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

MG Chemicals UK Limited

Version No:**A-2.00**
Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: **22/07/2021**
Revision Date: **22/07/2021**
L.REACH.GB.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-7.4ML | UFI:C3J0-M005-600K-12E8
Other means of identification	Black Flexible Epoxy, Thermally Conductive–Flame Retardant, Encapsulating and Potting Compound (Part B)

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

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H373 - Specific target organ toxicity - repeated exposure Category 2, H400 - Acute Aquatic Hazard Category 1, H302 - Acute Toxicity (Oral) Category 4, H361 - Reproductive Toxicity Category 2, H317 - Skin Sensitizer Category 1, H410 - Chronic Aquatic Hazard Category 1, H314 - Skin Corrosion/Irritation Category 1A
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567*

2.2. Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H373	May cause damage to organs through prolonged or repeated exposure.
H302	Harmful if swallowed.
H361	Suspected of damaging fertility or the unborn child.
H317	May cause an allergic skin reaction.
H410	Very toxic to aquatic life with long lasting effects.
H314	Causes severe skin burns and eye damage.

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Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P280	Wear protective gloves, protective clothing, eye protection and 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 [or 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 to authorised hazardous or special waste collection point in accordance with any local regulation.

2.3. Other hazards

Inhalation may produce health damage*.

Cumulative effects may result following exposure*.

Limited evidence of a carcinogenic effect*.

Possible respiratory sensitizer*.

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

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
1.21645-51-2 2.244-492-7 3.Not Available 4.Not Available	26	alumina hydrate	EUH210 [1]	Not Available
1.9046-10-0 2.Not Available 3.Not Available 4.Not Available	19	bis(2-aminopropyl ether) propoxylated	Corrosive to Metals Category 1, Acute Toxicity (Oral and Dermal) Category 4, Skin Corrosion/Irritation Category 1A, Serious Eye Damage/Eye Irritation Category 1, Chronic Aquatic Hazard Category 3; H290, H302+H312, H314, H318, H412 [1]	Not Available
1.68333-79-9 2.269-789-9 3.Not Available 4.Not Available	19	ammonium polyphosphate	Chronic Aquatic Hazard Category 4; H413 [1]	Not Available
1.1344-28-1. 2.215-691-6 3.Not Available	16	aluminium oxide	EUH210 [1]	Not Available

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1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
4.Not Available				
1.61788-44-1 2.262-975-0 3.Not Available 4.Not Available	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]	Not Available
1.12767-90-7 2.235-804-2 3.Not Available 4.Not Available	5	zinc borate	Eye Irritation Category 2, Reproductive Toxicity Category 1B, Chronic Aquatic Hazard Category 1; H319, H360, H410 [1]	Not Available
1.61788-46-3 2.262-977-1 3.612-285-00-4 4.Not Available	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, Skin Corrosion/Irritation Category 1B, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H302, H304, H335, H373, H314, H400, H410 [2]	Not Available
1.25620-58-0 2.247-134-8 3.Not Available 4.Not Available	3	trimethylhexamethylene diamine	Corrosive to Metals Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1B, Serious Eye Damage/Eye Irritation Category 1, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 3; H290, H302, H314, H318, H317, H412 [1]	Not Available
1.1333-86-4 2.215-609-9|435-640-3|422-130-0 3.Not Available 4.Not Available	0.5	carbon black	Carcinogenicity Category 2; H351 [1]	Not Available
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties*			

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. For amines: ▸ 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. ▸ 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. ▸ Seek immediate medical attention, preferably from an ophthalmologist.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor. For amines: ▸ In case of major exposure to liquid amine, promptly remove any contaminated clothing, including rings, watches, and shoe, preferably under a safety shower. ▸ Wash skin for 15 to 30 minutes with plenty of water and soap. Call a physician immediately. ▸ 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. ▸ Inform individuals responsible for cleaning of potential hazards associated with handling contaminated clothing. ▸ Discard contaminated leather articles such as shoes, belts, and watchbands. ▸ 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. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay. ▸ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. ▸ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs). ▸ 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. ▸ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered. **This must definitely be left to a doctor or person authorised by him/her.** (ICSC13719) For amines: ▸ All employees working in areas where contact with amine catalysts is possible should be thoroughly trained in the administration of appropriate first aid procedures. ▸ Experience has demonstrated that prompt administration of such aid can minimize the effects of accidental exposure. ▸ Promptly move the affected person away from the contaminated area to an area of fresh air.

