Kit Revision Date: 19/01/2022

8329TFS Thermally Conductive Epoxy Adhesive Kit

MG Chemicals Multipart Product Kit

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

Kit Content

Part	*Product Name*	*Product Use*
A	8329TFS-A	Thermally conductive adhesive for bonding and thermal management
B	8329TFS-B	Thermally conductive adhesive for bonding and thermal management

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

Transportation Instruction

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

8329TFS-A Thermally Conductive Epoxy Adhesive (Part A)

Mektronics

Version No: **8.18**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **19/01/2022**
Print Date: **19/01/2022**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	8329TFS-A
Synonyms	8329TFS-25ML, 8329TFS-50ML,SDS Code: 8329TFS-Part A
Other means of identification	Thermally Conductive Epoxy Adhesive (Part A)

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

Relevant identified uses	Thermally conductive adhesive for bonding and thermal management

Details of the supplier of the safety data sheet

Registered company name	**Mektronics**	**MG Chemicals (Head office)**
Address	Unit 3 8 Bonz Place, Seven Hills NSW 2147 Australia	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	1300 788 701	+(1) 800-201-8822
Fax	1300 722 004	+(1) 800-708-9888
Website	www.mektronics.com.au	www.mgchemicals.com
Email	sales@mektronics.com.au	Info@mgchemicals.com

Emergency telephone number

Association / Organisation	**Verisk 3E (Access Code: 335388)**
Emergency telephone numbers	+61 1 800 686 951
Other emergency telephone numbers	+61 280363166

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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

Hazard statement(s)

H319	Causes serious eye irritation.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H410	Very toxic to aquatic life with long lasting effects.
AUH019	May form explosive peroxides.

Precautionary statement(s) Prevention

P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.

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P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water 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 to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1344-28-1.	40	aluminium oxide
9003-36-5	26	phenol/ formaldehyde glycidyl ether copolymer
1314-13-2	25	zinc oxide
68609-97-2	4	(C12-14)alkylglycidyl ether
1675-54-3	2	bisphenol A diglycidyl ether
1333-86-4	0.7	carbon black
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ 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	If skin or hair contact occurs: ▸ Quickly but gently, wipe material off skin with a dry, clean cloth. ▸ Immediately remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

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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

Extinguishing media

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

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

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Use 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	‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) aldehydes metal oxides other pyrolysis products typical of burning organic material. When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.
HAZCHEM	•3Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ In the event of a spill of a reactive diluent, the focus is on containing the spill to prevent contamination of soil and surface or ground water. ‣ If irritating vapors are present, an approved air-purifying respirator with organic vapor canister is recommended for cleaning up spills and leaks. ‣ For small spills, reactive diluents should be absorbed with sand. Environmental hazard - contain spillage. ‣ 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.

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	▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Chemical Class: phenols and cresols For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS

LAND SPILL - SMALL

cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
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

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

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988
Industrial spills or releases of reactive diluents are infrequent and generally contained. If a large spill does occur, the material should be captured, collected, and reprocessed or disposed of according to applicable governmental requirements.
An approved air-purifying respirator with organic-vapor canister is recommended for emergency work.
Moderate hazard.

- 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.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ 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

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Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Metal can or drum ‣ Packaging as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate. Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt. 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 ‣ 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. Glycidyl ethers: ‣ 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 Reactive diluents are stable under recommended storage conditions, but can decompose at elevated temperatures.In some cases, decomposition can cause pressure build-up in closed systems. ‣ Avoid cross contamination between the two liquid parts of product (kit). ‣ If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur. ‣ This excess heat may generate toxic vapour ‣ Avoid reaction with amines, mercaptans, strong acids and oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	aluminium oxide	Aluminium oxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	zinc oxide	Zinc oxide (dust)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	zinc oxide	Zinc oxide (fume)	5 mg/m3	10 mg/m3	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
aluminium oxide	15 mg/m3	170 mg/m3	990 mg/m3
zinc oxide	10 mg/m3	15 mg/m3	2,500 mg/m3
bisphenol A diglycidyl ether	39 mg/m3	430 mg/m3	2,600 mg/m3
bisphenol A diglycidyl ether	90 mg/m3	990 mg/m3	5,900 mg/m3
carbon black	9 mg/m3	99 mg/m3	590 mg/m3

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Ingredient	Original IDLH	Revised IDLH
aluminium oxide	Not Available	Not Available
phenol/ formaldehyde glycidyl ether copolymer	Not Available	Not Available
zinc oxide	500 mg/m3	Not Available
(C12-14)alkylglycidyl ether	Not Available	Not Available
bisphenol A diglycidyl ether	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
phenol/ formaldehyde glycidyl ether copolymer	E	≤ 0.1 ppm
(C12-14)alkylglycidyl ether	E	≤ 0.1 ppm
bisphenol A diglycidyl ether	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

for 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

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

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

The Odour Safety Factor (OSF) is defined as:

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

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As 'A' for 50-90% of persons being distracted
C	1-26	As 'A' for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As 'D' for less than 10% of persons aware of being tested

The concentration of dust, for application of respirable dust limits, is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative log-normal function with a median aerodynamic diameter of 4.0 um (+-) 0.3 um and with a geometric standard deviation of 1.5 um (+-) 0.1 um, i.e..generally less than 5 um.
For epichlorohydrin
Odour Threshold Value: 0.08 ppm
NOTE: Detector tubes for epichlorohydrin, measuring in excess of 5 ppm, are commercially available.
Exposure at or below the recommended TLV-TWA is thought to minimise the potential for adverse respiratory, liver, kidney effects. Epichlorohydrin has been implicated as a human skin sensitiser, hence individuals who are hypersusceptible or otherwise unusually responsive to certain chemicals may NOT be adequately protected from adverse health effects.
Odour Safety Factor (OSF)
OSF=0.54 (EPICHLOROHYDRIN)

Exposure controls

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)

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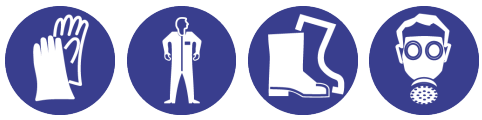
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling,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

Within each range the appropriate value depends on:

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

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

Personal protection



Eye and face protection

- 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

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
· Contaminated gloves should be replaced.
As defined in ASTM F-739-96 in any application, gloves are rated as:
· Excellent when breakthrough time > 480 min
· Good when breakthrough time > 20 min
· Fair when breakthrough time < 20 min
· Poor when glove material degrades
For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:
· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential
Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
When handling liquid-grade epoxy resins wear chemically protective gloves , boots and aprons.
The performance, based on breakthrough times ,of:

