

Slow Cure, Thermally Conductive Adhesive, Flowable (Part A)

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

1.1. Product Identifier

Product name	MC002977 Slow Cure, Thermally Conductive Adhesive, Flowable (Part A)
Synonyms	MC002977
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc oxide and bisphenol a/ diglycidyl ether resin, liquid)
Other means of identification	Not Available

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

Relevant identified uses	Thermally conductive adhesive for bonding and thermal management
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	Premier Farnell plc
Address	150 Armley Road, Leeds, LS12 2QQ
Telephone	+44 (0) 870 129 8608
Fax	-
Email	-

1.4. Emergency telephone number

Association / Organisation	Premier Farnell plc
Emergency telephone numbers	+44 1865 407333
Other emergency telephone numbers	NA

SECTION 2 HAZARDS IDENTIFICATION

2.1. Classification of the substance or mixture

Considered a hazardous mixture according to Reg. (EC) No 1272/2008 and their amendments. Classified as Dangerous Goods for transport purposes.

Classification according to regulation (EC) No 1272/2008 [CLP] ^[1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

2.2. Label elements

CLP label elements	
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SIGNAL WORD **WARNING**

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H410	Very toxic to aquatic life with long lasting effects.

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Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

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

Precautionary statement(s) Response

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

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

Inhalation and/or ingestion may produce health damage*.

Cumulative effects may result following exposure*.

May produce discomfort of the respiratory system*.

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	Classification according to regulation (EC) No 1272/2008 [CLP]
1.1344-28-1. 2.215-691-6 3.Not Available 4.01-2119817795-27-XXXX, 01-2119529248-35-XXXX	40	<u>aluminium oxide</u>	Not Applicable
1.28064-14-4 2.Not Available 3.Not Available 4.Not Available	26	<u>bisphenol F glycidyl ether/ formaldehyde copolymer</u>	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2; H315, H319, H317, H411, EUH019 ^[1]
1.1314-13-2 2.215-222-5 3.030-013-00-7 4.01-2119463881-32-XXXX	25	<u>zinc oxide</u>	Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H410 ^[3]

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1.68609-97-2 2.271-846-8 3.603-103-00-4 4.01-2119485289-22-XXXX	4	<u>(C12-14)alkylglycidyl ether</u>	Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1; H315, H317 ^[3]
1.25068-38-6 2.500-033-5 3.603-074-00-8 4.01-2119456619-26-XXXX	2	<u>bisphenol A/ diglycidyl ether resin, liquid</u>	Eye Irritation Category 2, Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2; H319, H315, H317, H411 ^[3]
1.1333-86-4 2.215-609-9 3. Not Available 4.01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX	0.7	<u>carbon black</u>	Carcinogenicity Category 2; H351 ^[1]
Legend: 1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI 4. Classification drawn from C&L			

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

General	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation. <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh 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. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary. ▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.
Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh 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. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

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

Treat symptomatically.

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

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

[Ellenhorn and Barceloux: Medical Toxicology]



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
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5.3. Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ 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. <p>Combustion products include; carbon dioxide (CO₂) aldehydes 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.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ 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. 																																								
Major Spills	<p>Environmental hazard - contain spillage. Chemical Class: phenols and cresols For release onto land: recommended sorbents listed in order of priority.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 30%;">SORBENT TYPE</th> <th style="width: 10%;">RANK</th> <th style="width: 20%;">APPLICATION</th> <th style="width: 15%;">COLLECTION</th> <th style="width: 25%;">LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>shovel</td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>wood fiber - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>foamed glass - pillow</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R, W, P, DGC</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>wood fibre - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, W, P, DGC</td> </tr> </tbody> </table>	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	wood fiber - pillow	1	throw	pitchfork	R, P, DGC, RT	foamed glass - pillow	2	shovel	shovel	R, W, P, DGC	sorbent clay - particulate	2	shovel	shovel	R, I, P	wood fibre - particulate	3	shovel	shovel	R, W, P, DGC
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Major Spills	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	R, SS, DGC
	wood fiber - particulate	4	blower	skiploader	R, W, P, DGC
	expanded moneral - particulate	4	blower	skiploader	R, I, W, P, DGC
<p>Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT: Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988 Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services. 					

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE

7.1. Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ 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. ▶ 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. ▶ DO NOT allow clothing wet with material to stay in contact with skin
Fire and explosion protection	See section 5
Other information	<ul style="list-style-type: none"> ▶ 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.



7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<p>For aluminas (aluminium oxide):</p> <ul style="list-style-type: none"> ▶ Incompatible with hot chlorinated rubber. ▶ In the presence of chlorine trifluoride may react violently and ignite. ▶ May initiate explosive polymerisation of olefin oxides including ethylene oxide. ▶ Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. ▶ Produces exothermic reaction with oxygen difluoride. ▶ May form explosive mixture with oxygen difluoride. ▶ Forms explosive mixtures with sodium nitrate. ▶ Reacts vigorously with vinyl acetate. <p>Zinc oxide:</p> <ul style="list-style-type: none"> ▶ slowly absorbs carbon dioxide from the air. ▶ may react, explosively with magnesium and chlorinated rubber when heated ▶ is incompatible with linseed oil (may cause ignition) ▶ WARNING: Avoid or control reaction with peroxides. All <i>transition metal</i> peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono- or poly-fluorobenzene show extreme sensitivity to heat and are explosive. ▶ Avoid reaction with borohydrides or cyanoborohydrides ▶ Avoid reaction with amines, mercaptans, strong acids and oxidising agents ▶ Phenols are incompatible with strong reducing substances such as hydrides, nitrides, alkali metals, and sulfides. ▶ Avoid use of aluminium, copper and brass alloys in storage and process equipment. ▶ Heat is generated by the acid-base reaction between phenols and bases. ▶ Phenols are sulfonated very readily (for example, by concentrated sulfuric acid at room temperature), these reactions generate heat. ▶ Phenols are nitrated very rapidly, even by dilute nitric acid. ▶ Nitrated phenols often explode when heated. Many of them form metal salts that tend toward detonation by rather mild shock. ▶ Avoid strong acids, bases. <p>Glycidyl ethers:</p> <ul style="list-style-type: none"> ▶ may form unstable peroxides on storage in air, light, sunlight, UV light or other ionising radiation, trace metals - inhibitor should be maintained at adequate levels ▶ may polymerise in contact with heat, organic and inorganic free radical producing initiators ▶ may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines ▶ react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide ▶ attack some forms of plastics, coatings, and rubber

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

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

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
aluminium oxide	Aluminum oxide; (Alumina)	1.5 mg/m ³	15 mg/m ³	25 mg/m ³
bisphenol F glycidyl ether/ formaldehyde copolymer	Phenol, polymer with formaldehyde, oxiranylmethyl ether	12 mg/m ³	130 mg/m ³	790 mg/m ³
zinc oxide	Zinc oxide	10 mg/m ³	15 mg/m ³	2500 mg/m ³

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bisphenol A/ diglycidyl ether resin, liquid	Epoxy resin (EPON 1001)	90 mg/m3	990 mg/m3	5900 mg/m3
bisphenol A/ diglycidyl ether resin, liquid	Epoxy resin (EPON 1007)	90 mg/m3	990 mg/m3	5900 mg/m3
bisphenol A/ diglycidyl ether resin, liquid	Epoxy resin (EPON 820)	41 mg/m3	450 mg/m3	2700 mg/m3
bisphenol A/ diglycidyl ether resin, liquid	Epoxy resin ERL-2795	32 mg/m3	350 mg/m3	2100 mg/m3
carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
aluminium oxide	Not Available	Not Available
bisphenol F glycidyl ether/ formaldehyde copolymer	Not Available	Not Available
zinc oxide	2,500 mg/m3	500 mg/m3
(C12-14)alkylglycidyl ether	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Not Available	Not Available
carbon black	N.E. mg/m3 / N.E. ppm	1,750 mg/m3

MATERIAL DATA

for zinc oxide:

Zinc oxide intoxication (intoxication zincale) is characterised by general depression, shivering, headache, thirst, colic and diarrhoea.

Exposure to the fume may produce metal fume fever characterised by chills, muscular pain, nausea and vomiting. Short-term studies with guinea pigs show pulmonary function changes and morphologic evidence of small airway inflammation. A no-observed-adverse-effect level (NOAEL) in guinea pigs was 2.7 mg/m3 zinc oxide. Based on present data, the current TLV-TWA may be inadequate to protect exposed workers although known physiological differences in the guinea pig make it more susceptible to functional impairment of the airways than humans.

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1.2 µm) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/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.

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

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

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8.2.1. Appropriate engineering controls	Within each range the appropriate value depends on:										
	<table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table>	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.											
8.2.2. Personal protection											
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 										
Skin protection	See Hand protection below										
Hands/feet protection	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> ▶ frequency and duration of contact, ▶ chemical resistance of glove material, ▶ glove thickness and ▶ dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> ▶ 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. <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <ul style="list-style-type: none"> ▶ When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butadiene rubber), boots and aprons. ▶ DO NOT use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber or polyethylene gloves (which absorb the resin). ▶ DO NOT use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use. 										
Body protection	See Other protection below										
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. 										
Thermal hazards	Not Available										

Respiratory protection

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate. Selection of the Class and Type of respirator will depend upon the level of breathingzone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.



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Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class 1	-
up to 50	1000	-	A-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	A-2
up to 100	10000	-	A-3
100+		-	Airline**

* -Continuous Flow

** -Continuous-flow or positive pressure demand.

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

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

9.1. Information on basic physical and chemical properties

Appearance	dark grey		
Physical state	Liquid	Relative density (Water = 1)	2.2
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)	>20.5
Initial boiling point and boiling range (°C)	>207	Molecular weight (g/mol)	Not Available
Flash point (°C)	>149	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1. Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> ▶ 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

<p>Inhaled</p>	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p>
<p>Ingestion</p>	<p>Acute toxic responses to aluminium are confined to the more soluble forms.</p> <p>The material has NOT been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p>
<p>Skin Contact</p>	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p>
<p>Eye</p>	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
<p>Chronic</p>	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers.</p> <p>When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.</p> <p>The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.</p> <p>Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.</p> <p>There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.</p> <p>Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.</p> <p>Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.</p> <p>After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.</p> <p>At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.</p>