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	‣ Keep the affected person calm and warm, but not hot. ‣ If breathing is difficult, oxygen may be administered by a qualified person. ‣ If breathing stops, give artificial respiration. Call a physician at once.
Ingestion	‣ For advice, contact a Poisons Information Centre or a doctor at once. ‣ Urgent hospital treatment is likely to be needed. ‣ If swallowed do **NOT** induce vomiting. ‣ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ‣ Observe the patient carefully. ‣ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ‣ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ‣ Transport to hospital or doctor without delay. For amines: ‣ If liquid amine are ingested, have the affected person drink several glasses of water or milk. ‣ Do not induce vomiting. ‣ 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

for phosphate salts intoxication:

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

Treat symptomatically.

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

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

[Ellenhorn and Barceloux: Medical Toxicology]
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

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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.
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. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Use fire fighting procedures suitable for surrounding area. ‣ **Do not** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use. For amines: ‣ 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. ‣ Airline and air purifying respirators should not be worn for firefighting or other emergency or upset conditions. ‣ 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. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) nitrogen oxides (NOx) phosphorus oxides (POx) metal oxides other pyrolysis products typical of burning organic material. When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles. May emit 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.

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- Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.
- Check regularly for spills and leaks.
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact with the substance, by using protective equipment.
- Contain and absorb spill with sand, earth, inert material or vermiculite.
- Wipe up.
- Place in a suitable, labelled container for waste disposal.

for amines:

- If possible (i.e., without risk of contact or exposure), stop the leak.
- Contain the spilled material by diking, then neutralize.
- Next, absorb the neutralized product with clay, sawdust, vermiculite, or other inert absorbent and shovel into containers.
- Store the containers outdoors.
- Brooms and mops should be disposed of, along with any remaining absorbent, in accordance with all applicable federal, state, and local regulations and requirements.
- 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
- Dispose of the material in full accordance with all federal, state, and local laws and regulations governing the disposal of chemical wastes.
- 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.
Chemical Class: amines, alkyl
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
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT,
treated wood fibre - pillow	3	throw	pitchfork	DGC, RT
foamed glass - pillow	4	throw	pitchfork	R, P, DGC, RT
LAND SPILL - MEDIUM				
cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	4	throw	skiploader	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

NOTE:

- Organic absorbents have been known to ignite when contaminated with amines in closed containers. Certain cellulosic materials used for spill cleanup such as wood chips or sawdust have shown reactivity with ethyleneamines and should be avoided.
- 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.

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	▸ 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. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin
Fire and explosion protection	See section 5
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ **DO NOT** store near acids, or oxidising agents ▸ 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. ▸ Plastic pail. ▸ Polyliner drum. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks. For low viscosity materials ▸ Drums and jerricans must be of the non-removable head type. ▸ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ▸ Removable head packaging; ▸ Cans with friction closures and ▸ low pressure tubes and cartridges may be used. - 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): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate. 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. ▸ Avoid contact with copper, aluminium and their alloys. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ▸ Avoid reaction with oxidising agents

