| | P | B | T |
|---|---|---|---|
| Relevant available data | Not Available | Not Available | Not Available |
| PBT Criteria fulfilled? | Not Available | Not Available | Not Available |

### 12.6. Other adverse effects

No data available

## SECTION 13 DISPOSAL CONSIDERATIONS

### 13.1. Waste treatment methods

| | |
|---|---|
| **Product / Packaging disposal** | ▸ Containers may still present a chemical hazard/ danger when empty.<br>▸ Return to supplier for reuse/ recycling if possible.<br>Otherwise: |

**Continued...**

| | |
|---|---|
| | ▸ 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.<br>▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product.<br>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.<br>A Hierarchy of Controls seems to be common - the user should investigate:<br>▸ Reduction<br>▸ Reuse<br>▸ Recycling<br>▸ Disposal (if all else fails)<br>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.<br>Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.<br>▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains.<br>▸ It may be necessary to collect all wash water for treatment before disposal.<br>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.<br>▸ Where in doubt contact the responsible authority.<br>▸ Recycle wherever possible.<br>▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.<br>▸ Treat and neutralise at an approved treatment plant.<br>▸ Treatment should involve: Neutralisation with suitable dilute acid followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material).<br>▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed. |
| **Waste treatment options** | Not Available |
| **Sewage disposal options** | Not Available |

## SECTION 14 TRANSPORT INFORMATION

### Labels Required

Limited Quantity: 834FX-450ML, 834FX-1.7L kits

### Land transport (ADR)

| | |
|---|---|
| **14.1.UN number** | 2735 |
| **14.2.UN proper shipping name** | AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains polypropylene glycol bis(2-aminopropyl ether), trimethylhexamethylene diamine and cocoamine) |
| **14.3. Transport hazard class(es)** | Class: 8<br>Subrisk: Not Applicable |
| **14.4.Packing group** | II |
| **14.5.Environmental hazard** | Environmentally hazardous |
| **14.6. Special precautions for user** | Hazard identification (Kemler): 80<br>Classification code: C7<br>Hazard Label: 8<br>Special provisions: 274<br>Limited quantity: 1 L |

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

| | |
|---|---|
| **14.1. UN number** | 2735 |
| **14.2. UN proper shipping name** | Amines, liquid, corrosive, n.o.s. * (contains polypropylene glycol bis(2-aminopropyl ether), trimethylhexamethylene diamine and cocoamine); Polyamines, liquid, corrosive, n.o.s. * (contains polypropylene glycol bis(2-aminopropyl ether), trimethylhexamethylene diamine and cocoamine) |
| **14.3. Transport hazard class(es)** | ICAO/IATA Class: 8<br>ICAO / IATA Subrisk: Not Applicable<br>ERG Code: 8L |
| **14.4. Packing group** | II |
| **14.5. Environmental hazard** | Environmentally hazardous |

**Continued...**

| | |
|---|---|
| **14.6. Special precautions for user** | Special provisions: A3 A803<br>Cargo Only Packing Instructions: 855<br>Cargo Only Maximum Qty / Pack: 30 L<br>Passenger and Cargo Packing Instructions: 851<br>Passenger and Cargo Maximum Qty / Pack: 1 L<br>Passenger and Cargo Limited Quantity Packing Instructions: Y840<br>Passenger and Cargo Limited Maximum Qty / Pack: 0.5 L |

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

| | |
|---|---|
| **14.1. UN number** | 2735 |
| **14.2. UN proper shipping name** | AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains polypropylene glycol bis(2-aminopropyl ether), trimethylhexamethylene diamine and cocoamine) |
| **14.3. Transport hazard class(es)** | IMDG Class: 8<br>IMDG Subrisk: Not Applicable |
| **14.4. Packing group** | II |
| **14.5. Environmental hazard** | Marine Pollutant |
| **14.6. Special precautions for user** | EMS Number: F-A , S-B<br>Special provisions: 274<br>Limited Quantities: 1 L |