	<p>Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produced 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.</p> <p>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.</p> <p>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 <i>in vitro</i>. 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).</p> <p>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. - <i>Chemistry in Australia</i>, August 1995]</p> <p>All glycidyl ethers show genotoxic potential due to their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.</p> <p>Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals.</p> <p>Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether.</p> <p>A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not <i>n</i>-butyl glycidyl ether, induced morphological transformation in mammalian cells <i>in vitro</i>. <i>n</i>-Butyl glycidyl ether induced micronuclei in mice <i>in vivo</i> following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations <i>in vivo</i> or chromosomal aberrations in animal cells <i>in vitro</i>. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in <i>Drosophila</i>. The glycidyl ethers were generally mutagenic to bacteria.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Zinc is necessary for normal fetal growth and development. Fetal damage may result from zinc deficiency. Only one report in the literature suggested adverse developmental effects in humans due to exposure to excessive levels of zinc. Four women were given zinc supplements of 0.6 mg zinc/kg/day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant. However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming 0.3 mg zinc/kg/day as zinc sulfate or zinc citrate or 0.06 mg zinc/kg/day as zinc aspartate during the last two trimesters. There has been a suggestion that increased serum zinc levels in pregnant women may be associated with an increase in neural tube defects, but others have failed to confirm this association. The developmental toxicity of zinc in experimental animals has been evaluated in a number of investigations. Exposure to high levels of zinc in the diet prior to and/or during gestation has been associated with increased fetal resorptions, reduced fetal weights, altered tissue concentrations of fetal iron and copper, and reduced growth in the offspring.</p> <p>Animal studies suggest that exposure to very high levels of dietary zinc is associated with reduced fetal weight, alopecia, decreased hematocrit, and copper deficiency in offspring. For example, second generation mice exposed to zinc carbonate during gestation and lactation (260 mg/kg/day in the maternal diet), and then continued on that diet for 8 weeks, had reduced body weight, alopecia, and signs of copper deficiency (e.g., lowered hematocrit and occasional achromotrichia [loss of hair colour]). Similarly, mink kits from dams that ingested a time-weighted-average dose of 20.8 mg zinc/kg/day as zinc sulfate also had alopecia and achromotrichia. It is likely that the alopecia resulted from zinc-induced copper deficiency, which is known to cause alopecia in monkeys. However, no adverse effects were observed in parental mice or mink. No effects on reproduction were reported in rats exposed to 50 mg zinc/kg/day as zinc carbonate; however, increased stillbirths were observed in rats exposed to 250 mg zinc/kg/day.</p> <p>Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in 'metal fume fever'; also known as 'brass chills', an industrial disease of short duration. [I.L.O] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas.</p> <p>Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure.</p> <p>Bisphenol F, bisphenol A, fluorine-containing bisphenol A (bisphenol AF), and other diphenylalkanes were found to be oestrogenic in a bioassay with MCF7 human breast cancer cells in culture. Bisphenol F (4,4'-dihydroxydiphenylmethane) has been reported to exhibit oestrogen agonistic properties in the uterotrophic assay. Bisphenol F (BPF) is present in the environment and as a contaminant of food. Humans may, therefore, be exposed to BP. BPF has been shown to have genotoxic and endocrine-disruptor properties in a human hepatoma cell line (HepG2), which is a model system for studies of xenobiotic toxicity. BPF was largely metabolised into the corresponding sulfate by the HepG2 cell line. BPF was metabolised into both sulfate and glucuronide by human hepatocytes, but with differences between individuals. The metabolism of BPF in both HepG2 cells and human hepatocytes suggests the existence of a detoxification pathway.</p> <p>Bisphenol F was orally administered at doses 0, 20, 100 and 500 mg/kg per day for at least 28 days, but no clear endocrine-mediated changes were detected, and it was concluded to have no endocrine-mediated effects in young adult rats. On the other hand, the main effect of bisphenol F was concluded to be liver toxicity based on clinical biochemical parameters and liver weight, but without histopathological changes. The no-observed-effect level for bisphenol F is concluded to be under 20 mg/kg per day since decreased body weight accompanied by decreased serum total cholesterol, glucose, and albumin values were observed in the female rats given 20 mg/kg per day or higher doses of bisphenol F.</p>
<p>Chronic</p>	

Safety Data Sheet



Chronic

Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review.

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

Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadias and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades'

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

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

A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells, whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potentially promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes.

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

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

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

Slow Cure, Thermally Conductive Adhesive, Flowable (Part A)	TOXICITY	IRRITATION
	Not Available	Not Available
aluminium oxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^[1]	Not Available
bisphenol F glycidyl ether/ formaldehyde copolymer	TOXICITY	IRRITATION
	dermal (rat) LD50: 4000 mg/kg ^[2]	* [Ciba-Geigy]
	Oral (rat) LD50: 4000 mg/kg ^[2]	Effects transient
		Eyes * (-) (-) Slight irritant
		May cause allergic response
	Skin * (-) (-) Slight irritant	
zinc oxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg ^[1]	Eye (rabbit) : 500 mg/24 h - mild
		Skin (rabbit) : 500 mg/24 h- mild

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(C12-14)alkylglycidyl ether	TOXICITY	IRRITATION
	Oral (rat) LD50: 16896 mg/kg ^[1]	Eye (rabbit): mild [Ciba]
		Skin (guinea pig): sensitiser
		Skin (human): Irritant
		Skin (human): non- sensitiser
		Skin (rabbit): moderate
		Skin : Moderate
bisphenol A/ diglycidyl ether resin, liquid	TOXICITY	IRRITATION
	dermal (rat) LD50: >800 mg/kg ^[1]	Eye (rabbit): 100mg - Mild
	Oral (rat) LD50: 13447 mg/kg ^[1]	
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2]	Not Available
	Oral (rat) LD50: >8000 mg/kg ^[1]	

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

Slow Cure, Thermally Conductive Adhesive, Flowable (Part A)	<p>Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative.</p> <p>for 1,2-butylene oxide (ethyloxirane):</p> <p>Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m³ ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m³) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals. Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic</p>
ZINC OXIDE	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	<p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg). Reproductive and Developmental Toxicity: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg. Carcinogenicity: IARC concluded that 'there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals. Its overall evaluation was 'Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3). In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997). Genotoxicity: In S. typhimurium strains TA100 and TA1535, BADGE (10-10,000ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg). Immunotoxicity: Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs</p>

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BISPHENOL A / DIGLYCIDYL ETHER RESIN, LIQUID	<p>Consumer exposure to BADGE is almost exclusively from migration of BADGE from cancoatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/ kg/bodyweight day from the 90-day study, and a NOAEL of 15 mg/kg body weight/day (malerats) from the 2-year carcinogenicity study. Both NOAELs are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELs of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs.</p> <p>Foototoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity); NOEL (maternal 60 mg/kg</p>		
CARBON BLACK	<p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p> <p>Inhalation (rat) TCLo: 50 mg/m³/6h/90D-I Nil reported</p>		
Slow Cure, Thermally Conductive Adhesive, Flowable (Part A) & BISPHENOL F GLYCIDYL ETHER/ FORMALDEHYDE COPOLYMER & (C12-14)ALKYLGLYCIDYL ETHER & BISPHENOL A / DIGLYCIDYL ETHER RESIN, LIQUID	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>		
Slow Cure, Thermally Conductive Adhesive, Flowable (Part A) & BISPHENOL F GLYCIDYL ETHER/ FORMALDEHYDE COPOLYMER & BISPHENOL A / DIGLYCIDYL ETHER RESIN, LIQUID	<p>The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics</p> <p>Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities.</p> <p>Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.</p>		
ALUMINIUM OXIDE & CARBON BLACK	No significant acute toxicological data identified in literature search.		
Acute Toxicity	☹	Carcinogenicity	☹
Skin Irritation/Corrosion	✓	Reproductivity	☹
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	☹
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	☹
Mutagenicity	☹	Aspiration Hazard	☹

Legend: ✗ – Data available but does not fill the criteria for classification
✓ – Data required to make classification available
☹ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
aluminium oxide	LC50	96	Fish	0.0029mg/L	2
aluminium oxide	EC50	168	Crustacea	0.0076mg/L	2
aluminium oxide	EC50	48	Crustacea	0.7364mg/L	2
aluminium oxide	EC50	96	Algae or other aquatic plants	0.0054mg/L	2
aluminium oxide	NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2
zinc oxide	BCF	336	Fish	4376.673mg/L	4
zinc oxide	EC20	72	Algae or other aquatic plants	0.023mg/L	4
zinc oxide	EC50	72	Algae or other aquatic plants	0.042mg/L	4
zinc oxide	LC50	96	Fish	0.112mg/L	2



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zinc oxide	EC50	48	Crustacea	0.105mg/L	2
zinc oxide	NOEC	72	Algae or other aquatic plants	0.0000013mg/L	2
(C12-14)alkylglycidyl ether	LC50	96	Fish	>5000mg/L	2
(C12-14)alkylglycidyl ether	EC50	48	Crustacea	6.07mg/L	2
(C12-14)alkylglycidyl ether	NOEC	48	Crustacea	<10mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	LC50	96	Fish	1.2mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	EC50	48	Crustacea	1.1mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	EC50	48	Crustacea	1.7mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	NOEC	504	Crustacea	0.3mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	EC50	72	Algae or other aquatic plants	9.4mg/L	2
carbon black	LC50	96	Fish	>100mg/L	2
carbon black	NOEC	720	Fish	17mg/L	2
carbon black	EC50	48	Crustacea	>100mg/L	2
carbon black	EC50	384	Crustacea	4.9mg/L	2
carbon black	EC50	96	Algae or other aquatic plants	95mg/L	2

Legend:

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - 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

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For bisphenol A and related bisphenols:

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

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

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

A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations. A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.

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

Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F (bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem, Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe³⁺ ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.

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

for 1,2-butylene oxide (ethyloxirane):

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

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



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

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

Ecotoxicity:

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

* Persistence and Bioaccumulation Regulations (Canada 2000).

Foraluminium and its compounds and salts:

Despiteits prevalence in the environment, no known form of life uses aluminium saltsmetabolically. In keeping with its pervasiveness, aluminium is well toleratedby plants and animals.Owing to their prevalence, potential beneficial (orotherwise) biological roles of aluminium compounds are of continuing interest.

Environmentalfate:

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

Acidificationof soils releases aluminium as a transportable solution. Mobilisation ofaluminium by acid rain results in aluminium becoming available for plantuptake.

As anelement, aluminium cannot be degraded in the environment, but may undergovarious precipitation or ligand exchange reactions. Aluminum in compounds hasonly one oxidation state (+3), and would not undergo oxidation-reductionreactions under environmental conditions. Aluminum can be complexed by variousligands present in the environment (e.g., fulvic and humic acids).

The solubility of aluminium in the environment will depend on the ligands presentand the pH.

The trivalent aluminium ion is surrounded by six water molecules in solution. Thehydrated aluminium ion, $[Al(H_2O)_6]^{3+}$, undergoes hydrolysis, in which a stepwiseprotonation of the coordinated water ligands forms bound hydroxide ligands(e.g., $[Al(H_2O)_5(OH)]^{2+}$, $[Al(H_2O)_4(OH)_2]^+$). The speciation of aluminium in wateris pH dependent. The hydrated trivalent aluminium ion is the predominant form atpH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are $Al(OH)_2^+$ and $Al(OH)_3$, while the solid $Al(OH)_3$ is most prevalent between pH 5.2and 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 aluminium hydroxidesappear between pH 4.7 and 10.5, and increase in size until they are transformedinto colloidal particles of amorphous $Al(OH)_3$, which crystallise into gibbsite inacid waters. Polymerisation is affected by the presence of dissolved silica,when enough silica is present, aluminium is precipitated as poorly crystallisedclay mineral species.

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

Monomeralaluminium compounds, typified by aluminium fluoride, chloride, and sulfate, areconsidered reactive or labile compounds, whereas polymeric aluminium speciesreact much more slowly in the environment. Aluminium has a stronger attractionfor fluoride in an acidic environment compared to other inorganic ligand.

Theadsorption of aluminium onto clay surfaces can be a significant factor incontrolling aluminium mobility in the environment, and these adsorptionreactions, measured in one study at pH 3.0-4.1, have been observed to be veryrapid. However, clays may act either as a sink or a source for soluble aluminiumdepending on the degree of aluminium saturation on the clay surface.

Withinthe pH range of 5-6, aluminium complexes with phosphate and is removed fromsolution. Because phosphate is a necessary nutrient in ecological systems, thisimmobilization of both aluminium and phosphate may result in depleted nutrientstates in surface water.

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

Aluminium is often taken up and concentrated in root tissue. Insub-alpine ecosystems, the large root biomass of the Douglas fir, *Abiesamabilis*, takes up aluminium and immobilizes it, preventing largeaccumulation in above-ground tissue. It is unclear to what extent aluminium istaken up into root food crops and leafy vegetables. An uptake factor(concentration of aluminium in the plant/concentration of aluminium in soil) of0.004 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 areunclear. Based upon these values, however, it is clear that aluminium is nottaken up in plants from soil, but is instead biolimited.

Aluminiumconcentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminiumresidue analyses in brook trout have shown that whole-body aluminium contentdecreases as the fish advance from larvae to juveniles. These results implythat the aging larvae begin to decrease their rate of aluminium uptake, toeliminate aluminium at a rate that exceeds uptake, or to maintain approximatelythe same amount of aluminium while the body mass increases. The decline inwhole-body aluminium residues in juvenile brook trout may be related to growthand dilution by edible muscle tissue that accumulated less aluminium than didthe other tissues.

Thegreatest fraction of the gill-associated aluminium was not sorbed to the gilltissue, but to the gill mucus. It is thought that mucus appears to retardaluminium transport from solution to the membrane surface, thus delaying theacute biological response of the fish. It has been reported that concentrationsof aluminium in whole-body tissue of the Atlantic salmon exposed to highconcentrations of aluminium ranging from 3 ug/g (for fish exposed to 33 ug/L) to96 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 aluminium exposureconcentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium(0.5-1.5 mg Ca/L), labile aluminium between 25 and 75 ug/L is toxic. Becausealuminium is toxic to many aquatic species, it is not bioaccumulated to asignificant degree (BCF <300) in most fish and shellfish; therefore,consumption of contaminated fish does not appear to be a significant source ofaluminium exposure in humans.

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

Ecotoxicity:

Freshwaterspecies 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

Freshwaterspecies 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 *Salmotrutta*, LC50 (21-42 d) 0.015- 0.105 mg/L

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

Alga: 1 sp NOEC growth 2.0 mg/L

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

The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4-5.4, with a maximum toxicity occurring around pH 5.0-5.2. The inorganic single univalent 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.)

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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.)
 SoilGuideline: none available.
 AirQuality Standards: none available.
 For zinc and its compounds:

Environmental fate:

Zinc is capable of forming complexes with a variety of organic and inorganic groups (ligands). Biological activity can affect the mobility of zinc in the aquatic environment, although the biota contains relatively little zinc compared to the sediments. Zinc bioconcentrates moderately in aquatic organisms; bioconcentration is higher in crustaceans and bivalve species than in fish. Zinc does not concentrate appreciably in plants, and it does not biomagnify significantly through terrestrial food chains.

However biomagnification may be of concern if concentration of zinc exceeds 1632 ppm in the top 12 inches of soil.

Zinc can persist in water indefinitely and can be toxic to aquatic life. The threshold concentration for fish is 0.1 ppm. Zinc may be concentrated in the aquatic food chain; it is concentrated over 200,000 times in oysters. Copper is synergistic but calcium is antagonistic to zinc toxicity in fish. Zinc can accumulate in freshwater animals at 5 - 1,130 times the concentration present in the water. Furthermore, although zinc actively bioaccumulates in aquatic systems, biota appears to represent a relatively minor sink compared to sediments. Steady-state zinc bioconcentration factors (BCFs) for 12 aquatic species range from 4 to 24,000. Crustaceans and fish can accumulate zinc from both water and food. A BCF of 1,000 was reported for both aquatic plants and fish, and a value of 10,000 was reported for aquatic invertebrates. The order of enrichment of zinc in different aquatic organisms was as follows (zinc concentrations in µg/g dry weight appear in parentheses): fish (25), shrimp (50), mussel (60), periphyton (260), zooplankton (330), and oyster (3,300). The high enrichment in oysters may be due to their ingestion of particulate matter containing higher concentrations of zinc than ambient water. Other investigators have also indicated that organisms associated with sediments have higher zinc concentrations than organisms living in the aqueous layer. With respect to bioconcentration from soil by terrestrial plants, invertebrates, and mammals, BCFs of 0.4, 8, and 0.6, respectively, have been reported. The concentration of zinc in plants depends on the plant species, soil pH, and the composition of the soil.

Plant species do not concentrate zinc above the levels present in soil.

In some fish, it has been observed that the level of zinc found in their bodies did not directly relate to the exposure concentrations. Bioaccumulation of zinc in fish is inversely related to the aqueous exposure. This evidence suggests that fish placed in environments with lower zinc concentrations can sequester zinc in their bodies.

The concentration of zinc in drinking water may increase as a result of the distribution system and household plumbing. Common piping materials used in distribution systems often contain zinc, as well as other metals and alloys. Trace metals may enter the water through corrosion products or simply by the dissolution of small amounts of metals with which the water comes in contact.

Reactions with materials of the distribution system, particularly in soft low-pH waters, very often have produced concentrations of zinc in tap water much greater than those in the raw or treated waters at the plant of origin. Zinc gives water a metallic taste at low levels. Overexposure to zinc also has been associated with toxic effects. Ingestion of zinc or zinc-containing compounds has resulted in a variety of systemic effects in the gastrointestinal and hematological systems and alterations in the blood lipid profile in humans and animals. In addition, lesions have been observed in the liver, pancreas, and kidneys of animals.

Environmental toxicity of zinc in water is dependent upon the concentration of other minerals and the pH of the solution, which affect the ligands that associate with zinc.

Zinc occurs in the environment mainly in the +2 oxidation state. Sorption is the dominant reaction, resulting in the enrichment of zinc in suspended and bed sediments. Zinc in aerobic waters is partitioned into sediments through sorption onto hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their concentrations, pH, redox potential (Eh), salinity, nature and concentrations of complexing ligands, cation exchange capacity, and the concentration of zinc. Precipitation of soluble zinc compounds appears to be significant only under reducing conditions in highly polluted water. Generally, at lower pH values, zinc remains as the free ion. The free ion (Zn⁺²) tends to be adsorbed and transported by suspended solids in unpolluted waters.

Zinc is an essential nutrient that is present in all organisms. Although biota appears to be a minor reservoir of zinc relative to soils and sediments, microbial decomposition of biota in water can produce ligands, such as humic acids, that can affect the mobility of zinc in the aquatic environment through zinc precipitation and adsorption.

The relative mobility of zinc in soil is determined by the same factors that affect its transport in aquatic systems (i.e., solubility of the compound, pH, and salinity)

The redox status of the soil may shift zinc partitioning. Reductive dissolution of iron and manganese (hydr)oxides under suboxic conditions release zinc into the aqueous phase; the persistence of suboxic conditions may then lead to a repartitioning of zinc into sulfide and carbonate solids. The mobility of zinc in soil depends on the solubility of the species of the element and on soil properties such as cation exchange capacity, pH, redox potential, and chemical species present in soil; under anaerobic conditions, zinc sulfide is the controlling species.

Since zinc sulfide is insoluble, the mobility of zinc in anaerobic soil is low. In a study of the effect of pH on zinc solubility: When the pH is <7, an inverse relationship exists between the pH and the amount of zinc insoluble. As negative charges on soil surfaces increase with increasing pH, additional sites for zinc adsorption are activated and the amount of zinc insoluble decreases. The active zinc species in the adsorbed state is the singly charged zinc hydroxide species (i.e., Zn[OH]⁺). Other investigators have also shown that the mobility of zinc in soil increases at lower soil pH under oxidizing conditions and at a lower cation exchange capacity of soil. On the other hand, the amount of zinc in solution generally increases when the pH is >7 in soils high in organic matter.

This is a result of the release of organically complexed zinc, reduced zinc adsorption at higher pH, or an increase in the concentration of chelating agents in soil. For calcareous soils, the relationship between zinc solubility and pH is nonlinear. At a high pH, zinc in solution is precipitated as Zn(OH)₂, zinc carbonate (ZnCO₃), or calcium zincate. Clay and metal oxides are capable of sorbing zinc and tend to retard its mobility in soil. Zinc was more mobile at pH 4 than at pH 6.5 as a consequence of sorption.

Zinc concentrations in the air are relatively low, except near industrial sources such as smelters. No estimate for the atmospheric lifetime of zinc is available at this time, but the fact that zinc is transported long distances in air indicates that its lifetime in air is at least on the order of days. There are few data regarding the speciation of zinc released to the atmosphere. Zinc is removed from the air by dry and wet deposition, but zinc particles with small diameters and low densities suspended in the atmosphere travel long distances from emission sources.

12.2. Persistence and degradability

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

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
zinc oxide	LOW (BCF = 217)
bisphenol A/ diglycidyl ether resin, liquid	LOW (LogKOW = 2.6835)

12.4. Mobility in soil

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



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12.5. Results of PBT and vPvB assessment

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

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ 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. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ 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 or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

	<p>For MC002977-25ML</p> <p>NOT REGULATED by Ground ADR Special Provision 375 NOT REGULATED by Air IATA Special Provision A197 NOT REGULATED by Sea IMDG per 2.10.2.7 NOT REGULATED by ADN Special Provision 274 (The provision of 3.1.2.8 apply)</p>
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Land transport (ADR)

14.1. UN number	3082				
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc oxide and bisphenol a/ diglycidyl ether resin, liquid)				
14.3. Transport hazard class(es)	<table border="1"> <tbody> <tr> <td>Class</td> <td>9</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </tbody> </table>	Class	9	Subrisk	Not Applicable
Class	9				
Subrisk	Not Applicable				



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14.4. Packing group	III
14.5. Environmental hazard	Not Applicable
14.6. Special precautions for user	Hazard identification (Kemler) 90
	Classification code M6
	Hazard Label 9
	Special provisions 274 335 375 601
	Limited quantity 5 L

Air transport (ICAO-IATA / DGR)

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

Sea transport (IMDG-Code / GGVSee)

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

Inland waterways transport (ADN)

14.1. UN number	3082
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc oxide and bisphenol a/ diglycidyl ether resin, liquid)
14.3. Transport hazard class(es)	9 Not Applicable

14.4. Packing group	III	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Classification code	M6
	Special provisions	274; 335; 375; 601
	Limited quantity	5 L
	Equipment required	PP
	Fire cones number	0

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

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

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Customs Inventory of Chemical Substances ECICS (English)

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

UK Workplace Exposure Limits (WELs)

BISPHENOL F GLYCIDYL ETHER/ FORMALDEHYDE COPOLYMER(28064-14-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

ZINC OXIDE(1314-13-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

European Customs Inventory of Chemical Substances ECICS (English)

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

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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

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

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Customs Inventory of Chemical Substances ECICS (English)

European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

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

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

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

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

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

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)

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

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

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

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Customs Inventory of Chemical Substances ECICS (English)

European List of Notified Chemical Substances (ELINCS)

European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

UK Workplace Exposure Limits (WELs)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC,

1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

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15.2. Chemical safety assessment

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

ECHA SUMMARY

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

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Not Classified	GHS08, Dgr, Wng, GHS09, GHS02	H370, H332, H335, H372, H341, H317, H350, H361, H220, H315, H319, H302, H225
2	Not Classified, STOT SE 3, Acute Tox. 4, STOT RE 1, Muta. 2, Skin Sens. 1, Carc. 1B, Repr. 2, Aquatic Chronic 3, Skin Irrit. 2, Eye Irrit. 2, STOT RE 2, Flam. Liq. 2, Aquatic Chronic 4	GHS08, Dgr, Wng, GHS09, GHS02	H370, H332, H335, H372, H341, H317, H350, H361, H220, H315, H319, H302, H225

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

Ingredient	CAS number	Index No	ECHA Dossier
bisphenol F glycidyl ether/ formaldehyde copolymer	28064-14-4	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317, H319
2	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Skin Sens. 1B, Not Classified, STOT SE 3	GHS07, GHS09, Wng	H315, H317, H319, H335