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7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
alumina hydrate	Inhalation 10.76 mg/m³ (Systemic, Chronic) Inhalation 10.76 mg/m³ (Local, Chronic) *Oral 4.74 mg/kg bw/day (Systemic, Chronic)* *	Not Available
bis(2-aminopropyl ether) propoxylated	Dermal 2.5 mg/kg bw/day (Systemic, Chronic) Inhalation 1.36 mg/m³ (Systemic, Chronic)	0.015 mg/L (Water (Fresh)) 0.014 mg/L (Water - Intermittent release) 0.15 mg/L (Water (Marine)) 0.132 mg/kg sediment dw (Sediment (Fresh Water)) 0.125 mg/kg sediment dw (Sediment (Marine)) 0.018 mg/kg soil dw (Soil) 7.5 mg/L (STP) 6.93 mg/kg food (Oral)
ammonium polyphosphate	Inhalation 18.06 mg/m³ (Systemic, Chronic) *Inhalation 4.45 mg/m³ (Systemic, Chronic)* * *Oral 1.28 mg/kg bw/day (Systemic, Chronic)* *	Not Available
aluminium oxide	Dermal 0.84 mg/kg bw/day (Systemic, Chronic) Inhalation 3 mg/m³ (Systemic, Chronic) Inhalation 3 mg/m³ (Local, Chronic) *Dermal 0.3 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 0.75 mg/m³ (Systemic, Chronic)* * *Oral 1.32 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 0.75 mg/m³ (Local, Chronic)* *	74.9 µg/L (Water (Fresh)) 20 mg/L (STP)
phenol, styrenated	Dermal 21 mg/kg bw/day (Systemic, Chronic) Inhalation 74 mg/m³ (Systemic, Chronic) *Dermal 7.5 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 13.1 mg/m³ (Systemic, Chronic)* * *Oral 7.5 mg/kg bw/day (Systemic, Chronic)* *	30 µg/L (Water (Fresh)) 3 µg/L (Water - Intermittent release) 46 µg/L (Water (Marine)) 1.86 mg/kg sediment dw (Sediment (Fresh Water)) 0.186 mg/kg sediment dw (Sediment (Marine)) 0.355 mg/kg soil dw (Soil) 36.2 mg/L (STP)
zinc borate	Dermal 1 585 mg/kg bw/day (Systemic, Chronic) Inhalation 22.4 mg/m³ (Systemic, Chronic) *Dermal 1 205 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 8.3 mg/m³ (Systemic, Chronic)* * *Oral 2.4 mg/kg bw/day (Systemic, Chronic)* *	2.9 mg/L (Water (Fresh)) 2.9 mg/L (Water - Intermittent release) 13.7 mg/L (Water (Marine)) 117.8 mg/kg sediment dw (Sediment (Fresh Water)) 56.5 mg/kg sediment dw (Sediment (Marine)) 5.7 mg/kg soil dw (Soil) 10 mg/L (STP)
trimethylhexamethylene diamine	*Oral 0.05 mg/kg bw/day (Systemic, Chronic)* *	0.102 mg/L (Water (Fresh)) 0.01 mg/L (Water - Intermittent release) 0.315 mg/L (Water (Marine)) 0.622 mg/kg sediment dw (Sediment (Fresh Water)) 0.062 mg/kg sediment dw (Sediment (Marine)) 10 mg/kg soil dw (Soil) 72 mg/L (STP)
carbon black	Inhalation 1 mg/m³ (Systemic, Chronic) Inhalation 0.5 mg/m³ (Local, Chronic) *Inhalation 0.06 mg/m³ (Systemic, Chronic)* *	1 mg/L (Water (Fresh)) 0.1 mg/L (Water - Intermittent release) 10 mg/L (Water (Marine))

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: respirable dust	4 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: inhalable dust	10 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m3	7 mg/m3	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
alumina hydrate	8.7 mg/m3	73 mg/m3	440 mg/m3
bis(2-aminopropyl ether) propoxylated	4.8 mg/m3	53 mg/m3	320 mg/m3
aluminium oxide	15 mg/m3	170 mg/m3	990 mg/m3
carbon black	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH

Continued...

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

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
bis(2-aminopropyl ether) propoxylated	C	> 1 to ≤ 10 parts per million (ppm)
phenol, styrenated	E	≤ 0.1 ppm
zinc borate	E	≤ 0.01 mg/m³
cocoamine	E	≤ 0.1 ppm
trimethylhexamethylene diamine	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

For aluminium oxide and pyrophoric grades of aluminium:
Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/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,

Continued...

	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 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. ‣ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ‣ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ‣ Alternatively a gas mask may replace splash goggles and face shields. ‣ 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] For amines: SPECIAL PRECAUTION: ‣ 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. ‣ 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. CAUTION: ‣ Ordinary safety glasses or face-shields will not prevent eye irritation from high concentrations of vapour. ‣ 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. ‣ 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 ‣ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. **NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. For amines: ‣ 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 ‣ Where there is a possibility of exposure to liquid amines skin protection should include: rubber gloves, (neoprene, nitrile, or butyl). ‣ DO NOT USE latex.

Continued...

Body protection	See Other protection below
Other protection	‣ Overalls. ‣ PVC Apron. ‣ PVC protective suit may be required if exposure severe. ‣ Eyewash unit. ‣ Ensure there is ready access to a safety shower.

Respiratory protection

Type AK-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	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-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.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

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.

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 BuAC = 1	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	0.1	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Nanoform Solubility	Not Available	**Nanoform Particle Characteristics**	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

Continued...