### Inland waterways transport (ADN)

| | |
|---|---|
| **14.1. UN number** | 2735 |
| **14.2. UN proper shipping name** | AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains polypropylene glycol bis(2-aminopropyl ether), trimethylhexamethylene diamine and cocoamine) |
| **14.3. Transport hazard class(es)** | 8 Not Applicable |
| **14.4. Packing group** | II |
| **14.5. Environmental hazard** | Environmentally hazardous |
| **14.6. Special precautions for user** | Classification code: C7<br>Special provisions: 274<br>Limited quantity: 1 L<br>Equipment required: PP, EP<br>Fire cones number: 0 |

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

Not Applicable

## SECTION 15 REGULATORY INFORMATION

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

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

European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**POLYPROPYLENE GLYCOL BIS(2-AMINOPROPYL ETHER)(9046-10-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Not Applicable

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

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

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

European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
UK Workplace Exposure Limits (WELs)

**PHENOL, STYRENATED(61788-44-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

**Continued...**

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Customs Inventory of Chemical Substances ECICS (English)

**ZINC BORATE HYDRATE(138265-88-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Not Applicable

**COCOAMINE(61788-46-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

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

**TRIMETHYLHEXAMETHYLENE DIAMINE(25620-58-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

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

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

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Customs Inventory of Chemical Substances ECICS (English)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
European List of Notified Chemical Substances (ELINCS)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
UK Workplace Exposure Limits (WELs)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments

### 15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

**ECHA SUMMARY**

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| alumina hydrate | 21645-51-2 | Not Available | 01-2119529246-39-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | Skin Irrit. 2, Eye Irrit. 2, STOT SE 3, Aquatic Acute 1, Aquatic Chronic 1 | GHS07, Wng, GHS09 | H315, H319, H335, H410 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| polypropylene glycol bis(2-aminopropyl ether) | 9046-10-0 | Not Available | 01-2119557899-12-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Acute Tox. 4, Asp. Tox. 1, Acute Tox. 3, Skin Corr. 1C, Eye Dam. 1, Aquatic Chronic 3 | GHS05, GHS06, Dgr | H302, H304, H311, H314, H412 |
| 2 | Skin Corr. 1C, Eye Dam. 1, Aquatic Chronic 3, Acute Tox. 4, Asp. Tox. 1, Acute Tox. 3, Skin Corr. 1B, STOT SE 3, Aquatic Chronic 2, Skin Sens. 1, Met. Corr. 1, Skin Irrit. 2, Eye Irrit. 2 | GHS05, Dgr, GHS06, GHS09, GHS08 | H314, H304, H311, H335, H318, H301, H411, H317, H332, H290 |
| 1 | Skin Corr. 1B | GHS05, Dgr | H314 |
| 2 | Skin Corr. 1B | GHS05, Dgr | H314 |
| 1 | Skin Corr. 1B | GHS05, Dgr | H314 |
| 2 | Skin Corr. 1B, Asp. Tox. 1, Skin Corr. 1C, Eye Dam. 1, Aquatic Chronic 3, Aquatic Chronic 2, Skin Irrit. 2, STOT SE 3, Acute Tox. 4 | GHS05, Dgr, GHS08, GHS09 | H314, H304, H318, H411, H335, H302, H312 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| ammonium polyphosphate | 68333-79-9 | Not Available | Not Available |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | Acute Tox. 4, Eye Irrit. 2 | GHS07, Wng | H302, H319 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| aluminium oxide | 1344-28-1. | Not Available | 01-2119529248-35-XXXX, 01-2119817795-27-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | STOT SE 3, STOT RE 1, Skin Sens. 1, Muta. 2, Carc. 1B, Repr. 2, Aquatic Chronic 3, Acute Tox. 4, Skin Irrit. 2, Eye Irrit. 2, STOT RE 2, Flam. Liq. 2 | GHS08, Dgr, GHS09, GHS02 | H370, H335, H372, H317, H341, H350, H361, H412, H302, H332, H315, H319, H220, H225 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| phenol, styrenated | 61788-44-1 | Not Available | 01-2119557886-19-XXXX, 01-2119979575-18-XXXX, 01-2119980970-27-XXXX |