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

Ingredient	CAS number	Index No	ECHA Dossier
zinc oxide	1314-13-2	030-013-00-7	01-2119463881-32-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Aquatic Acute 1	GHS09, Wng	H360, H335, H370, H372, H314, H318, H350, H300, H330, H317, H341
2	Aquatic Acute 1, Aquatic Chronic 1, Acute Tox. 4, Repr. 1A, STOT RE 2, Skin Irrit. 2, Eye Irrit. 2, STOT SE 3, STOT SE 1, Not Classified, STOT RE 1, Skin Corr. 1B, Eye Dam. 1, Carc. 1A, Acute Tox. 2, Skin Sens. 1, Muta. 2	GHS09, Wng, GHS08, Dgr, GHS05, GHS06	H360, H335, H370, H372, H314, H318, H350, H300, H330, H317, H341
1	Aquatic Acute 1, Aquatic Chronic 1	GHS09, Wng	H360, H335, H370, H372, H314, H318, H350, H300, H330, H317, H341
2	Aquatic Acute 1, Aquatic Chronic 1	GHS09, Wng	H360, H335, H370, H372, H314, H318, H350, H300, H330, H317, H341

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

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

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2, Skin Sens. 1	GHS07, Wng	H315, H317
2	Skin Irrit. 2, Skin Sens. 1, Aquatic Chronic 2, Not Classified, Acute Tox. 4, Eye Irrit. 2	GHS07, Wng, GHS09	H315, H317

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

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



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Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317, H319
2	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Not Classified, Acute Tox. 4, Aquatic Chronic 3, Aquatic Chronic 4, Skin Corr. 1A, Aquatic Acute 1, Aquatic Chronic 1	GHS09, Wng, GHS08, Dgr, GHS07	H315, H317, H319, H372
1	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317, H319
2	Skin Irrit. 2, Skin Sens. 1, Eye Irrit. 2, Aquatic Chronic 2, Skin Sens. 1B, Skin Sens. 1A, Not Classified	GHS07, GHS09, Wng	H315, H317, H319
1	Skin Irrit. 2, Skin Sens. 1A, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317
2	Skin Irrit. 2, Skin Sens. 1A, Aquatic Chronic 2	GHS07, GHS09, Wng	H315, H317

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

Ingredient	CAS number	Index No	ECHA Dossier
carbon black	1333-86-4	Not Available	01-2119384822-32-XXXX, 01-2119498801-30-XXXX, 01-2119475601-40-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Not Classified	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332
2	Not Classified, Carc. 2, STOT SE 3, Eye Irrit. 2, STOT RE 2, STOT RE 1, Aquatic Chronic 4, Self-heat. 1, Self-heat. 2, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Flam. Sol. 2, Acute Tox. 4	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332
2	Not Classified, Carc. 2, STOT SE 3, Eye Irrit. 2, STOT RE 2, STOT RE 1, Aquatic Chronic 4, Self-heat. 1, Self-heat. 2, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Flam. Sol. 2, Acute Tox. 4	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332

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

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N ((C12-14)alkylglycidyl ether; bisphenol A/ diglycidyl ether resin, liquid; aluminium oxide; bisphenol F glycidyl ether/ formaldehyde copolymer; carbon black)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (bisphenol F glycidyl ether/ formaldehyde copolymer)
Japan - ENCS	N ((C12-14)alkylglycidyl ether; bisphenol A/ diglycidyl ether resin, liquid)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Full text Risk and Hazard codes

H220	Extremely flammable gas.
H225	Highly flammable liquid and vapour.
H228	Flammable solid.
H251	Self-heating: may catch fire.
H300	Fatal if swallowed.
H302	Harmful if swallowed.
H314	Causes severe skin burns and eye damage.
H318	Causes serious eye damage.
H330	Fatal if inhaled.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.

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H341	Suspected of causing genetic defects.
H350	May cause cancer.
H351	Suspected of causing cancer.
H360	May damage fertility or the unborn child.
H361	Suspected of damaging fertility or the unborn child.
H370	Causes damage to organs.
H372	Causes damage to organs.
H411	Toxic to aquatic life with long lasting effects.

Other information

Ingredients with multiple cas numbers

Name	CAS No
bisphenol F glycidyl ether/ formaldehyde copolymer	28064-14-4, 42616-71-7, 59029-73-1, 94422-39-6
zinc oxide	1314-13-2, 175449-32-8
bisphenol A/ diglycidyl ether resin, liquid	25068-38-6, 25085-99-8

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

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

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

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

Slow Cure, Thermally Conductive Adhesive, Flowable (Part B)

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

1.1. Product Identifier

Product name	MC002977 Slow Cure, Thermally Conductive Adhesive, Flowable (Part B)
Synonyms	MC002977 Part B
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)
Other means of identification	Not Available

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

Relevant identified uses	Thermally conductive adhesive for bonding and thermal management
Uses advised against	Not for use as a spray coating

1.3. Details of the supplier of the safety data sheet

Registered company name	Premier Farnell plc
Address	150 Armley Road, Leeds, LS12 2QQ
Telephone	+44 (0) 870 129 8608
Fax	-
Email	-

1.4. Emergency telephone number

Association / Organisation	Premier Farnell plc
Emergency telephone numbers	+44 1865 407333
Other emergency telephone numbers	NA

SECTION 2 HAZARDS IDENTIFICATION

2.1. Classification of the substance or mixture

Considered a dangerous mixture according to Directive 1999/45/EC, Reg. (EC) No 1272/2008 (if applicable) and their amendments. Classified as Dangerous Goods for transport purposes.

Classification according to regulation (EC) No 1272/2008 [CLP] ^[1]	Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1, Eye Irritation Category 2, Chronic Aquatic Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

2.2. Label elements

CLP label elements	
SIGNAL WORD	WARNING

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H410	Very toxic to aquatic life with long lasting effects.

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Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

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

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

Inhalation and/or ingestion may produce health damage*.

Cumulative effects may result following exposure*.

May produce discomfort of the respiratory system*.

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	Classification according to regulation (EC) No 1272/2008 [CLP]
1.1344-28-1. 2.215-691-6 3.Not Available 4.01-2119817795-27-XXXX, 01-2119529248-35-XXXX	39	<u>aluminium oxide</u>	Not Applicable
1.1314-13-2 2.215-222-5 3.030-013-00-7 4.01-2119463881-32-XXXX	25	<u>zinc oxide</u>	Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H410 ^[3]
1.68541-13-9 2.Not Available 3.Not Available 4. Not Available	18	<u>linoleic acid/4,7,10-trioxa- 1,13-tridecanediamine polyamid</u>	Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1; H315, H318 ^[1]
1.68082-29-1 2.500-191-5 3.Not Available 4. Not Available	9	<u>tall oil/ triethylenetetramine polyamides</u>	Not Applicable

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1.4246-51-9 2.224-207-2 3. Not Available 4. Not Available	3	<u>diethylene glycol, di(3-aminopropyl) ether</u>	Metal Corrosion Category 1, Skin Corrosion/Irritation Category 1A, Serious Eye Damage Category 1, Chronic Aquatic Hazard Category 3; H290, H314, H318, H412 ^[1]
1.108-65-6 2.203-603-9 3.607-195-00-7 4.01-2119475791-29-XXXX	1	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u>	Flammable Liquid Category 3; H226 ^[3]
1.112-24-3 2.203-950-6 3.612-059-00-5 4. Not Available	0.8	<u>triethylenetetramine</u>	Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1B, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 3; H312, H314, H317, H412 ^[3]
1.1333-86-4 2.215-609-9 3. Not Available 4.01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX	0.5	<u>carbon black</u>	Carcinogenicity Category 2; H351 ^[1]

Legend: 1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI 4. Classification drawn from C&L

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

General	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation. <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh 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. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▶ 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. ▶ 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. ▶ Seek medical advice.
Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh 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. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ 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.
Ingestion	<ul style="list-style-type: none"> ▶ 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. ▶ Seek medical advice.



4.2 Most important symptoms and effects, both acute and delayed

See Section 11

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

Treat symptomatically.

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

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

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
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5.3. Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ 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. <p>Combustion products include; carbon dioxide (CO₂) nitrogen oxides (NO_x) 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.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ 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.
Major Spills	<p>Environmental hazard - contain spillage. Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services.

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE

7.1. Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ 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.
Fire and explosion protection	<ul style="list-style-type: none"> ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ 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. ▶ DO NOT allow clothing wet with material to stay in contact with skin <p>See section 5</p>
Other information	<ul style="list-style-type: none"> ▶ 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.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
	<p>For aluminas (aluminium oxide):</p> <ul style="list-style-type: none"> ▶ 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.

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Storage incompatibility

- ▶ Forms explosive mixtures with sodium nitrate.
 - ▶ Reacts vigorously with vinyl acetate.
- Zinc oxide:
- ▶ slowly absorbs carbon dioxide from the air.
 - ▶ may react, explosively with magnesium and chlorinated rubber when heated
 - ▶ is incompatible with linseed oil (may cause ignition)
 - ▶ WARNING: Avoid or control reaction with peroxides. All *transition metal* peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.
 - ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive.
 - ▶ Avoid reaction with borohydrides or cyanoborohydrides
 - ▶ Avoid strong acids, bases.
 - ▶ Avoid reaction with oxidising agents

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides inhalable dust / Aluminium oxides respirable dust	10 mg/m ³ / 4 mg/m ³	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl acetate	274 mg/m ³ / 50 ppm	548 mg/m ³ / 100 ppm	Not Available	Sk
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	propylene glycol monomethyl ether acetate, alpha-isomer	2-Methoxy-1-methylethylacetate	275 mg/m ³ / 50 ppm	550 mg/m ³ / 100 ppm	Not Available	Skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl-2-acetate	275 mg/m ³ / 50 ppm	550 mg/m ³ / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m ³	7 mg/m ³	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
aluminium oxide	Aluminum oxide; (Alumina)	1.5 mg/m ³	15 mg/m ³	25 mg/m ³
zinc oxide	Zinc oxide	10 mg/m ³	15 mg/m ³	2500 mg/m ³
diethylene glycol, di(3-aminopropyl) ether	Diethylene glycol di(3-aminopropyl) ether; (Polyglycol diamine)	13 mg/m ³	140 mg/m ³	850 mg/m ³
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
triethylenetetramine	Triethylenetetramine	3 ppm	5.7 ppm	83 ppm
carbon black	Carbon black	9 mg/m ³	99 mg/m ³	590 mg/m ³

Ingredient	Original IDLH	Revised IDLH
aluminium oxide	Not Available	Not Available
zinc oxide	2,500 mg/m ³	500 mg/m ³
linoleic acid/4,7,10-trioxo-1,13-tridecanediamine polyamid	Not Available	Not Available

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tall oil/ triethylenetetramine polyamides	Not Available	Not Available
diethylene glycol, di(3-aminopropyl) ether	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
triethylenetetramine	Not Available	Not Available
carbon black	N.E. mg/m3 / N.E. ppm	1,750 mg/m3

MATERIAL DATA

for zinc oxide:

Zinc oxide intoxication (intoxication zincale) is characterised by general depression, shivering, headache, thirst, colic and diarrhoea.

Exposure to the fume may produce metal fume fever characterised by chills, muscular pain, nausea and vomiting. Short-term studies with guinea pigs show pulmonary function changes and morphologic evidence of small airway inflammation. A no-observed-adverse-effect level (NOAEL) in guinea pigs was 2.7 mg/m3 zinc oxide. Based on present data, the current TLV-TWA may be inadequate to protect exposed workers although known physiological differences in the guinea pig make it more susceptible to functional impairment of the airways than humans.