- Ethyl Vinyl Alcohol (EVAL laminate) is generally excellent
- Butyl Rubber ranges from excellent to good

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	· Nitrile Butyl Rubber (NBR) from excellent to fair. · Neoprene from excellent to fair · Polyvinyl (PVC) from excellent to poor As defined in ASTM F-739-96 · Excellent breakthrough time > 480 min · Good breakthrough time > 20 min · Fair breakthrough time < 20 min · Poor glove material degradation Gloves should be tested against each resin system prior to making a selection of the most suitable type. Systems include both the resin and any hardener, individually and collectively) · **DO NOT** use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin). · **DO NOT** use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use. Replacement time should be considered when selecting the most appropriate glove. It may be more effective to select a glove with lower chemical resistance but which is replaced frequently than to select a more resistant glove which is reused many times
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the 'Exposure Standard' (or ES), respiratory protection is required.
Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS	-	A-PAPR-AUS / Class 1
up to 50 x ES	-	A-AUS / Class 1	-
up to 100 x ES	-	A-2	A-PAPR-2 ^

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

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

SECTION 9 Physical and chemical properties

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

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Flash point (°C)	>149	**Taste**	Not Available
Evaporation rate	Not Available BuAC = 1	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	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. Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. In animal testing, exposure to aerosols of some reactive diluents (notably o-cresol glycidyl ether, CAS RN: 2210-79-9) has been reported to affect the adrenal gland, central nervous system, kidney, liver, ovaries, spleen, testes, thymus, and respiratory tract. 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. 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. Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.
Ingestion	Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. Reactive diluents exhibit a range of ingestion hazards. Small amounts swallowed incidental to normal handling operations are not likely to cause injury. However, swallowing larger amounts may cause injury. Male rats exposed to a single oral dose of bisphenol A diglycidyl ether (BADGE) at 750, 1000, and 2000 mg/kg/day showed a significantly increase in the number of immature and maturing sperm on the testis. There were no significant differences with respect to sperm head count, sperm motility, and sperm abnormality in the BADGE treatment groups At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes) At sufficiently high doses the material may be nephrotoxic (i.e. poisonous to the kidney). Acute toxic responses to aluminium are confined to the more soluble forms. 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. Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material may accentuate any pre-existing dermatitis condition

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	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. Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Bisphenol A diglycidyl ether (BADGE) may produce contact dermatitis characterised by erythema and oedema, with weeping followed by crusting and scaling. A liquid resin with a molecular weight of 350 produced severe skin irritation in rabbits when applied daily for 4 hours over 20 days. Following the initial contact there may be a discrete erythematous lesion, confined to the point of contact, which may persist for 48 hours to 10 days; the erythema may give way to a papular, vesicular rash with scaling. In animals uncured resin produces moderate ante-mortem depression, loss of body weight and diarrhoea. Local irritation, inflammation and death resulting from respiratory system depression are recorded. Higher molecular weight resins generally produce lower toxicity. Skin contact with reactive diluents may cause slight to moderate irritation with local redness. Repeated or prolonged skin contact may cause burns. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but moderate, 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.
Eye	Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury. Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity.. Testicular abnormalities (including testicular atrophy with decreased spermatogenic activity) following exposure to glycidyl ethers have been reported. Haemopoietic abnormalities following exposure to glycidyl ethers, including alteration of the leukocyte count, atrophy of lymphoid tissue, and bone marrow cytotoxicity have also been reported. These abnormalities were usually observed along with pneumonia and/or toxemia, and therefore may be secondary effects. However, especially in light of the generalized reduction in leukocytes and the atrophy of lymphoid tissues, the observed haemopoietic abnormalities may have been predisposing factors to pneumonia. While none of the individual research reports are conclusive with respect to the ability of glycidyl ethers to produce permanent changes to the testes or haemopoietic system in laboratory animals, the pattern of displayed effects is reason for concern Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether. A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not n-butyl glycidyl ether, induced morphological transformation in mammalian cells in vitro. n-Butyl glycidyl ether induced micronuclei in mice in vivo following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations in vivo or chromosomal aberrations in animal cells in vitro. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in Drosophila. The glycidyl ethers were generally mutagenic to bacteria. Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production. Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure. When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity. Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction. There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.

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Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.
Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.
After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.
At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.
Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer s disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.
Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.
Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of 'tau' a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).
Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [*Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995*]
Bisphenol A diglycidyl ethers (BADGEs) produce sensitisation dermatitis characterised by a papular, vesicular eczema with considerable itching of the back of the hand, the forearm and face and neck. This lesion may persist for 10-14 days after withdrawal from exposure and recur immediately on re-exposure. This dermatitis may persist for longer periods following each exposure but is unlikely to become more intense. Lesions may develop a brownish colour and scaling occurs frequently. Lower molecular weight species produce sensitisation more readily.
In mice technical grades of bisphenol A diglycidyl ether produced epidermal tumours and a small increase in the incidence kidney tumours in males and of lymphoreticular/ haematopoietic tumours in females. Subcutaneous injection produced a small number of fibrosarcomas in rats. BADGE is listed as an IARC Group 3 carcinogen, meaning it is 'not classifiable as to its carcinogenicity to humans'. Concern has been raised over this possible carcinogenicity because BADGE is used in epoxy resins in the lining of some tin cans for foodstuffs, and unreacted BADGE may end up in the contents of those cans.
For some reactive diluents, prolonged or repeated skin contact may result in absorption of potentially harmful amounts or allergic skin reactions
Exposure to some reactive diluents (notably neopentylglycol diglycidyl ether, CAS RN:17557-23-2) has caused cancer in some animal testing.
Following an oral intake of extremely high doses of zinc (where 300 mg Zn/d – 20 times the US Recommended Dietary Allowance (RDA) – is a 'low intake' overdose), nausea, vomiting, pain, cramps and diarrhea may occur. There is evidence of induced copper deficiency, alterations of blood lipoprotein levels, increased levels of LDL, and decreased levels of HDL at long-term intakes of 100 mg Zn/d. The USDA RDA is 15 mg Zn/d.
There is also a condition called the 'zinc shakes' or 'zinc chills' or metal fume fever that can be induced by the inhalation of freshly formed zinc oxide formed during the welding of galvanized materials.
Supplemental zinc can prevent iron absorption, leading to iron deficiency and possible peripheral neuropathy, with loss of sensation in extremities.
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