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	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. 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. 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. 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. 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	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 effects of stricture formation. The material is not thought to produce adverse health effects following ingestion (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. 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. Ingestion of amine epoxy-curing agents (hardeners) may cause severe abdominal pain, nausea, vomiting or diarrhoea. The vomitus may contain blood and mucous. If death does not occur within 24 hours there may be an improvement in the patients condition for 2-4 days only to be followed by the sudden onset of abdominal pain, board-like abdominal rigidity or hypo-tension; this indicates that delayed gastric or oesophageal corrosive damage has occurred. Inorganic polyphosphates are used extensively in domestic and industrial products. Rats fed 10% sodium trimetaphosphate for a month exhibited transient tubular necrosis; those given 10% sodium metaphosphate exhibited growth retardation; 10% sodium hexametaphosphate produced pale and swollen kidneys. Salts of this type appear to be hydrolysed in the bowel to produce phosphoric acid and systemic acidosis may result following absorption. Higher molecular weight species, absorbed from the alimentary canal, may produce hypocalcaemic tetany due to binding of ionised calcium by the absorbed phosphate. This is reported in at least one case following ingestion of sodium tripolyphosphate. Acute toxic responses to aluminium are confined to the more soluble forms.

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Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material can produce severe chemical burns following direct contact with the skin. Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur. Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions. Individuals exhibiting 'amine dermatitis' may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis. NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided. 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. 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. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. 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. 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 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. 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. Direct local contact with the liquid may produce eye damage which may be permanent in the case of the lower molecular weight species.
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. 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. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production. Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure. When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity. Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction. There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.

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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*]
In chronic animal studies inorganic polyphosphates produced growth inhibition, increased kidney weights (with calcium deposition and desquamation), bone decalcification, parathyroid hypertrophy and hyperplasia, inorganic phosphaturia, hepatic focal necrosis and alterations to the size of muscle fibres.
Inorganic phosphates are not genotoxic in bacterial systems nor are they carcinogenic in rats. No reproductive or developmental toxicity was seen in studies using rats exposed to sodium hexametaphosphate or sodium trimetaphosphate.

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

TOXICITY	IRRITATION
Not Available	Not Available

alumina hydrate

TOXICITY	IRRITATION
Inhalation(Rat) LC50; >2.3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
Oral(Rat) LD50; >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

bis(2-aminopropyl ether) propoxylated

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 2979.7 mg/kg[1]	Eye (rabbit): 100 mg - SEVERE
Oral(Rat) LD50; 2627.2 mg/kg[1]	Eye (rabbit): SEVERE ***
	Eye: adverse effect observed (irreversible damage)[1]
	Skin (rabbit): SEVERE ***
	Skin: adverse effect observed (corrosive)[1]

ammonium polyphosphate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >3160 mg/kg[2]	Not Available

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	Inhalation(Rat) LC50; >4.85 mg/l4h[1]
	Oral(Rat) LD50; >=300<=2000 mg/kg[1]

aluminium oxide	
TOXICITY	**IRRITATION**
Inhalation(Rat) LC50; >2.3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
Oral(Rat) LD50; >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

phenol, styrenated	
TOXICITY	**IRRITATION**
dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): not irritating *
Inhalation(Rat) LC50; >4.92 mg/l4h[1]	Skin (rabbit): slight *
Oral(Rat) LD50; >2000 mg/kg[1]	

zinc borate	
TOXICITY	**IRRITATION**
Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye (rabbit): mild *
Inhalation(Rat) LC50; 4.95 mg/l4h[1]	Eye: adverse effect observed (irritating)[1]
Oral(Rat) LD50; >5000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
	Skin: non-irritant *

cocoamine	
TOXICITY	**IRRITATION**
dermal (rat) LD50: >2000 mg/kg[2]	Corrosive (Eye)
Oral(Rat) LD50; >2000 mg/kg[2]	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 (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Oral(Rat) LD50; >8000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[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*

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

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

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	effects in humans at lower exposures are inconsistent Reproductive and developmental toxicity: Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI. Genotoxicity Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels. Carcinogenicity. The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons. Neurodegenerative diseases. Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease.” There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases.Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment. Contact sensitivity: It has been suggested that the body burden of aluminium may be linked to different iseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome. Aluminium acts not only as an adjuvant,stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitisers causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response.Once inside the skin, the metal ions must bind to proteins to become immunologically reactive.The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminium Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste
BIS(2-AMINOPROPYL ETHER) PROPOXYLATED	Convulsions, stomach ulceration, haemorrhage, respiratory tract changes, dermatitis after systemic administration recorded. * Reichard ** Bayer Inc. Canada *** Texaco ****Epoxylite Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69 Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