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

Polyamide hardeners have much reduced volatility, toxicity and are much less irritating to the skin and eyes than amine hardeners. However commercial polyamides may contain a percentage of residual unreacted amine and all unnecessary contact should be avoided.

for propylene glycol monomethyl ether acetate (PGMEA)

Saturated vapour concentration: 4868 ppm at 20 C.

A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternabral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

8.2. Exposure 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.

8.2.1. Appropriate engineering controls

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

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8.2.1. Appropriate engineering controls	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
	3: Intermittent, low production.	3: High production, heavy use
	4: Large hood or large air mass in motion	4: Small hood-local control only
	Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.	
8.2.2. Personal protection		
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 	
Skin protection	See Hand protection below	
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> ▶ frequency and duration of contact, ▶ chemical resistance of glove material, ▶ glove thickness and ▶ dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> ▶ 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. <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>	
Body protection	See Other protection below	
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. 	
Thermal hazards	Not Available	



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Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

'Forsberg Clothing Performance Index'.

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

Slow Cure, Thermally Conductive Adhesive, Flowable (Part B)

Material	CPI
BUTYL	A
NEOPRENE	A
NITRILE	A
PE/EVAL/PE	A
VITON	A

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

Type 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 / Class 1 P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	AK-3 P2	-
100+ x ES	-	Air-line**	-

* - Continuous-flow; ** - Continuous-flow or positive pressure demand

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)

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	grey		
Physical state	Liquid	Relative density (Water = 1)	2.0
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)	>20.5
Initial boiling point and boiling range (°C)	>145	Molecular weight (g/mol)	Not Available
Flash point (°C)	>110	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2.Chemical stability	<ul style="list-style-type: none"> ▶ 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	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant 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.</p> <p>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Zinc is necessary for normal fetal growth and development. Fetal damage may result from zinc deficiency. Only one report in the literature suggested adverse developmental effects in humans due to exposure to excessive levels of zinc. Four women were given zinc supplements of 0.6 mg zinc/kg/day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant. However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming 0.3 mg zinc/kg/day as zinc sulfate or zinc citrate or 0.06 mg zinc/kg/day as zinc aspartate during the last two trimesters. There has been a suggestion that increased serum zinc levels in pregnant women may be associated with an increase in neural tube defects, but others have failed to confirm this association. The developmental toxicity of zinc in experimental animals has been evaluated in a number of investigations. Exposure to high levels of zinc in the diet prior to and/or during gestation has been associated with increased fetal resorptions, reduced fetal weights, altered tissue concentrations of fetal iron and copper, and reduced growth in the offspring.</p> <p>Animal studies suggest that exposure to very high levels of dietary zinc is associated with reduced fetal weight, alopecia, decreased hematocrit, and copper deficiency in offspring. For example, second generation mice exposed to zinc carbonate during gestation and lactation (260 mg/kg/day in the maternal diet), and then continued on that diet for 8 weeks, had reduced body weight, alopecia, and signs of copper deficiency (e.g., lowered hematocrit and occasional achromotrichia [loss of hair colour]). Similarly, mink kits from dams that ingested a time-weighted-average dose of 20.8 mg zinc/kg/day as zinc sulfate also had alopecia and achromotrichia. It is likely that the alopecia resulted from zinc-induced copper deficiency, which is known to cause alopecia in monkeys. However, no adverse effects were observed in parental mice or mink. No effects on reproduction were reported in rats exposed to 50 mg zinc/kg/day as zinc carbonate; however, increased stillbirths were observed in rats exposed to 250 mg zinc/kg/day.</p> <p>Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in 'metal fume fever'; also known as 'brass chills', an industrial disease of short duration. [I.L.O] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas.</p> <p>Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Acute toxic responses to aluminium are confined to the more soluble forms.</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>

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<p>Eye</p>	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
<p>Chronic</p>	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis (aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity. Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction. There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 um) are able to produce pathogenic effects in the lungs. Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic. Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer. After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans. At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet. Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produced 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</p>

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aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia*, August 1995]

Zinc is necessary for normal fetal growth and development. Fetal damage may result from zinc deficiency. Only one report in the literature suggested adverse developmental effects in humans due to exposure to excessive levels of zinc. Four women were given zinc supplements of 0.6 mg zinc/kg/day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant. However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming 0.3 mg zinc/kg/day as zinc sulfate or zinc citrate or 0.06 mg zinc/kg/day as zinc aspartate during the last two trimesters. There has been a suggestion that increased serum zinc levels in pregnant women may be associated with an increase in neural tube defects, but others have failed to confirm this association. The developmental toxicity of zinc in experimental animals has been evaluated in a number of investigations. Exposure to high levels of zinc in the diet prior to and/or during gestation has been associated with increased fetal resorptions, reduced fetal weights, altered tissue concentrations of fetal iron and copper, and reduced growth in the offspring. Animal studies suggest that exposure to very high levels of dietary zinc is associated with reduced fetal weight, alopecia, decreased hematocrit, and copper deficiency in offspring. For example, second generation mice exposed to zinc carbonate during gestation and lactation (260 mg/kg/day in the maternal diet), and then continued on that diet for 8 weeks, had reduced body weight, alopecia, and signs of copper deficiency (e.g., lowered hematocrit and occasional achromotrichia [loss of hair colour]). Similarly, mink kits from dams that ingested a time-weighted-average dose of 20.8 mg zinc/kg/day as zinc sulfate also had alopecia and achromotrichia. It is likely that the alopecia resulted from zinc-induced copper deficiency, which is known to cause alopecia in monkeys. However, no adverse effects were observed in parental mice or mink. No effects on reproduction were reported in rats exposed to 50 mg zinc/kg/day as zinc carbonate; however, increased stillbirths were observed in rats exposed to 250 mg zinc/kg/day.

Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in 'metal fume fever'; also known as 'brass chills', an industrial disease of short duration. [I.L.O] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas.

Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure.

Overexposure to respirable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity, chest infections

Repeated exposures, in an occupational setting, to high levels of fine- divided dusts may produce a condition known as pneumoconiosis which is the lodgement of any inhaled dusts in the lung irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50,000 inch), are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion (exertional dyspnea), increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Other signs or symptoms include altered breath sounds, diminished lung capacity, diminished oxygen uptake during exercise, emphysema and pneumothorax (air in lung cavity) as a rare complication.

Removing workers from possibility of further exposure to dust generally leads to halting the progress of the lung abnormalities. Where worker-exposure potential is high, periodic examinations with emphasis on lung dysfunctions should be undertaken

Dust inhalation over an extended number of years may produce pneumoconiosis. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. It is further classified as being of noncollagenous or collagenous types. Noncollagenous pneumoconiosis, the benign form, is identified by minimal stromal reaction, consists mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible.

Chronic

Slow Cure, Thermally Conductive Adhesive, Flowable (Part B)	TOXICITY	IRRITATION
	Not Available	Not Available
aluminium oxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^[1]	Not Available
zinc oxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg ^[1]	Eye (rabbit) : 500 mg/24 h - mild Skin (rabbit) : 500 mg/24 h - mild
linoleic acid/4,7,10-trioxa- 1,13-tridecanediamine polyamid	TOXICITY	IRRITATION
	Not Available	Not Available
tall oil/ triethylenetetramine polyamides	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg*d ^[2]	* [Gibson-Homans Co.] **[Devoe]
		Nil reported
diethylene glycol, di(3-aminopropyl) ether	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2500 mg/kg ^[2] Oral (rat) LD50: 4290 mg/kgd ^[2]	Nil reported

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propylene glycol monomethyl ether acetate, alpha-isomer	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	* [CCINFO]
	Inhalation (rat) LC50: 4345 ppm/6h ^[2]	Nil reported
	Oral (rat) LD50: >14.1 ml ^[1]	
triethylenetetramine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 805 mg/kg ^[2]	Eye (rabbit):20 mg/24 h - moderate
	Oral (rat) LD50: 2500 mg/kg ^[2]	Eye (rabbit); 49 mg - SEVERE
		Skin (rabbit): 490 mg open SEVERE
		Skin (rabbit): 5 mg/24 SEVERE
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2]	Not Available
	Oral (rat) LD50: >8000 mg/kg ^[1]	

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

<p>Slow Cure; Thermally Conductive Adhesive, Flowable (Part B)</p>	<p>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.</p> <p>For Fatty Nitrogen Derived (FND)Amides (including several high molecular weight alkyl amino acid amides)</p> <p>The chemicals in the FattyNitrogen Derived (FND) Amides of surfactants are similar to the class ingeneral as to physical/chemical properties, environmental fate and toxicity. Humanexposure to these chemicals is substantially documented.</p> <p>Some typical applications of FNDAmides are: masonry cement additive; curingagent for epoxy resins; closed hydrocarbon systems in oil field production,refineries and chemical plants; and slip and antiblocking additives forpolymers.</p> <p>The safety of the FND Amides tohumans is recognised by the U.S. FDA, which has approved stearamide, oleamideand/or erucamide for adhesives; coatings for articles in food contact; coatingsfor polyolefin films; defoaming agents for manufacture of paper and paperboard;animal glue (defoamer in food packaging); in EVA copolymers for food packaging;lubricants for manufacture of metallic food packaging; irradiation of preparedfoods; release agents in manufacture of food packaging materials, food contactsurface of paper and paperboard; cellophane in food packaging; closure sealinggaskets; and release agents in polymeric resins and petroleum wax. The loworder of toxicity indicates that the use of FND Amides does not pose asignificant hazard to human health. The differences in chain length,degree of saturation of the carbon chains, source of the natural oils, oraddition of an amino group in the chain would not be expected to have an impacton the toxicity profile. This conclusion is supported by a number of studies inthe FND family of chemicals (amines, cationics, and amides as separatecategories) that show no differences in the length or degree of saturation ofthe alkyl substituents and is also supported by the limited toxicity of thesealong-chain substituted chemicals</p> <p>The Fatty nitrogen-derived amides(FND amides) comprise four categories: Subcategory I: Substituted Amides Subcategory II: Fatty Acid Reaction Products with Amino Compounds(Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicalsas major components) Subcategory III: Imidazole Derivatives Subcategory IV: FND Amphoterics</p> <p>Acute Toxicity: The lowacute oral toxicity of the FND Amides is well established across allSubcategories by the available data. The limited acute toxicity of thesechemicals is also confirmed by four acute dermal and two acute inhalationstudies</p> <p>Repeated Dose and ReproductiveToxicity: Two subchronic toxicity studies demonstrating low toxicity areavailable for Subcategory I chemicals. In addition, a 5-day repeated dose studyfor a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory IIchemicals, and based on the low repeat-dose toxicity of the amino compounds(e.g. diethanolamine, triethanolamine) used for producing the Subcategory IIderivatives, the Subcategory I repeat-dose toxicity studies adequately supportSubcategory II.</p> <p>Two subchronic toxicity studiesin Subcategory III confirmed the low order of repeat dose toxicity for the FNDAmides Imidazole derivatives. For Subcategory IV, two subchronic toxicitystudies for one of the chemicals indicated a low order of repeat-dose toxicity forthe FND amphoteric salts similar to that seen in the other categories.</p> <p>Genetic Toxicity in vitro:Based on the lack of effect of one or more chemicals in eachsubcategory, adequate data for mutagenic activity as measured by the <i>Salmonella</i>reverse mutation assay exist for all of the subcategories.</p> <p>Developmental Toxicity: Adevelopmental toxicity study in Subcategory I and in Subcategory IV and a thirdstudy for a chemical in Subcategory III are available. The studies indicatethese chemicals are not developmental toxicants, as expected based on theirstructures, molecular weights, physical properties and knowledge of similarchemicals. As above for repeat-dose toxicity, the data for Subcategory I areadequate to support Subcategory II.</p> <p>In evaluating potential toxicityof the FND Amides chemicals, it is also useful to review the available data forthe related FND Cationic and FND Amines Category chemicals. Acute oral toxicitystudies (approximately 80 studies for 40 chemicals in the three categories)provide LD50 values from approximately 400</p>
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<p>Slow Cure; Thermally Conductive Adhesive, Flowable (Part B)</p>	<p>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 100mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (<i>in vitro</i> bacterial and mammalian cells as well as <i>in vivo</i> 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.</p>
<p>ALUMINIUM OXIDE</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>ZINC OXIDE</p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<p>LINOLEIC ACID/4,7,10-TRIOXA-1,13-TRIDECANEDIAMINE POLYAMID</p>	<p>No significant acute toxicological data identified in literature search. For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides) The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented. Some typical applications of FND Amides are: masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers. The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health. 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 Fatty nitrogen-derived amides (FND amides) comprise four categories: Subcategory I: Substituted Amides Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components) Subcategory III: Imidazole Derivatives Subcategory IV: FND Amphoteric Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory I chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory I derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II. Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories. Genetic Toxicity <i>in vitro</i>: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the <i>Salmonella</i> reverse mutation assay exist for all of the subcategories. Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II. In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines 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 100mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (<i>in vitro</i> bacterial and mammalian cells as well as <i>in vivo</i> 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.</p>
<p>DIETHYLENE GLYCOL, DI(3-AMINOPROPYL) ETHER</p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.</p>