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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.
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.
Bisphenol F, bisphenol A, fluorine-containing bisphenol A (bisphenol AF), and other diphenylalkanes were found to be oestrogenic in a bioassay with MCF7 human breast cancer cells in culture Bisphenol F (4,4'-dihydroxydiphenylmethane) has been reported to exhibit oestrogen agonistic properties in the uterotrophic assay. Bisphenol F (BPF) is present in the environment and as a contaminant of food. Humans may, therefore, be exposed to BP. BPF has been shown to have genotoxic and endocrine-disruptor properties in a human hepatoma cell line (HepG2), which is a model system for studies of xenobiotic toxicity. BPF was largely metabolised into the corresponding sulfate by the HepG2 cell line. BPF was metabolised into both sulfate and glucuronide by human hepatocytes, but with differences between individuals. The metabolism of BPF in both HepG2 cells and human hepatocytes suggests the existence of a detoxification pathway
Bisphenol F was orally administered at doses 0, 20, 100 and 500 mg/kg per day for at least 28 days, but no clear endocrine-mediated changes were detected, and it was concluded to have no endocrine-mediated effects in young adult rats. On the other hand, the main effect of bisphenol F was concluded to be liver toxicity based on clinical biochemical parameters and liver weight, but without histopathological changes. The no-observed-effect level for bisphenol F is concluded to be under 20 mg/kg per day since decreased body weight accompanied by decreased serum total cholesterol, glucose, and albumin values were observed in the female rats given 20 mg/kg per day or higher doses of bisphenol F.
Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. The presence of the p-hydroxy group on the benzene rings is though to be responsible for the oestradiol mimicry.
. Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review.
A 2009 study on Chinese workers in bisphenol A factories found that workers were four times more likely to report erectile dysfunction, reduced sexual desire and overall dissatisfaction with their sex life than workers with no heightened bisphenol A exposure. Bisphenol A workers were also seven times more likely to have ejaculation difficulties. They were also more likely to report reduced sexual function within one year of beginning employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties.
Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/ litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades'
One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials'
One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood.
A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells.[whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes.
Bisphenol A is the isopropyl adduct of 4,4'-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called 'cytostatic hormones'. Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown to be oestrogenic in vitro; such sealants may represent an additional source of xenoestrogens in humans and may be the cause of additional concerns in children.
Concerns have been raised about the possible developmental effects on the foetus/embryo or neonate resulting from the leaching of bisphenol A from epoxy linings in metal cans which come in contact with food-stuffs.
Many drugs, including naproxen, salicylic acid, carbamazepine and mefenamic acid can, in vitro, significantly inhibit bisphenol A glucuronidation (detoxification).
BPA belongs to the list of compounds having this property as the rodent models have shown that BPA exposure is linked with increased body weigh (obesogens)t. Several mechanisms can help explain the effect of BPA on body weight increase. A possible mechanism leading to triglyceride accumulation is the decreased production of the hormone adiponectin from all human adipose tissue tested when exposed to very low levels (below nanomolar range) of BPA in cell or explant culture settings . The expression of leptin as well as several enzymes and transcription factors is also affected by BPA exposure in vivo as well as in vitro. Together, the altered expression and activity of these important mediators of fat metabolism could explain the increase in weight following BPA exposure in rodent models. These results also suggest that, together with other obesogens, low, environmentally relevant levels of BPA may contribute to the human obesity phenomenon.

8329TFS-A Thermally Conductive Epoxy Adhesive (Part A)

TOXICITY	IRRITATION
Not Available	Not Available

aluminium oxide

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

phenol/ formaldehyde glycidyl ether copolymer

TOXICITY	IRRITATION
dermal (rat) LD50: >400 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50; >5000 mg/kg[2]	Skin: adverse effect observed (irritating)[1]

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zinc oxide

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) : 500 mg/24 h - mild
Inhalation(Rat) LC50; >1.79 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50; >5000 mg/kg[1]	Skin (rabbit) : 500 mg/24 h- mild
	Skin: no adverse effect observed (not irritating)[1]

(C12-14)alkylglycidyl ether

TOXICITY	IRRITATION
Oral (Rat) LD50; >10000 mg/kg[2]	Eye (rabbit): mild [Ciba]
	Eye: adverse effect observed (irritating)[1]
	Skin (guinea pig): sensitiser
	Skin (human): Irritant
	Skin (human): non- sensitiser
	Skin (rabbit): moderate
	Skin : Moderate
	Skin: adverse effect observed (irritating)[1]

bisphenol A diglycidyl ether

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 2 mg/24h - SEVERE
Oral (Rat) LD50; >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit): 500 mg - mild
	Skin: adverse effect observed (irritating)[1]

carbon black

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >3000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50; >8000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

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

8329TFS-A Thermally Conductive Epoxy Adhesive (Part A)