**Continued...**

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Aquatic Chronic 2 | GHS09 | H411 |
| 2 | Aquatic Chronic 2, Aquatic Chronic 4, Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 1 | GHS09, Wng, GHS06 | H315, H317, H319, H410 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| zinc borate hydrate | 138265-88-0 | Not Available | Not Available |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | Aquatic Acute 1, Aquatic Chronic 1, Eye Irrit. 2, Repr. 2, Aquatic Chronic 2 | GHS09, Wng, GHS08 | H400, H410, H319, H361fd |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| cocoamine | 61788-46-3 | 612-285-00-4 | 01-2119971069-29-XXXX, 01-2119473798-17-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Acute Tox. 4, Asp. Tox. 1, Skin Corr. 1B, STOT SE 3, STOT RE 2, Aquatic Acute 1, Aquatic Chronic 1 | GHS09, GHS08, GHS05, Dgr | H302, H304, H314, H335, H373, H410 |
| 2 | Acute Tox. 4, Asp. Tox. 1, Skin Corr. 1B, STOT SE 3, STOT RE 2, Aquatic Acute 1, Aquatic Chronic 1 | GHS09, GHS08, GHS05, Dgr | H302, H304, H314, H335, H373, H410, H400 |
| 1 | Acute Tox. 4, Skin Corr. 1A, Aquatic Acute 1 | GHS09, GHS05, Dgr | H302, H314, H400 |
| 2 | Acute Tox. 4, Asp. Tox. 1, Skin Corr. 1A, Eye Dam. 1, STOT SE 3, STOT RE 2, Aquatic Acute 1, Aquatic Chronic 1, Skin Irrit. 2, Eye Irrit. 2, Skin Corr. 1C | GHS09, GHS08, GHS05, Dgr | H302, H304, H314, H318, H335, H373, H400, H410 |
| 1 | Acute Tox. 4, Skin Corr. 1B, Eye Dam. 1, STOT SE 3, STOT RE 2, Aquatic Acute 1 | GHS09, GHS08, GHS05, Dgr | H302, H314, H318, H335, H373, H400 |
| 2 | Acute Tox. 4, Skin Corr. 1B, Eye Dam. 1, STOT SE 3, STOT RE 2, Aquatic Acute 1, Aquatic Chronic 1, Asp. Tox. 1, Met. Corr. 1, Skin Corr. 1A, Aquatic Chronic 2, Eye Irrit. 2 | GHS09, GHS08, GHS05, Dgr | H302, H314, H318, H335, H373, H400, H410, H304, H290, H313 |
| 1 | Acute Tox. 4, Asp. Tox. 1, Skin Corr. 1B, STOT SE 3, STOT RE 2, Aquatic Acute 1, Aquatic Chronic 1 | GHS09, GHS08, GHS05, Dgr | H302, H304, H314, H335, H373, H410 |
| 2 | Acute Tox. 4, Asp. Tox. 1, Skin Corr. 1B, STOT SE 3, STOT RE 2, Aquatic Acute 1, Aquatic Chronic 1, Skin Corr. 1A, Eye Dam. 1 | GHS09, GHS08, GHS05, Dgr | H302, H304, H314, H335, H373, H410, H318, H400 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| trimethylhexamethylene diamine | 25620-58-0 | Not Available | 01-2119560598-25-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Acute Tox. 4, Skin Corr. 1A, Skin Sens. 1A, Aquatic Chronic 3 | GHS05, Dgr | H302, H314, H317, H412 |
| 2 | Acute Tox. 4, Skin Corr. 1A, Skin Sens. 1A, Aquatic Chronic 3, Skin Corr. 1B, Skin Sens. 1, Eye Dam. 1, Skin Corr. 1C | GHS05, Dgr | H302, H314, H317, H412, H318 |
| 1 | Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3 | GHS05, Dgr | H302, H314, H317, H412 |
| 2 | Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3, Skin Corr. 1A, Skin Sens. 1A, Eye Dam. 1, Skin Corr. 1C, Aquatic Chronic 1, STOT SE 3 | GHS05, Dgr, GHS08, GHS09 | H302, H314, H317, H318, H410, H312, H335 |
| 1 | Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3 | GHS05, Dgr | H302, H314, H317, H412 |
| 2 | Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3 | GHS05, Dgr | H302, H314, H317, H412 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| carbon black | 1333-86-4 | Not Available | 01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 2 | Carc. 2, STOT SE 3, Eye Irrit. 2, STOT RE 2, STOT RE 1, Aquatic Chronic 4, Self-heat. 1, Self-heat. 2, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Flam. Sol. 2, Acute Tox. 4 | GHS08, Dgr, GHS06, GHS02, GHS09 | H351, H335, H319, H372, H251, H228, H315, H370, H410, H332 |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| National Inventory | Status |
|---|---|
| Australia - AICS | Y |
| Canada - DSL | N (zinc borate hydrate) |
| Canada - NDSL | N (phenol, styrenated; polypropylene glycol bis(2-aminopropyl ether); zinc borate hydrate; aluminium oxide; trimethylhexamethylene diamine; alumina hydrate; carbon black; ammonium polyphosphate) |
| China - IECSC | Y |