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	PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology http://doi.org/10.5487/TR.2015.31.2.105 The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
PHENOL, STYRENATED	for styrenated phenols: **Acute toxicity**: Available acute oral and dermal toxicity data indicated members of this category are not acutely toxic. **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. **Genotoxicity.** Genotoxicity test indicate that the styrenated phenols do not have potential to cause mutations. 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. 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 isobutylenated methylstyrenated phenol. 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. For hindered phenols: Available data shows that acute toxicity of these substances is low. **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. **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 **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. **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 **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 **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. NOAEL 50 mg/kg * LOAEL 158 mg/kg* * IUCLID Database
COCOAMINE	For Fatty Nitrogen-Derived ether amines and Fatty Nitrogen-derived amines (FND ether amines and FND amines): 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. 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 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. 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 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. 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	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
834FX-B Black Flexible Epoxy, Thermally Conductive–Flame Retardant, Encapsulating and	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

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Potting Compound (Part B) & BIS(2-AMINOPROPYL ETHER) PROPOXYLATED & COCOAMINE & TRIMETHYLHEXAMETHYLENE DIAMINE	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. 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. ▸ 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. ▸ 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. Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion. **Inhalation:** Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies. While most polyurethane amine catalysts are not sensitisers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease. Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema. **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**
834FX-B Black Flexible Epoxy, Thermally Conductive–Flame Retardant, Encapsulating and Potting Compound (Part B) & COCOAMINE & TRIMETHYLHEXAMETHYLENE DIAMINE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
ALUMINA HYDRATE & ALUMINIUM OXIDE & CARBON BLACK	No significant acute toxicological data identified in literature search.
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. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence). The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Acute Toxicity	✔	**Carcinogenicity**	✘
Skin Irritation/Corrosion	✔	**Reproductivity**	✔

Continued...

Serious Eye Damage/Irritation	✗	STOT - Single Exposure	✗
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	✓
Mutagenicity	✗	Aspiration Hazard	✗

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

11.2.1. Endocrine Disruption Properties

Not Available

SECTION 12 Ecological information

12.1. Toxicity

834FX-B 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
NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
LC50	96h	Fish	0.57mg/l	2
EC50	48h	Crustacea	>0.065mg/l	4
EC50	96h	Algae or other aquatic plants	0.46mg/l	2

bis(2-aminopropyl ether) propoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	0.32mg/l	2
EC50	72h	Algae or other aquatic plants	2.1mg/l	2
LC50	96h	Fish	772.14mg/l	2
EC50	48h	Crustacea	80mg/l	2

ammonium polyphosphate

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

aluminium oxide

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

phenol, styrenated

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	0.115mg/l	2
EC50	72h	Algae or other aquatic plants	1.35mg/l	2
LC50	96h	Fish	1mg/l	1
EC50	48h	Crustacea	>=0.58<=5.25mg/l	2

zinc borate

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

cocoamine

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	96h	Algae or other aquatic plants	<0.001mg/l	1
LC50	96h	Fish	0.1mg/l	1

Continued...

	EC50	48h	Crustacea	0.045mg/l	1
	EC50	96h	Algae or other aquatic plants	0.001mg/l	1

trimethylhexamethylene diamine					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	29.5mg/l	1
	EC10(ECx)	72h	Algae or other aquatic plants	16.3mg/l	1

carbon black					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	48h	Crustacea	33.076-41.968mg/l	4
	NOEC(ECx)	24h	Crustacea	3200mg/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.
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.
In air ammonia is persistent whilst, in water, it biodegrades rapidly to nitrate, producing a high oxygen demand. Ammonia is strongly adsorbed to soil. Ammonia is non-persistent in water (half-life 2 days) and is moderately toxic to fish under normal temperature and pH conditions. Ammonia is harmful to aquatic life at low concentrations but does not concentrate in the food chain. Ammonium ions may be toxic to fish at 0.3 mg/l
Drinking Water Standards:
0.5 mg/l (UK max.)
1.5 mg/l (WHO Levels)
Soil Guidelines: none available.
Air Quality Standards: none available.

The principal problems of phosphate contamination of the environment relates to eutrophication processes in lakes and ponds. Phosphorus is an essential plant nutrient and is usually the limiting nutrient for blue-green algae. A lake undergoing eutrophication shows a rapid growth of algae in surface waters. Planktonic algae cause turbidity and flotation films. Shore algae cause ugly muddying, films and damage to reeds. Decay of these algae causes oxygen depletion in the deep water and shallow water near the shore. The process is self-perpetuating because anoxic conditions at the sediment/water interface causes the release of more adsorbed phosphates from the sediment. The growth of algae produces undesirable effects on the treatment of water for drinking purposes, on fisheries, and on the use of lakes for recreational purposes.
For aluminium and its compounds and salts:
Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals.Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.
Environmental fate:
Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.
Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.
As an element, 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.