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

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	weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI. Genotoxicity Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels. Carcinogenicity. The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons. Neurodegenerative diseases. Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease.” There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases.Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment. Contact sensitivity: It has been suggested that the body burden of aluminium may be linked to different iseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome. Aluminium acts not only as an adjuvant,stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitisers causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response.Once inside the skin, the metal ions must bind to proteins to become immunologically reactive.The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminium Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste The various members of the bisphenol family produce hormone like effects, seemingly as a result of binding to estrogen receptor-related receptors (ERRs; not to be confused with estrogen receptors) A suspected estrogen-related receptors (ERR) binding agent: Estrogen-related receptors (ERR, oestrogen-related receptors) are so named because of sequence homology with estrogen receptors but do not appear to bind estrogens or other tested steroid hormones. The ERR family have been demonstrated to control energy homeostasis, oxidative metabolism and mitochondrial biogenesis ,while effecting mammalian physiology in the heart, brown adipose tissue, white adipose tissue, placenta, macrophages, and demonstrated additional roles in diabetes and cancer. ERRs bind enhancers throughout the genome where they exert effects on gene regulation Although their overall functions remain uncertain, they also share DNA-binding sites, co-regulators, and target genes with the conventional estrogen receptors ERalpha and ERbeta and may function to modulate estrogen signaling pathways. · ERR-alpha has wide tissue distribution but it is most highly expressed in tissues that preferentially use fatty acids as energy sources such as kidney, heart, brown adipose tissue, cerebellum, intestine, and skeletal muscle. ERRalpha has been detected in normal adrenal cortex tissues, in which its expression is possibly related to adrenal development, with a possible role in fetal adrenal function, in dehydroepiandrosterone (DHEAS) production in adrenarche, and also in steroid production of post-adrenarche/adult life. DHEA and other adrenal androgens such as androstenedione, although relatively weak androgens, are responsible for the androgenic effects of adrenarche, such as early pubic and axillary hair growth, adult-type body odor, increased oiliness of hair and skin, and mild acne. · ERR-beta is a nuclear receptor . Its function is unknown; however, a similar protein in mouse plays an essential role in placental development · ERR-gamma is a nuclear receptor that behaves as a constitutive activator of transcription. There is evidence that bisphenol A functions as an endocrine disruptor by binding strongly to ERRgamma BPA as well as its nitrated and chlorinated metabolites seems to binds strongly to ERR-gamma (dissociation constant = 5.5 nM), but not to the estrogen receptor (ER). BPA binding to ERR-gamma preserves its basal constitutive activity.Different expression of ERR-gamma in different parts of the body may account for variations in bisphenol A effects. For instance, ERR-gamma has been found in high concentration in the placenta, explaining reports of high bisphenol A accumulation there
PHENOL/ FORMALDEHYDE GLYCIDYL ETHER COPOLYMER	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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ZINC OXIDE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
BISPHENOL A DIGLYCIDYL ETHER	Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. The presence of the p-hydroxy group on the benzene rings is though to be responsible for the oestradiol mimicry. . Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review. A 2009 study on Chinese workers in bisphenol A factories found that workers were four times more likely to report erectile dysfunction, reduced sexual desire and overall dissatisfaction with their sex life than workers with no heightened bisphenol A exposure. Bisphenol A workers were also seven times more likely to have ejaculation difficulties. They were also more likely to report reduced sexual function within one year of beginning employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties. Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/ litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades' One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials' One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood. A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells.[whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes. Bisphenol A is the isopropyl adduct of 4,4'-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called 'cytostatic hormones'. Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown to be oestrogenic in vitro; such sealants may represent an additional source of xenoestrogens in humans and may be the cause of additional concerns in children. Concerns have been raised about the possible developmental effects on the foetus/embryo or neonate resulting from the leaching of bisphenol A from epoxy linings in metal cans which come in contact with food-stuffs. Many drugs, including naproxen, salicylic acid, carbamazepine and mefenamic acid can, in vitro, significantly inhibit bisphenol A glucuronidation (detoxification). BPA belongs to the list of compounds having this property as the rodent models have shown that BPA exposure is linked with increased body weigh (obesogens)t. Several mechanisms can help explain the effect of BPA on body weight increase. A possible mechanism leading to triglyceride accumulation is the decreased production of the hormone adiponectin from all human adipose tissue tested when exposed to very low levels (below nanomolar range) of BPA in cell or explant culture settings . The expression of leptin as well as several enzymes and transcription factors is also affected by BPA exposure in vivo as well as in vitro. Together, the altered expression and activity of these important mediators of fat metabolism could explain the increase in weight following BPA exposure in rodent models. These results also suggest that, together with other obesogens, low, environmentally relevant levels of BPA may contribute to the human obesity phenomenon. All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity.. Testicular abnormalities (including testicular atrophy with decreased spermatogenic activity) following exposure to glycidyl ethers have been reported. Haemopoietic abnormalities following exposure to glycidyl ethers, including alteration of the leukocyte count, atrophy of lymphoid tissue, and bone marrow cytotoxicity have also been reported. These abnormalities were usually observed along with pneumonia and/or toxemia, and therefore may be secondary effects. However, especially in light of the generalized reduction in leukocytes and the atrophy of lymphoid tissues, the observed haemopoietic abnormalities may have been predisposing factors to pneumonia. While none of the individual research reports are conclusive with respect to the ability of glycidyl ethers to produce permanent changes to the testes or haemopoietic system in laboratory animals, the pattern of displayed effects is reason for concern Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether. A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not n-butyl glycidyl ether, induced morphological transformation in mammalian cells in vitro. n-Butyl glycidyl ether induced micronuclei in mice in vivo following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations in vivo or chromosomal aberrations in animal cells in vitro. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in Drosophila. The glycidyl ethers were generally mutagenic to bacteria. 55badger 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.
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
8329TFS-A Thermally Conductive Epoxy Adhesive (Part A) & PHENOL/ FORMALDEHYDE GLYCIDYL ETHER COPOLYMER & (C12-14)ALKYLGLYCIDYL ETHER & BISPHENOL A	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.

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DIGLYCIDYL ETHER	
8329TFS-A Thermally Conductive Epoxy Adhesive (Part A) & BISPHENOL A DIGLYCIDYL ETHER	In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg). **Reproductive and Developmental Toxicity**: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg. **Carcinogenicity:** IARC concluded that 'there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals.' Its overall evaluation was 'Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3). In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997). **Genotoxicity**: In S. typhimurium strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg). **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 - **Consumer exposure** to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/ kg/body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weigh/day (male rats) from the 2-year carcinogenicity study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs.
8329TFS-A Thermally Conductive Epoxy Adhesive (Part A) & PHENOL/ FORMALDEHYDE GLYCIDYL ETHER COPOLYMER	The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics. Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities. Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor. In vitro cell models were used to evaluate the ability of 22 bisphenols (BPs) to induce or inhibit estrogenic and androgenic activity. BPA, Bisphenol AF (BPAF), bisphenol Z (BPZ), bisphenol C (BPC), tetramethyl bisphenol A (TMBPA), bisphenol S (BPS), bisphenol E (BPE), 4,4-bisphenol F (4,4-BPF), bisphenol AP (BPAP), bisphenol B (BPB), tetrachlorobisphenol A (TCBPA), and benzylparaben (PHBB) induced estrogen receptor (ER)alpha and/or ERbeta-mediated activity. With the exception of BPS, TCBPA, and PHBB, these same BPs were also androgen receptor (AR) antagonists. Only 3 BPs were found to be ER antagonists. Bisphenol P (BPP) selectively inhibited ERbeta-mediated activity and 4-(4-phenylmethoxyphenyl)sulfonylphenol (BPS-MPE) and 2,4-bisphenol S (2,4-BPS) selectively inhibited ERalpha-mediated activity. None of the BPs induced AR-mediated activity.
8329TFS-A Thermally Conductive Epoxy Adhesive (Part A) & (C12-14)ALKYLGLYCIDYL ETHER & BISPHENOL A DIGLYCIDYL ETHER	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.
ALUMINIUM OXIDE & CARBON BLACK	No significant acute toxicological data identified in literature search.
(C12-14)ALKYLGLYCIDYL ETHER & BISPHENOL A DIGLYCIDYL ETHER	for 1,2-butylene oxide (ethyloxirane): Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/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