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PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER

for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butylether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methylether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono-, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibit low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all >2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m³ for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m³. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m³), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating. None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m³ (600 ppm) for PnB and 2,010 mg/m³ (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m³ (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m³ (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m³) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m³). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m³), with decreased body weights occurring at 3000 ppm (11058 mg/m³).

For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two-generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.

The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.]

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TRIETHYLENETETRAMINE

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.

Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds.

Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines.

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TRIETHYLENETETRAMINE

In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

For alkyl polyamines:

The alkyl polyamines cluster consists of organic compounds containing two terminal primary amine groups and at least one secondary amine group. Typically these substances are derivatives of ethylenediamine, propylenediamine or hexanediamine. The molecular weight range for the entire cluster is relatively narrow, ranging from 103 to 232

Acute toxicity of the alkyl polyamines cluster is low to moderate via oral exposure and a moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin sensitisers in experimental animals. Repeated exposure in rats via the oral route indicates a range of toxicity from low to high hazard. Most cluster members gave positive results in tests for potential genotoxicity.

Limited carcinogenicity studies on several members of the cluster showed no evidence of carcinogenicity. Unlike aromatic amines, aliphatic amines are not expected to be potential carcinogens because they are not expected to undergo metabolic activation, nor would activated intermediates be stable enough to reach target macromolecules.

Polyamines potentiate NMDA induced whole-cell currents in cultured striatal neurons

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

Triethylenetetramine (TETA) is a severe irritant to skin and eyes and induces skin sensitisation.

TETA is of moderate acute toxicity: LD50(oral, rat) > 2000mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure to saturated vapour via inhalation was tolerated without impairment. Exposure to aerosol leads to reversible irritations of the mucous membranes in the respiratory tract.

Following repeated oral dosing via drinking water only in mice but not in rats at concentration of 3000 ppm there were signs of impairment. The NOAEL is 600 ppm [92 mg/kg bw (oral, 90 days)]. Lifelong dermal application to mice (1.2 mg/mouse) did not result in tumour formation.

There are differing results of the genetic toxicity for TETA. The positive results of the in vitro tests may be the result of a direct genetic action as well as a result of an interference with essential metal ions. Due to this uncertainty of the in vitro tests, the genetic toxicity of TETA has to be assessed on the basis of in vivo tests.

The in vivo micronucleus tests (i.p. and oral) and the SLRL test showed negative results.

There are no human data on reproductive toxicity (fertility assessment). The analogue diethylenetriamine had no effects on reproduction. TETA shows developmental toxicity in animal studies if the chelating property of the substance is effective. The NOEL is 830 mg/kg bw (oral).

Experience with female patients suffering from Wilson's disease demonstrated that no miscarriages and no foetal abnormalities occur during treatment with TETA.

In rats, there are several studies concerning developmental toxicity. The oral treatment of rats with 75, 375 and 750 mg/kg resulted in no effects on dams and fetuses, except slight increased fetal body weight. After oral treatment of rats with 830 or 1670 mg/kg bw only in the highest dose group increased foetal abnormalities in 27/44 fetuses (69,2%) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significant foetal abnormalities of the highest dose group to 3/51 (6,5%) foetus. These findings suggest that the developmental toxicity is produced as a secondary consequence of the chelating properties of TETA.

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

CARBON BLACK

No significant acute toxicological data identified in literature search.

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
Inhalation (rat) TCLo: 50 mg/m³/6h/90D-I Nil reported

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

Legend:
 ✗ – Data available but does not fill the criteria for classification
 ✓ – Data required to make classification available
 ⊘ – Data Not Available to make classification



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SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
aluminium oxide	LC50	96	Fish	0.0029mg/L	2
aluminium oxide	EC50	168	Crustacea	0.0076mg/L	2
aluminium oxide	EC50	48	Crustacea	0.7364mg/L	2
aluminium oxide	EC50	96	Algae or other aquatic plants	0.0054mg/L	2
aluminium oxide	NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2
zinc oxide	BCF	336	Fish	4376.673mg/L	4
zinc oxide	EC20	72	Algae or other aquatic plants	0.023mg/L	4
zinc oxide	EC50	72	Algae or other aquatic plants	0.042mg/L	4
zinc oxide	LC50	96	Fish	0.112mg/L	2
zinc oxide	EC50	48	Crustacea	0.105mg/L	2
zinc oxide	NOEC	72	Algae or other aquatic plants	0.0000013mg/L	2
tall oil/ triethylenetetramine polyamides	LC50	96	Fish	7.07mg/L	2
tall oil/ triethylenetetramine polyamides	EC50	24	Crustacea	9.72mg/L	2
tall oil/ triethylenetetramine polyamides	EC50	48	Crustacea	7.07mg/L	2
tall oil/ triethylenetetramine polyamides	EC50	72	Algae or other aquatic plants	4.34mg/L	2
tall oil/ triethylenetetramine polyamides	NOEC	72	Algae or other aquatic plants	0.5mg/L	2
diethylene glycol, di(3-aminopropyl) ether	EC50	96	Algae or other aquatic plants	268.339mg/L	3
diethylene glycol, di(3-aminopropyl) ether	LC50	96	Fish	>215- <464mg/L	2
diethylene glycol, di(3-aminopropyl) ether	EC50	48	Crustacea	218.16mg/L	2
diethylene glycol, di(3-aminopropyl) ether	NOEC	Not Applicable	Crustacea	>1mg/L	2
diethylene glycol, di(3-aminopropyl) ether	EC50	72	Algae or other aquatic plants	>500mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	96	Algae or other aquatic plants	9.337mg/L	3
propylene glycol monomethyl ether acetate, alpha-isomer	LC50	96	Fish	100mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	NOEC	336	Fish	47.5mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	48	Crustacea	373mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	504	Crustacea	>100mg/L	2
triethylenetetramine	EC50	48	Crustacea	31.1mg/L	1
triethylenetetramine	EC10	72	Algae or other aquatic plants	0.67mg/L	1
triethylenetetramine	EC50	72	Algae or other aquatic plants	2.5mg/L	1
triethylenetetramine	NOEC	72	Algae or other aquatic plants	<2.5mg/L	1
triethylenetetramine	LC50	96	Fish	180mg/L	1
carbon black	LC50	96	Fish	>100mg/L	2
carbon black	NOEC	720	Fish	17mg/L	2

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carbon black	EC50	48	Crustacea	>100mg/L	2
carbon black	EC50	384	Crustacea	4.9mg/L	2
carbon black	EC50	96	Algae or other aquatic plants	95mg/L	2

Legend:

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - 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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

Foraluminium and its compounds and salts:

Despiteits prevalence in the environment, no known form of life uses aluminium saltsmetabolically. In keeping with its pervasiveness, aluminium is well toleratedby plants and animals.Owing to their prevalence, potential beneficial (orotherwise) biological roles of aluminium compounds are of continuing interest.

Environmentalfate:

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

Acidificationof soils releases aluminium as a transportable solution. Mobilisation ofaluminium by acid rain results in aluminium becoming available for plantuptake.

As anelement, aluminium cannot be degraded in the environment, but may undergovarious precipitation or ligand exchange reactions. Aluminium in compounds hasonly one oxidation state (+3), and would not undergo oxidation-reductionreactions under environmental conditions. Aluminium can be complexed by variousligands present in the environment (e.g., fulvic and humic acids).

Thesolubility of aluminium in the environment will depend on the ligands presentand the pH.

The trivalent aluminium ion is surrounded by six water molecules in solution. Thehydrated aluminium ion, $[Al(H_2O)_6]^{3+}$, undergoes hydrolysis, in which a stepwiseprotonation of the coordinated water ligands forms bound hydroxide ligands(e.g., $[Al(H_2O)_5(OH)]^{2+}$, $[Al(H_2O)_4(OH)_2]^+$). The speciation of aluminium in wateris pH dependent. The hydrated trivalent aluminium ion is the predominant form atpH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are $Al(OH)_2^+$ and $Al(OH)_3$, while the solid $Al(OH)_3$ is most prevalent between pH 5.2and 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 aluminium hydroxidesappear between pH 4.7 and 10.5, and increase in size until they are transformedinto colloidal particles of amorphous $Al(OH)_3$, which crystallise to gibbsite inacidic waters. Polymerisation is affected by the presence of dissolved silica;when enough silica is present, aluminium is precipitated as poorly crystallisedclay mineral species.

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

Monomeraluminium compounds, typified by aluminium fluoride, chloride, and sulfate, areconsidered reactive or labile compounds, whereas polymeric aluminium speciesreact much more slowly in the environment. Aluminium has a stronger attractionfor fluoride in an acidic environment compared to other inorganic ligand.

Theadsorption of aluminium onto clay surfaces can be a significant factor incontrolling aluminium mobility in the environment, and these adsorptionreactions, measured in one study at pH 3.0-4.1, have been observed to be veryrapid. However, clays may act either as a sink or a source for soluble aluminiumdepending on the degree of aluminium saturation on the clay surface.

Withinthe pH range of 5-6, aluminium complexes with phosphate and is removed fromsolution. Because phosphate is a necessary nutrient in ecological systems, thisimmobilization of both aluminium and phosphate may result in depleted nutrientstates in surface water.

Plantspecies and cultivars of the same species differ considerably in their ability to take up and translocate aluminium to above-ground parts. Tea leaves maycontain very high concentrations of aluminium, >5,000 mg/kg in old leaves.Other plants that may contain high levels of aluminium include Lycopodium(Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites(Proteaceae). Aluminium is often taken up and concentrated in root tissue. Insub-alpine ecosystems, the large root biomass of the Douglas fir, *Abiesamabilis*, takes up aluminium and immobilizes it, preventing largeaccumulation in above-ground tissue. It is unclear to what extent aluminium istaken up into root food crops and leafy vegetables. An uptake factor(concentration of aluminium in the plant/concentration of aluminium in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported,but the pH and plant species from which these uptake factors were derived areunclear. Based upon these values, however, it is clear that aluminium is nottaken up in plants from soil, but is instead biodiluted.

Aluminiumconcentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminiumresidue analyses in brook trout have shown that whole-body aluminium contentdecreases as the fish advance from larvae to juveniles. These results implythat the aging larvae begin to decrease their rate of aluminium uptake, toeliminate aluminium at a rate that exceeds uptake, or to maintain approximatelythe same amount of aluminium while the body mass increases. The decline inwhole-body aluminium residues in juvenile brook trout may be related to growthand dilution by edible muscle tissue that accumulated less aluminium than didthe other tissues.

Thegreatest fraction of the gill-associated aluminium was not sorbed to the gilltissue, but to the gill mucus. It is thought that mucus appears to retardaluminium transport from solution to the membrane surface, thus delaying theacute biological response of the fish. It has been reported that concentrationsof aluminium in whole-body tissue of the Atlantic salmon exposed to highconcentrations of aluminium ranging from 3 ug/g (for fish exposed to 33 ug/L) to96 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 aluminium exposureconcentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium(0.5-1.5 mg Ca/L), labile aluminium between 25 and 75 ug/L is toxic. Becausealuminium is toxic to many aquatic species, it is not bioaccumulated to asignificant degree (BCF <300) in most fish and shellfish; therefore,consumption of contaminated fish does not appear to be a significant source ofaluminium exposure in humans.

Bioconcentrationof aluminium 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 thesnail. Bioconcentration of aluminium has also been reported for aquatic insects.

Ecotoxicity:

Freshwaterspecies 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 an8-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

CrustaceansLC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

AlgaeEC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0mg/L

Freshwaterspecies 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 *Salmotrutta*, LC50 (21-42 d) 0.015-0.105 mg/L

AmphibiansLC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)

Alga: 1sp NOEC growth 2.0 mg/L

Amongfreshwater aquatic plants, single-celled plants are generally the mostsensitive to aluminium. Fish are generally more sensitive to aluminium thanaquatic invertebrates. Aluminium is a gill toxicant to fish, causing bothionoregulatory and respiratory effects.

Thebioavailability and toxicity of aluminium is generally greatest in acidsolutions. Aluminium in acid habitats has been observed to be toxic to fish andphytoplankton. 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 unitaluminium species ($Al(OH)_2^+$) is thought to be the most toxic. Under very acidconditions, the toxic effects of the high H^+ concentration appear to be moreimportant than the effects of low concentrations of aluminium; at approximatelyneutral pH values, the toxicity of aluminium is greatly reduced. The solubilityof aluminium is also enhanced under alkaline conditions, due to its amphotericcharacter, and some researchers found that the acute toxicity of aluminiumincreased from pH 7 to pH 9. However, the opposite relationship was found inother studies. The uptake and toxicity of aluminium in freshwater organismsgenerally decreases with increasing water hardness under acidic, neutral andalkaline conditions. Complexing agents such as fluoride, citrate and humicsubstances reduce the availability of aluminium to organisms, resulting inlower toxicity. Silicon can also reduce aluminium toxicity to fish.



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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.

For zinc and its compounds:

Environmental fate:

Zinc is capable of forming complexes with a variety of organic and inorganic groups (ligands). Biological activity can affect the mobility of zinc in the aquatic environment, although the biota contains relatively little zinc compared to the sediments. Zinc bioconcentrates moderately in aquatic organisms; bioconcentration is higher in crustaceans and bivalve species than in fish. Zinc does not concentrate appreciably in plants, and it does not biomagnify significantly through terrestrial food chains.

However biomagnification may be of concern if concentration of zinc exceeds 1632 ppm in the top 12 inches of soil.

Zinc can persist in water indefinitely and can be toxic to aquatic life. The threshold concentration for fish is 0.1 ppm. Zinc may be concentrated in the aquatic food chain; it is concentrated over 200,000 times in oysters. Copper is synergistic but calcium is antagonistic to zinc toxicity in fish. Zinc can accumulate in freshwater animals at 5 - 1,130 times the concentration present in the water.

Furthermore, although zinc actively bioaccumulates in aquatic systems, biota appears to represent a relatively minor sink compared to sediments. Steady-state zinc bioconcentration factors (BCFs) for 12 aquatic species range from 4 to 24,000. Crustaceans and fish can accumulate zinc from both water and food. A BCF of 1,000 was reported for both aquatic plants and fish, and a value of 10,000 was reported for aquatic invertebrates. The order of enrichment of zinc in different aquatic organisms was as follows (zinc concentrations in µg/g dry weight appear in parentheses): fish (25), shrimp (50), mussel (60), periphyton (260), zooplankton (330), and oyster (3,300). The high enrichment in oysters may be due to their ingestion of particulate matter containing higher concentrations of zinc than ambient water. Other investigators have also indicated that organisms associated with sediments have higher zinc concentrations than organisms living in the aqueous layer. With respect to bioconcentration from soil by terrestrial plants, invertebrates, and mammals, BCFs of 0.4, 8, and 0.6, respectively, have been reported. The concentration of zinc in plants depends on the plant species, soil pH, and the composition of the soil.

Plant species do not concentrate zinc above the levels present in soil.

In some fish, it has been observed that the level of zinc found in their bodies did not directly relate to the exposure concentrations. Bioaccumulation of zinc in fish is inversely related to the aqueous exposure. This evidence suggests that fish placed in environments with lower zinc concentrations can sequester zinc in their bodies.

The concentration of zinc in drinking water may increase as a result of the distribution system and household plumbing. Common piping materials used in distribution systems often contain zinc, as well as other metals and alloys. Trace metals may enter the water through corrosion products or simply by the dissolution of small amounts of metals with which the water comes in contact.

Reactions with materials of the distribution system, particularly in soft low-pH waters, very often have produced concentrations of zinc in tap water much greater than those in the raw or treated waters at the plant of origin. Zinc gives water a metallic taste at low levels. Overexposure to zinc also has been associated with toxic effects. Ingestion of zinc or zinc-containing compounds has resulted in a variety of systemic effects in the gastrointestinal and hematological systems and alterations in the blood lipid profile in humans and animals. In addition, lesions have been observed in the liver, pancreas, and kidneys of animals.

Environmental toxicity of zinc in water is dependent upon the concentration of other minerals and the pH of the solution, which affect the ligands that associate with zinc.

Zinc occurs in the environment mainly in the +2 oxidation state. Sorption is the dominant reaction, resulting in the enrichment of zinc in suspended and bed sediments. Zinc in aerobic waters is partitioned into sediments through sorption onto hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their concentrations, pH, redox potential (Eh), salinity, nature and concentrations of complexing ligands, cation exchange capacity, and the concentration of zinc. Precipitation of soluble zinc compounds appears to be significant only under reducing conditions in highly polluted water. Generally, at lower pH values, zinc remains as the free ion. The free ion (Zn²⁺) tends to be adsorbed and transported by suspended solids in unpolluted waters.

Zinc is an essential nutrient that is present in all organisms. Although biota appears to be a minor reservoir of zinc relative to soils and sediments, microbial decomposition of biota in water can produce ligands, such as humic acids, that can affect the mobility of zinc in the aquatic environment through zinc precipitation and adsorption.

The relative mobility of zinc in soil is determined by the same factors that affect its transport in aquatic systems (i.e., solubility of the compound, pH, and salinity)

The redox status of the soil may shift zinc partitioning. Reductive dissolution of iron and manganese (hydr)oxides under suboxic conditions release zinc into the aqueous phase; the persistence of suboxic conditions may then lead to a repartitioning of zinc into sulfide and carbonate solids. The mobility of zinc in soil depends on the solubility of the speciated forms of the element and on soil properties such as cation exchange capacity, pH, redox potential, and chemical species present in soil; under anaerobic conditions, zinc sulfide is the controlling species.

Since zinc sulfide is insoluble, the mobility of zinc in anaerobic soil is low. In a study of the effect of pH on zinc solubility: When the pH is <7, an inverse relationship exists between the pH and the amount of zinc insoluble. As negative charges on soil surfaces increase with increasing pH, additional sites for zinc adsorption are activated and the amount of zinc insoluble decreases. The active zinc species in the adsorbed state is the singly charged zinc hydroxide species (i.e., Zn[OH]⁺). Other investigators have also shown that the mobility of zinc in soil increases at lower soil pH under oxidizing conditions and at a lower cation exchange capacity of soil. On the other hand, the amount of zinc in solution generally increases when the pH is >7 in soils high in organic matter.

This is a result of the release of organically complexed zinc, reduced zinc adsorption at higher pH, or an increase in the concentration of chelating agents in soil. For calcareous soils, the relationship between zinc solubility and pH is nonlinear. At a high pH, zinc in solution is precipitated as Zn(OH)₂, zinc carbonate (ZnCO₃), or calcic zincate. Clay and metal oxides are capable of sorbing zinc and tend to retard its mobility in soil. Zinc was more mobile at pH 4 than at pH 6.5 as a consequence of sorption

Zinc concentrations in the air are relatively low, except near industrial sources such as smelters. No estimate for the atmospheric lifetime of zinc is available at this time, but the fact that zinc is transported long distances in air indicates that its lifetime in air is at least on the order of days. There are few data regarding the speciation of zinc released to the atmosphere. Zinc is removed from the air by dry and wet deposition, but zinc particles with small diameters and low densities suspended in the atmosphere travel long distances from emission sources.

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
diethylene glycol, di(3-aminopropyl) ether	HIGH	HIGH
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
triethylenetetramine	LOW	LOW

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12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
zinc oxide	LOW (BCF = 217)
diethylene glycol, di(3-aminopropyl) ether	LOW (LogKOW = -1.4594)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
triethylenetetramine	LOW (LogKOW = -2.6464)

12.4. Mobility in soil

Ingredient	Mobility
diethylene glycol, di(3-aminopropyl) ether	LOW (KOC = 10)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
triethylenetetramine	LOW (KOC = 309.9)

12.5. Results of PBT and vPvB assessment

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

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none">▶ Containers may still present a chemical hazard/ danger when empty.▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none">▶ 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. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none">▶ Reduction▶ Reuse▶ Recycling▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type.</p> <p>Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none">▶ 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 or consult manufacturer for recycling options.▶ Consult State Land Waste Authority for disposal.▶ Bury or incinerate residue at an approved site.▶ Recycle containers if possible, or dispose of in an authorised landfill.
Waste treatment options	Not Available
Sewage disposal options	Not Available

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SECTION 14 TRANSPORT INFORMATION

Labels Required

	<p>For MC002977 - 25 ML</p> <p>NOT REGULATED by Ground ADR Special Provision 375 NOT REGULATED by Air IATA Special Provision A197 NOT REGULATED by Sea IMDG per 2.10.2.7 NOT REGULATED by ADN Special Provision 274 (The provision of 3.1.2.8 apply)</p>
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Land transport (ADR)

14.1.UN number	3077										
14.2.Packing group	III										
14.3.UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)										
14.4.Environmental hazard	Not Applicable										
14.5. Transport hazard class(es)	<table border="1" style="width: 100%;"> <tr> <td>Class</td> <td>9</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	9	Subrisk	Not Applicable						
Class	9										
Subrisk	Not Applicable										
14.6. Special precautions for user	<table border="1" style="width: 100%;"> <tr> <td>Hazard identification (Kemler)</td> <td>90</td> </tr> <tr> <td>Classification code</td> <td>M7</td> </tr> <tr> <td>Hazard Label</td> <td>9</td> </tr> <tr> <td>Special provisions</td> <td>274 335 375 601</td> </tr> <tr> <td>Limited quantity</td> <td>5 kg</td> </tr> </table>	Hazard identification (Kemler)	90	Classification code	M7	Hazard Label	9	Special provisions	274 335 375 601	Limited quantity	5 kg
Hazard identification (Kemler)	90										
Classification code	M7										
Hazard Label	9										
Special provisions	274 335 375 601										
Limited quantity	5 kg										