Continued...

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.
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	✗	✗	✗
vPvB	✗	✗	✗

PBT Criteria fulfilled?	No
vPvB	No

12.6. Endocrine Disruption Properties

Not Available

12.7. Other adverse effects

Not Available

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Continued...

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ 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. ▸ Treat and neutralise at an approved treatment plant. ▸ 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). ▸ 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

Land transport (ADR-RID)

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 bis(2-aminopropyl ether) propoxylated, trimethylhexamethylene diamine and cocoamine)
14.3. **Transport hazard class(es)**	Class: 8 Subrisk: Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Hazard identification (Kemler): 80 Classification code: C7 Hazard Label: 8 Special provisions: 274 Limited quantity: 1 L Tunnel Restriction Code: 2 (E)

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	2735
14.2. **UN proper shipping name**	Polyamines, liquid, corrosive, n.o.s. * (contains bis(2-aminopropyl ether) propoxylated, trimethylhexamethylene diamine and cocoamine); Amines, liquid, corrosive, n.o.s. * (contains bis(2-aminopropyl ether) propoxylated, trimethylhexamethylene diamine and cocoamine)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 8 ICAO / IATA Subrisk: Not Applicable ERG Code: 8L
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A3 A803 Cargo Only Packing Instructions: 855

Continued...

Cargo Only Maximum Qty / Pack	30 L
Passenger and Cargo Packing Instructions	851
Passenger and Cargo Maximum Qty / Pack	1 L
Passenger and Cargo Limited Quantity Packing Instructions	Y840
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 bis(2-aminopropyl ether) propoxylated, trimethylhexamethylene diamine and cocoamine)
14.3. **Transport hazard class(es)**	IMDG Class: 8 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 Special provisions: 274 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 bis(2-aminopropyl ether) propoxylated, 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 Special provisions: 274 Limited quantity: 1 L Equipment required: PP, EP Fire cones number: 0

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

Not Applicable

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

Product name	Group
alumina hydrate	Not Available
bis(2-aminopropyl ether) propoxylated	Not Available
ammonium polyphosphate	Not Available
aluminium oxide	Not Available
phenol, styrenated	Not Available
zinc borate	Not Available
cocoamine	Not Available
trimethylhexamethylene diamine	Not Available
carbon black	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
alumina hydrate	Not Available
bis(2-aminopropyl ether) propoxylated	Not Available
ammonium polyphosphate	Not Available
aluminium oxide	Not Available
phenol, styrenated	Not Available
zinc borate	Not Available
cocoamine	Not Available
trimethylhexamethylene diamine	Not Available

Continued...

Product name	Ship Type
carbon black	Not Available

SECTION 15 Regulatory information

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

alumina hydrate is found on the following regulatory lists

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

bis(2-aminopropyl ether) propoxylated is found on the following regulatory lists

Not Applicable

ammonium polyphosphate is found on the following regulatory lists

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

aluminium oxide is found on the following regulatory lists

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

phenol, styrenated is found on the following regulatory lists

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

zinc borate is found on the following regulatory lists

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

cocoamine is found on the following regulatory lists

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

trimethylhexamethylene diamine is found on the following regulatory lists

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

carbon black is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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

15.2. Chemical safety assessment

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (alumina hydrate; bis(2-aminopropyl ether) propoxylated; ammonium polyphosphate; aluminium oxide; phenol, styrenated; trimethylhexamethylene diamine; carbon black)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (bis(2-aminopropyl ether) propoxylated)
Japan - ENCS	No (ammonium polyphosphate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (ammonium polyphosphate; phenol, styrenated; cocoamine)
Vietnam - NCI	Yes
Russia - FBEPH	No (phenol, styrenated)

Continued...

National Inventory	Status
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	22/07/2021
Initial Date	27/06/2017

Full text Risk and Hazard codes

H290	May be corrosive to metals.
H302+H312	Harmful if swallowed or if contact with skin.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects.
H351	Suspected of causing cancer.
H360	May damage fertility or the unborn child.
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

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.
For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:
EN 166 Personal eye-protection
EN 340 Protective clothing
EN 374 Protective gloves against chemicals and micro-organisms
EN 13832 Footwear protecting against chemicals
EN 133 Respiratory protective devices

Definitions and abbreviations

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

Reason For Change

A-2.00 - modifications to safety data sheet format

end of SDS