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

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

SECTION 12 Ecological information

Toxicity

8329TFS-A Thermally Conductive Epoxy Adhesive (Part A)

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

aluminium oxide

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

phenol/ formaldehyde glycidyl ether copolymer

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

zinc oxide

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	0.005mg/l	2
BCF	1344h	Fish	19-110	7
LC50	96h	Fish	0.927-2.589mg/l	4
EC50	72h	Algae or other aquatic plants	0.036-0.049mg/l	4
EC50	48h	Crustacea	0.301-0.667mg/l	4
EC50	96h	Algae or other aquatic plants	0.3mg/l	2

(C12-14)alkylglycidyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48h	Crustacea	6.07mg/l	2
LC50	96h	Fish	>5000mg/l	2
EC50	48h	Crustacea	6.07mg/l	2

bisphenol A diglycidyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	0.3mg/l	2
EC50	72h	Algae or other aquatic plants	9.4mg/l	2
LC50	96h	Fish	1.2mg/l	2
EC50	48h	Crustacea	1.1mg/l	2

carbon black

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	24h	Crustacea	3200mg/l	1
LC50	96h	Fish	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
EC50	48h	Crustacea	33.076-41.968mg/l	4

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

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.
Liquid epoxy resins and some reactive diluents are not readily biodegradable, although its epoxy functional groups are hydrolysed in contact with water, they have the potential to bio-accumulate and are moderately toxic to aquatic organisms. They are generally classified as dangerous for the environment according to the European Union classification criteria.
Uncured solid resins on the other hand are not readily bio-available, not toxic to aquatic and terrestrial organisms, not readily biodegradable, but hydrolysable.They present no significant hazard for the environment.
For bisphenol A and related bisphenols:
Environmental fate:
Biodegradability (28 d) 89% - Easily biodegradable
Bioconcentration factor (BCF) 7.8 mg/l
Bisphenol A, its derivatives and analogues, can be released from polymers, resins and certain substances by metabolic products
Substance does not meet the criteria for PBT or vPvB according to Regulation (EC) No 1907/2006, Annex XIII
As an environmental contaminant, bisphenol A interferes with nitrogen fixation at the roots of leguminous plants associated with the bacterial symbiont Sinorhizobium meliloti. Despite a half-life in the soil of only 1-10 days, its ubiquity makes it an important pollutant. According to Environment Canada, 'initial assessment shows that at low levels, bisphenol A can harm fish and organisms over time. Studies also indicate that it can currently be found in municipal wastewater.' However, a study conducted in the United States found that 91-98% of

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bisphenol A may be removed from water during treatment at municipal water treatment plants.
Ecotoxicity:
Fish LC50 (96 h): 4.6 mg/l (freshwater fish); 11 mg/l (saltwater fish): NOEC 0.016 mg/l (freshwater fish- 144 d); 0.064 mg/l (saltwater fish 164 d)
Fresh water invertebrates EC50 (48 h): 10.2 mg/l: NOEC 0.025 mg/l - 328 d)
Marine water invertebrate EC50 (96 h): 1.1 mg/l; NOEC 0.17 mg/l (28 d)
Freshwater algae (96 h): 2.73 mg/l
Marine water algae (96 h): 1.1 mg/l
Fresh water plant EC50 (7 d): 20 mg/l: NOEC 7.8 mg/l
In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms.
Among freshwater organisms, fish appear to be the most sensitive species. Evidence of endocrine-related effects in fish, aquatic invertebrates, amphibians and reptiles has been reported at environmentally relevant exposure levels lower than those required for acute toxicity. There is a widespread variation in reported values for endocrine-related effects, but many fall in the range of 1 ug/L to 1 mg/L
A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations.
A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.
Although abundant data are available on the toxicity of bisphenol-A (2,2-bis (4-hydroxydiphenyl)propane;(BPA) A variety of BPs were examined for their acute toxicity against Daphnia magna, mutagenicity, and oestrogenic activity using the Daphtoxkit (Creasel Ltd.), the umu test system, and the yeast two-hybrid system, respectively, in comparison with BPA. BPA was moderately toxic to D. magna (48-h EC50 was 10 mg/l) according to the current U.S. EPA acute toxicity evaluation standard, and it was weakly oestrogenic with 5 orders of magnitude lower activity than that of the natural estrogen 17 beta-oestradiol in the yeast screen, while no mutagenicity was observed. All seven BPs tested here showed moderate to slight acute toxicity, no mutagenicity, and weak oestrogenic activity as well as BPA. Some of the BPs showed considerably higher oestrogenic activity than BPA, and others exhibited much lower activity. Bisphenol S (bis(4-hydroxydiphenyl)sulfone) and bis(4-hydroxyphenyl)sulfide) showed oestrogenic activity.
Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F ([bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem,
Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe3+ ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.

Reactive diluents generally have a low to moderate potential for bioconcentration (tendency to accumulate in the food chain) and a high to very high potential for mobility in soil. Small amounts that escape to the atmosphere will photodegrade.
They would not be expected to persist in the environment.
Most reactive diluents should be considered slightly to moderately toxic to aquatic organisms on an acute basis while some might also be considered harmful to the environment.
Environmental toxicity is a function of the n-octanol/water partition coefficient (log Pow, log Kow). Compounds with log Pow >5 act as neutral organics, but at a lower log Pow, the toxicity of epoxide-containing polymers is greater than that predicted for simple narcotics.
Significant environmental findings are limited. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit common characteristics with respect to environmental fate and ecotoxicology. One such oxirane is ethyloxirane and data presented here may be taken as representative.
for 1,2-butylene oxide (ethyloxirane):
Environmental fate: Ethyloxirane is highly soluble in water and has a very low soil-adsorption coefficient, which suggests that if released to water, adsorption of ethyloxirane to sediment and suspended solids is not expected. Volatilisation of ethyloxirane from water surfaces would be expected based on the moderate estimated Henry's Law constant. If ethyloxirane is released to soil, it is expected to have low adsorption and thus very high mobility. Volatilisation from moist soil and dry soil surfaces is expected, based on its vapour pressure. It is expected that ethyloxirane exists solely as a vapour in ambient atmosphere, based on its very high vapour pressure. Ethyloxirane may also be removed from the atmosphere by wet deposition processes, considering its relatively high water solubility.
Persistence: The half-life in air is about 5.6 days from the reaction of ethyloxirane with photochemically produced hydroxyl radicals which indicates that this chemical meets the persistence criterion in air (half-life of = 2 days)*.
Ethyloxirane is hydrolysable, with a half-life of 6.5 days, and biodegradable up to 100% degradation and is not expected to persist in water. A further model-predicted biodegradation half-life of 15 days in water was obtained and used to predict the half-life of this chemical in soil and sediment by applying Boethling's extrapolation factors (t1/2water : t1/2 soil : t1/2sediment = 1: 1: 4) (Boethling 1995). According to these values, it can be concluded that ethyloxirane does not meet the persistence criteria in water and soil (half-lives = 182 days) and sediments (half-life = 365 days).
Experimental and modelled log Kow values of 0.68 and 0.86, respectively, indicate that the potential for bioaccumulation of ethyloxirane in organisms is likely to be low. Modelled bioaccumulation -factor (BAF) and bioconcentration -factor (BCF) values of 1 to 17 L/kg indicate that ethyloxirane does not meet the bioaccumulation criteria (BCF/BAF = 5000)*
Ecotoxicity:
Experimental ecotoxicological data for ethyloxirane (OECD 2001) indicate low to moderate toxicity to aquatic organisms. For fish and water flea, acute LC50/EC50 values vary within a narrow range of 70-215 mg/L; for algae, toxicity values exceed 500 mg/L, while for bacteria they are close to 5000 mg/L