**Continued...**

| | |
|---|---|
| Europe - EINEC / ELINCS / NLP | N (polypropylene glycol bis(2-aminopropyl ether); zinc borate hydrate) |
| Japan - ENCS | N (phenol, styrenated; zinc borate hydrate; aluminium oxide) |
| Korea - KECI | N (zinc borate hydrate) |
| New Zealand - NZIoC | Y |
| Philippines - PICCS | N (zinc borate hydrate) |
| USA - TSCA | N (zinc borate hydrate) |
| ***Legend:*** | *Y = All ingredients are on the inventory*<br>*N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)* |

## SECTION 16 OTHER INFORMATION

### Full text Risk and Hazard codes

| | |
|---|---|
| **H220** | Extremely flammable gas. |
| **H225** | Highly flammable liquid and vapour. |
| **H228** | Flammable solid. |
| **H251** | Self-heating: may catch fire. |
| **H290** | May be corrosive to metals. |
| **H301** | Toxic if swallowed. |
| **H304** | May be fatal if swallowed and enters airways. |
| **H311** | Toxic in contact with skin. |
| **H312** | Harmful in contact with skin. |
| **H313** | May be harmful in contact with skin |
| **H315** | Causes skin irritation. |
| **H318** | Causes serious eye damage. |
| **H319** | Causes serious eye irritation. |
| **H332** | Harmful if inhaled. |
| **H335** | May cause respiratory irritation. |
| **H341** | Suspected of causing genetic defects. |
| **H350** | May cause cancer. |
| **H351** | Suspected of causing cancer. |
| **H360** | May damage fertility or the unborn child. |
| **H361fd** | Suspected of damaging fertility. Suspected of damaging the unborn child. |
| **H370** | Causes damage to organs. |
| **H372** | Causes damage to organs through prolonged or repeated exposure. |
| **H400** | Very toxic to aquatic life. |
| **H411** | Toxic to aquatic life with long lasting effects. |
| **H412** | Harmful to aquatic life with long lasting effects. |
| **H413** | May cause long lasting harmful effects to aquatic life. |

### Other information

### Ingredients with multiple cas numbers

| Name | CAS No |
|---|---|
| alumina hydrate | 14762-49-3, 21645-51-2 |
| phenol, styrenated | 61788-44-1, 9010-16-6 |
| cocoamine | 61788-46-3, 2016-42-4, 68155-27-1, 130169-56-1 |
| trimethylhexamethylene diamine | 25620-58-0, 25513-64-8, 3236-53-1, 105759-40-8, 112360-55-1, 125146-87-4, 130014-36-7, 161075-53-2, 172084-55-8, 178861-94-4, 72258-26-5, 76582-77-9, 87748-70-7, 93365-28-7, 3236-54-2 |

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

**Continued...**

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