Air transport (ICAO-IATA / DGR)

14.1. UN number	3077														
14.2. Packing group	III														
14.3. UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. * (contains zinc oxide)														
14.4. Environmental hazard	Not Applicable														
14.5. Transport hazard class(es)	<table border="1" style="width: 100%;"> <tr> <td>ICAO/IATA Class</td> <td>9</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>9L</td> </tr> </table>	ICAO/IATA Class	9	ICAO / IATA Subrisk	Not Applicable	ERG Code	9L								
ICAO/IATA Class	9														
ICAO / IATA Subrisk	Not Applicable														
ERG Code	9L														
14.6. Special precautions for user	<table border="1" style="width: 100%;"> <tr> <td>Special provisions</td> <td>A97 A158 A179 A197</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>956</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>400 kg</td> </tr> <tr> <td>Passenger and Cargo Packing Instructions</td> <td>956</td> </tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td> <td>400 kg</td> </tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td> <td>Y956</td> </tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td> <td>30 kg G</td> </tr> </table>	Special provisions	A97 A158 A179 A197	Cargo Only Packing Instructions	956	Cargo Only Maximum Qty / Pack	400 kg	Passenger and Cargo Packing Instructions	956	Passenger and Cargo Maximum Qty / Pack	400 kg	Passenger and Cargo Limited Quantity Packing Instructions	Y956	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G
Special provisions	A97 A158 A179 A197														
Cargo Only Packing Instructions	956														
Cargo Only Maximum Qty / Pack	400 kg														
Passenger and Cargo Packing Instructions	956														
Passenger and Cargo Maximum Qty / Pack	400 kg														
Passenger and Cargo Limited Quantity Packing Instructions	Y956														
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G														



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Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3077						
14.2. Packing group	III						
14.3. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)						
14.4. Environmental hazard	Marine Pollutant						
14.5. Transport hazard class(es)	<table border="1"><tr><td>IMDG Class</td><td>9</td></tr><tr><td>IMDG Subrisk</td><td>Not Applicable</td></tr></table>	IMDG Class	9	IMDG Subrisk	Not Applicable		
IMDG Class	9						
IMDG Subrisk	Not Applicable						
14.6. Special precautions for user	<table border="1"><tr><td>EMS Number</td><td>F-A, S-F</td></tr><tr><td>Special provisions</td><td>274 335 966 967 969</td></tr><tr><td>Limited Quantities</td><td>5 kg</td></tr></table>	EMS Number	F-A, S-F	Special provisions	274 335 966 967 969	Limited Quantities	5 kg
EMS Number	F-A, S-F						
Special provisions	274 335 966 967 969						
Limited Quantities	5 kg						

Inland waterways transport (ADN)

14.1. UN number	3077										
14.2. Packing group	III										
14.3. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)										
14.4. Environmental hazard	Not Applicable										
14.5. Transport hazard class(es)	<table border="1"><tr><td>9</td><td>Not Applicable</td></tr></table>	9	Not Applicable								
9	Not Applicable										
14.6. Special precautions for user	<table border="1"><tr><td>Classification code</td><td>M7</td></tr><tr><td>Special provisions</td><td>274; 335; 375; 601</td></tr><tr><td>Limited quantity</td><td>5 kg</td></tr><tr><td>Equipment required</td><td>PP, A***</td></tr><tr><td>Fire cones number</td><td>0</td></tr></table>	Classification code	M7	Special provisions	274; 335; 375; 601	Limited quantity	5 kg	Equipment required	PP, A***	Fire cones number	0
Classification code	M7										
Special provisions	274; 335; 375; 601										
Limited quantity	5 kg										
Equipment required	PP, A***										
Fire cones number	0										

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

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

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Customs Inventory of Chemical Substances ECICS (English)

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

UK Workplace Exposure Limits (WELs)

ZINC OXIDE(1314-13-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Customs Inventory of Chemical Substances ECICS (English)

European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

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

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

LINOLEIC ACID/4,7,10-TRIOXA-1,13-TRIDECANEDIAMINE POLYAMID(68541-13-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

TALL OIL/ TRIETHYLENETETRAMINE POLYAMIDES(68082-29-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)

DIETHYLENE GLYCOL, DI(3-AMINOPROPYL) ETHER(4246-51-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Customs Inventory of Chemical Substances ECICS (English)

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

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER(108-65-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Customs Inventory of Chemical Substances ECICS (English)

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

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Bulgarian)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Czech)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Danish)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Dutch)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Estonian)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Finnish)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Maltese)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Polish)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Portuguese)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Romanian)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovak)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovenian)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Spanish)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Swedish)

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

UK Workplace Exposure Limits (WELs)

TRIETHYLENETETRAMINE(112-24-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Customs Inventory of Chemical Substances ECICS (English)

European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

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

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

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

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

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

European Customs Inventory of Chemical Substances ECICS (English)

European List of Notified Chemical Substances (ELINCS)

European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

UK Workplace Exposure Limits (WELs)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

15.2. Chemical safety assessment

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

ECHA SUMMARY

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

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Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Not Classified	GHS08, Dgr, Wng, GHS09, GHS02	H370, H332, H335, H372, H341, H317, H350, H361, H220, H315, H319, H302, H225
2	Not Classified, STOT SE 3, Acute Tox. 4, STOT RE 1, Muta. 2, Skin Sens. 1, Carc. 1B, Repr. 2, Aquatic Chronic 3, Skin Irrit. 2, Eye Irrit. 2, STOT RE 2, Flam. Liq. 2, Aquatic Chronic 4	GHS08, Dgr, Wng, GHS09, GHS02	H370, H332, H335, H372, H341, H317, H350, H361, H220, H315, H319, H302, H225

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

Ingredient	CAS number	Index No	ECHA Dossier
zinc oxide	1314-13-2	030-013-00-7	01-2119463881-32-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Aquatic Acute 1	GHS09, Wng	H360, H335, H370, H372, H314, H318, H350, H300, H330, H317, H341
2	Aquatic Acute 1, Aquatic Chronic 1, Acute Tox. 4, Repr. 1A, STOT RE 2, Skin Irrit. 2, Eye Irrit. 2, STOT SE 3, STOT SE 1, Not Classified, STOT RE 1, Skin Corr. 1B, Eye Dam. 1, Carc. 1A, Acute Tox. 2, Skin Sens. 1, Muta. 2	GHS09, Wng, GHS08, Dgr, GHS05, GHS06	H360, H335, H370, H372, H314, H318, H350, H300, H330, H317, H341
1	Aquatic Acute 1, Aquatic Chronic 1	GHS09, Wng	H360, H335, H370, H372, H314, H318, H350, H300, H330, H317, H341
2	Aquatic Acute 1, Aquatic Chronic 1	GHS09, Wng	H360, H335, H370, H372, H314, H318, H350, H300, H330, H317, H341

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

Ingredient	CAS number	Index No	ECHA Dossier
linoleic acid/4,7,10-trioxo-1,13-tridecanediamine polyamid	68541-13-9	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Eye Irrit. 2	GHS07, Wng	H319
2	Not Classified, Eye Irrit. 2, Skin Irrit. 2, Skin Sens. 1, Eye Dam. 1	Wng, GHS05, Dgr	H315, H317, H318

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

Ingredient	CAS number	Index No	ECHA Dossier
tall oil/ triethylenetetramine polyamides	68082-29-1	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Skin Irrit. 2, Skin Sens. 1A, Eye Dam. 1, Aquatic Chronic 2, Skin Sens. 1, Aquatic Chronic 3, Eye Irrit. 2, Acute Tox. 4, Resp. Sens. 1, Not Classified, Met. Corr. 1, Aquatic Chronic 4, Skin Corr. 1B, Aquatic Acute 1, Aquatic Chronic 1	GHS09, GHS05, Dgr, Wng, GHS08, GHS06	H317, H318, H334, H312, H290, H314

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

Ingredient	CAS number	Index No	ECHA Dossier
diethylene glycol, di(3-aminopropyl) ether	4246-51-9	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Corr. 1B	GHS05, Dgr	H314
2	Skin Corr. 1B, Skin Sens. 1, Eye Dam. 1, Skin Corr. 1C, Aquatic Chronic 3, Met. Corr. 1, Not Classified, Eye Irrit. 2	GHS05, Dgr, Wng	H314, H317, H318, H290
1	Skin Corr. 1B, Aquatic Chronic 4	GHS05, Dgr	H314
2	Skin Corr. 1B, Aquatic Chronic 4	GHS05, Dgr	H314

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

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Ingredient	CAS number	Index No	ECHA Dossier
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6	607-195-00-7	01-2119475791-29-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Flam. Liq. 3, Eye Irrit. 2, Eye Dam. 1, Not Classified, STOT SE 3	GHS02, Wng, GHS03, GHS05, Dgr	H226, H319, H335, H336

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

Ingredient	CAS number	Index No	ECHA Dossier
triethylenetetramine	112-24-3	612-059-00-5	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3	GHS07, GHS05, Dgr	H312, H314, H317
2	Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3, Eye Dam. 1, Acute Tox. 3, Resp. Sens. 1, STOT SE 3, Aquatic Chronic 2, Not Classified	GHS05, Dgr, GHS06, GHS08, GHS09	H314, H317, H318, H302, H311, H334, H335

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

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

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Not Classified	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332
2	Not Classified, Carc. 2, STOT SE 3, Eye Irrit. 2, STOT RE 2, STOT RE 1, Aquatic Chronic 4, Self-heat. 1, Self-heat. 2, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Flam. Sol. 2, Acute Tox. 4	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332
2	Not Classified, Carc. 2, STOT SE 3, Eye Irrit. 2, STOT RE 2, STOT RE 1, Aquatic Chronic 4, Self-heat. 1, Self-heat. 2, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Flam. Sol. 2, Acute Tox. 4	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332

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

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (propylene glycol monomethyl ether acetate, alpha-isomer; tall oil/ triethylenetetramine polyamides; linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid; aluminium oxide; carbon black; triethylenetetramine)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid)
Japan - ENCS	N (tall oil/ triethylenetetramine polyamides; linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Full text Risk and Hazard codes

H220	Extremely flammable gas.
H225	Highly flammable liquid and vapour.
H226	Flammable liquid and vapour.
H228	Flammable solid.
H251	Self-heating: may catch fire.

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H290	May be corrosive to metals.
H300	Fatal if swallowed.
H302	Harmful if swallowed.
H311	Toxic in contact with skin.
H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H318	Causes serious eye damage.
H330	Fatal if inhaled.
H332	Harmful if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H351	Suspected of causing cancer.
H360	May damage fertility or the unborn child.
H361	Suspected of damaging fertility or the unborn child.
H370	Causes damage to organs.
H372	Causes damage to organs.
H412	Harmful to aquatic life with long lasting effects.

Other information

Ingredients with multiple cas numbers

Name	CAS No
zinc oxide	1314-13-2, 175449-32-8
diethylene glycol, di(3-aminopropyl) ether	25265-19-4, 4246-51-9
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 142300-82-1, 84540-57-8

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

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

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

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

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Safety Data Sheet



Part Number

MC002977

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