* *Persistence and Bioaccumulation Regulations* (Canada 2000).
Reactive diluents which are only slightly soluble in water and do not evaporate quickly are expected to sink to the bottom or float to the top, depending on the density, where they would be expected to biodegrade slowly.
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. Overexposures to zinc also have 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

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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 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 in solution. As negative charges on soil surfaces increase with increasing pH, additional sites for zinc adsorption are activated and the amount of zinc in solution 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.
For aluminium and its compounds and salts:
Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals.Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.
Environmental fate:
Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.
Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.
As an element, aluminum cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminum in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminum can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminum in the environment will depend on the ligands present and the pH.
The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion, [Al(H2O)6]3+, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H2O)5(OH)]2+, [Al(H2O)4(OH)2]+). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)2+ and Al(OH)2+, while the solid Al(OH)3 is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)4- is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)3, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.
Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.
Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.
The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.
Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.
Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.
Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.
The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.
Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.
Ecotoxicity:
Freshwater species pH >6.5
Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar)* - 106 mg/L; Chronic NOEC (8-28 d): 7 spp,NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.
Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L
Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L
Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L
Freshwater species pH <6.5 (all between pH 4.5 and 6.0)
Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L
Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)
Alga: 1 sp NOEC growth 2.0 mg/L
Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.
The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4.5.4, with a maximum toxicity occurring around pH 5.0.5.2. The inorganic single unit aluminium species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.
Drinking Water Standards:
aluminium: 200 ug/l (UK max.)
200 ug/l (WHO guideline)

Continued...

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.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
zinc oxide	LOW (BCF = 217)
bisphenol A diglycidyl ether	MEDIUM (LogKOW = 3.8446)

Mobility in soil

Ingredient	Mobility
bisphenol A diglycidyl ether	LOW (KOC = 1767)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Waste Management Production waste from epoxy resins and resin systems should be treated as hazardous waste in accordance with National regulations. Fire retarded resins containing halogenated compounds should also be treated as special waste. Accidental spillage of resins, curing agents and their formulations should be contained and absorbed by special mineral absorbents to prevent them from entering the environment. Contaminated or surplus product should not be washed down the sink, but preferably be fully reacted to form cross-linked solids which is non-hazardous and can be more easily disposed. Finished articles made from fully cured epoxy resins are hard, infusible solids presenting no hazard to the environment. However, finished articles from flame-retarded material containing halogenated resins should be considered hazardous waste, and disposed as required by National laws. Articles made from epoxy resins, like other thermosets, can be recycled by grinding and used as fillers in other products. Another way of disposal and recovery is combustion with energy recovery. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. Removal of bisphenol A (BPA) from aqueous solutions was accomplished by adsorption of enzymatically generated quinone derivatives on chitosan beads. The use of chitosan in the form of beads was found to be more effective because heterogeneous removal of BPA with chitosan beads was much faster than homogeneous removal of BPA with chitosan solutions, and the removal efficiency was enhanced by increasing the amount of chitosan beads dispersed in the BPA solutions and BPA was completely removed by quinone adsorption in the presence of chitosan beads more than 0.10 cm3/cm3. In addition, a variety of bisphenol derivatives were completely or effectively removed by the procedure constructed in this study, although the enzyme dose or the amount of chitosan beads was further increased as necessary for some of the bisphenol derivatives used. M. Suzuki, and E Musashi J Appl Polym Sci, 118(2):721 - 732; October 2010 ▸ 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.

SECTION 14 Transport information

Labels Required

Continued...

	NOT REGULATED by Ground ADR Special Provision (SP AU01) - ADG Code 7th Ed. NOT REGULATED by Air IATA Special Provision A197 NOT REGULATED by Sea IMDG per 2.10.2.7

Land transport (ADG)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc oxide)
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

UN number	3082
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains zinc oxide)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A97 A158 A197 A215 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 Passenger and Cargo Maximum Qty / Pack: 450 L Passenger and Cargo Limited Quantity Packing Instructions: Y964 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc oxide)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-A , S-F Special provisions: 274 335 969 Limited Quantities: 5 L

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

Not Applicable

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

Product name	Group
aluminium oxide	Not Available
phenol/ formaldehyde glycidyl ether copolymer	Not Available
zinc oxide	Not Available
(C12-14)alkylglycidyl ether	Not Available

Continued...

Product name	Group
bisphenol A diglycidyl ether	Not Available
carbon black	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
aluminium oxide	Not Available
phenol/ formaldehyde glycidyl ether copolymer	Not Available
zinc oxide	Not Available
(C12-14)alkylglycidyl ether	Not Available
bisphenol A diglycidyl ether	Not Available
carbon black	Not Available

SECTION 15 Regulatory information

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

aluminium oxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

phenol/ formaldehyde glycidyl ether copolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

zinc oxide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)

(C12-14)alkylglycidyl ether is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

bisphenol A diglycidyl ether is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

carbon black is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (aluminium oxide; phenol/ formaldehyde glycidyl ether copolymer; (C12-14)alkylglycidyl ether; bisphenol A diglycidyl ether; carbon black)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (phenol/ formaldehyde glycidyl ether copolymer; (C12-14)alkylglycidyl ether)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No ((C12-14)alkylglycidyl ether; bisphenol A diglycidyl ether)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	19/01/2022

Continued...

Initial Date	30/03/2016

SDS Version Summary

Version	Date of Update	Sections Updated
7.18	19/01/2022	Chronic Health, Classification, Environmental, Physical Properties

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

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

8329TFS-B Thermally Conductive Epoxy Adhesive

Mektronics

Version No: **4.6**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **19/01/2022**
Print Date: **19/01/2022**
L.GHS.AUS.EN

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

Product Identifier

Product name	8329TFS-B
Synonyms	SDS Code: 8329TFS-Part B; 8329TFS-25ML, 8329TFS-50ML
Other means of identification	Thermally Conductive Epoxy Adhesive

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

Relevant identified uses	Thermally conductive adhesive for bonding and thermal management

Details of the supplier of the safety data sheet

Registered company name	**Mektronics**	**MG Chemicals (Head office)**
Address	Unit 3 8 Bonz Place, Seven Hills NSW 2147 Australia	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	1300 788 701	+(1) 800-201-8822
Fax	1300 722 004	+(1) 800-708-9888
Website	www.mektronics.com.au	www.mgchemicals.com
Email	sales@mektronics.com.au	Info@mgchemicals.com

Emergency telephone number

Association / Organisation	**Verisk 3E (Access Code: 335388)**
Emergency telephone numbers	+61 1 800 686 951
Other emergency telephone numbers	+61 280363166

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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

Hazard statement(s)

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

Precautionary statement(s) Prevention

P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.

Page 1 continued...

P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water 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 to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1344-28-1.	39	aluminium oxide
1314-13-2	25	zinc oxide
68541-13-9	18	linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid
68082-29-1*	9	tall oil/ triethylenetetramine polyamides
4246-51-9	3	diethylene glycol, di(3-aminopropyl) ether
108-65-6	1	propylene glycol monomethyl ether acetate, alpha-isomer
112-24-3	<1	triethylenetetramine
1333-86-4	0.5	carbon black
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ 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	If skin or hair contact occurs: ▸ Quickly but gently, wipe material off skin with a dry, clean cloth. ▸ Immediately remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

Continued...

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

Extinguishing media

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

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

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Use 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	‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) nitrogen oxides (NOx) metal oxides other pyrolysis products typical of burning organic material. When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ‣ 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.

Continued...

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

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ 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
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate. Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt. 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

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Continued...

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	aluminium oxide	Aluminium oxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	zinc oxide	Zinc oxide (dust)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	zinc oxide	Zinc oxide (fume)	5 mg/m3	10 mg/m3	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
aluminium oxide	15 mg/m3	170 mg/m3	990 mg/m3
zinc oxide	10 mg/m3	15 mg/m3	2,500 mg/m3
diethylene glycol, di(3-aminopropyl) ether	13 mg/m3	140 mg/m3	850 mg/m3
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available	Not Available
triethylenetetramine	3 ppm	14 ppm	83 ppm
carbon black	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
aluminium oxide	Not Available	Not Available
zinc oxide	500 mg/m3	Not Available
linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid	Not Available	Not Available
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	1,750 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid	E	≤ 0.1 ppm
tall oil/ triethylenetetramine polyamides	E	≤ 0.1 ppm
diethylene glycol, di(3-aminopropyl) ether	C	> 1 to ≤ 10 parts per million (ppm)
triethylenetetramine	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

for 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
These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:

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D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996)
The concentration of dust, for application of respirable dust limits, is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative log-normal function with a median aerodynamic diameter of 4.0 um (+-) 0.3 um and with a geometric standard deviation of 1.5 um (+-) 0.1 um, i.e..generally less than 5 um.
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.

Exposure controls

Appropriate engineering controls

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

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

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

Within each range the appropriate value depends on:

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

Personal protection

Eye and face protection

- 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

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

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	The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
'Forsberg Clothing Performance Index'.
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
8329TFS-B Thermally Conductive Epoxy Adhesive

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

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment

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(powered, positive flow, full face apparatus may be an option).
· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.
Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.
P2 is a respiratory filter rating under various international standards, Filters at least 94% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Grey

Physical state	Liquid	**Relative density (Water = 1)**	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)	>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	Immiscible	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	‣ Unstable in the presence of incompatible materials. ‣ Product is considered stable. ‣ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

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Inhaled	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. Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Inhalation of epoxy resin amine hardener vapours (including polyamines and amine adducts) may produce bronchospasm and coughing episodes lasting days after cessation of the exposure. Even faint traces of these vapours may trigger an intense reaction in individuals showing 'amine asthma'. The literature records several instances of systemic intoxications following the use of amines in epoxy resin systems. Excessive exposure to the vapours of epoxy amine curing agents may cause both respiratory irritation and central nervous system depression. Signs and symptoms of central nervous system depression, in order of increasing exposure, are headache, dizziness, drowsiness, and incoordination. In short, a single prolonged (measured in hours) or excessive inhalation exposure may cause serious adverse effects, including death. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation.
Ingestion	Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. Ingestion of amine epoxy-curing agents (hardeners) may cause severe abdominal pain, nausea, vomiting or diarrhoea. The vomitus may contain blood and mucous. If death does not occur within 24 hours there may be an improvement in the patients condition for 2-4 days only to be followed by the sudden onset of abdominal pain, board-like abdominal rigidity or hypo-tension; this indicates that delayed gastric or oesophageal corrosive damage has occurred. Acute toxic responses to aluminium are confined to the more soluble forms. 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. Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material may accentuate any pre-existing dermatitis condition 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. Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur. Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions. Individuals exhibiting 'amine dermatitis' may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis. NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Repeated or excessive handling, coupled with poor personal hygiene, may result in acne-like eruptions known as 'zinc oxide pox'. The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: ▸ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non allergic). 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.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.

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Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers
Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.
Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.
Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.
Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.
Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed
at periods up to 12 months following the last exposure.
When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.
The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.
Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.
There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.
Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.
Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.
After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.
At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.
Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer s disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.
Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.
Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of 'tau' a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).
Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [*Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995*]
Following an oral intake of extremely high doses of zinc (where 300 mg Zn/d – 20 times the US Recommended Dietary Allowance (RDA) – is a 'low intake' overdose), nausea, vomiting, pain, cramps and diarrhea may occur. There is evidence of induced copper deficiency, alterations of blood lipoprotein levels, increased levels of LDL, and decreased levels of HDL at long-term intakes of 100 mg Zn/d. The USDA RDA is 15 mg Zn/d.
There is also a condition called the 'zinc shakes' or 'zinc chills' or metal fume fever that can be induced by the inhalation of freshly formed zinc

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oxide formed during the welding of galvanized materials.
Supplemental zinc can prevent iron absorption, leading to iron deficiency and possible peripheral neuropathy, with loss of sensation in extremities.
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.
Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur.
Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions.
Individuals exhibiting 'amine dermatitis' may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis.
NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided.

Sensitisation may give severe responses to very low levels of exposure, in situations where exposure may occur.

8329TFS-B Thermally Conductive Epoxy Adhesive

TOXICITY	IRRITATION
Not Available	Not Available

aluminium oxide

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

zinc oxide

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) : 500 mg/24 h - mild
Inhalation(Rat) LC50; >1.79 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50; >5000 mg/kg[1]	Skin (rabbit) : 500 mg/24 h- mild
	Skin: no adverse effect observed (not irritating)[1]

linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid

TOXICITY	IRRITATION
Not Available	Not Available

tall oil/ triethylenetetramine polyamides

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Not Available
Oral (Rat) LD50; >2000 mg/kg[1]	

diethylene glycol, di(3-aminopropyl) ether

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 2500 mg/kg[2]	Not Available
Oral (Rat) LD50; 4290 mg/kg[2]	

propylene glycol monomethyl ether acetate, alpha-isomer

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50; 3739 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

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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] / Eye: no adverse effect observed (not irritating)[1] Oral (Rat) LD50; >8000 mg/kg[1] / Skin: no adverse effect observed (not irritating)[1]
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

8329TFS-B Thermally Conductive Epoxy Adhesive

For aluminium compounds:
Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate).
Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability.
For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe.
Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium.
The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account
Systemic toxicity after repeated exposure
No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium.
When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose.
The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent
Reproductive and developmental toxicity:
Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity
High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI.
Genotoxicity
Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.
Carcinogenicity.
The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to

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	humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons. Neurodegenerative diseases. Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease.” There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases.Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment. Contact sensitivity: It has been suggested that the body burden of aluminium may be linked to different iseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome. Aluminium acts not only as an adjuvant,stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitisers causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response.Once inside the skin, the metal ions must bind to proteins to become immunologically reactive.The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminium Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste
DIETHYLENE GLYCOL, DI(3-AMINOPROPYL) ETHER	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.
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	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.I] *Shin-Etsu SDS for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. 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 exhibits 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 sensitisers. 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

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	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.I]
TRIETHYLENETETRAMINE	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. 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 Triethylenetetramine (TETA) is a severe irritant to skin and eyes and induces skin sensitisation. TETA is of moderate acute toxicity: LD50(oral, rat) > 2000 mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure to saturated vapour via inhalation was tolerated without impairment. Exposure to 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 fetus (69,2 %) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significant the fetal 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	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
8329TFS-B Thermally Conductive Epoxy Adhesive & 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.
8329TFS-B Thermally Conductive Epoxy Adhesive & LINOLEIC ACID/4,7,10-	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.

Continued...

TRIOXA-1,13-TRIDECANEDIAMINE POLYAMID	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 Amphoterics 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 II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II 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 in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonella 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 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole. 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.
ALUMINIUM OXIDE & LINOLEIC ACID/4,7,10-TRIOXA-1,13-TRIDECANEDIAMINE POLYAMID & CARBON BLACK	No significant acute toxicological data identified in literature search.
ZINC OXIDE & DIETHYLENE GLYCOL, DI(3-AMINOPROPYL) ETHER	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.
DIETHYLENE GLYCOL, DI(3-AMINOPROPYL) ETHER & TRIETHYLENETETRAMINE	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.

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

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

SECTION 12 Ecological information

Toxicity

8329TFS-B Thermally Conductive Epoxy Adhesive

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

aluminium oxide

Endpoint	Test Duration (hr)	Species	Value	Source

Continued...

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

zinc oxide

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	0.005mg/l	2
BCF	1344h	Fish	19-110	7
LC50	96h	Fish	0.927-2.589mg/l	4
EC50	72h	Algae or other aquatic plants	0.036-0.049mg/l	4
EC50	48h	Crustacea	0.301-0.667mg/l	4
EC50	96h	Algae or other aquatic plants	0.3mg/l	2

linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

tall oil/ triethylenetetramine polyamides

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	0.5mg/l	2
LC50	96h	Fish	7.07mg/l	2
EC50	72h	Algae or other aquatic plants	4.34mg/l	2
EC50	48h	Crustacea	7.07mg/l	2

diethylene glycol, di(3-aminopropyl) ether

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	Not Available	Crustacea	>1mg/l	2
LC50	96h	Fish	>215<464mg/l	2
EC50	72h	Algae or other aquatic plants	>500mg/l	2
EC50	48h	Crustacea	218.16mg/l	2

propylene glycol monomethyl ether acetate, alpha-isomer

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	336h	Fish	47.5mg/l	2
LC50	96h	Fish	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>1000mg/l	2
EC50	48h	Crustacea	373mg/l	2
EC50	96h	Algae or other aquatic plants	>1000mg/l	2

triethylenetetramine

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	180mg/l	1
EC50	48h	Crustacea	31.1mg/l	1
EC10(ECx)	72h	Algae or other aquatic plants	0.67mg/l	1
BCF	1008h	Fish	<0.5	7
EC50	72h	Algae or other aquatic plants	2.5mg/l	1
ErC50	72h	Algae or other aquatic plants	2.5mg/l	1

carbon black

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	24h	Crustacea	3200mg/l	1
LC50	96h	Fish	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
EC50	48h	Crustacea	33.076-41.968mg/l	4

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

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.

Continued...

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. Overexposures to zinc also have 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 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 in solution. As negative charges on soil surfaces increase with increasing pH, additional sites for zinc adsorption are activated and the amount of zinc in solution 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.

For aluminium and its compounds and salts:

Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals.Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.

Environmental fate:

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

Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.

As an element, aluminum cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminum in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminum can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminum in the environment will depend on the ligands present and the pH.

The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion, [Al(H2O)6]3+, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H2O)5(OH)]2+, [Al(H2O)4(OH)2]+). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)2+ and Al(OH)2+, while the solid Al(OH)3 is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)4- is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)3, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.

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

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

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

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

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

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

The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs

Continued...

ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.
Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail.
Bioconcentration of aluminum has also been reported for aquatic insects.
Ecotoxicity:
Freshwater species pH >6.5
Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar)* - 106 mg/L; Chronic NOEC (8-28 d): 7 spp,NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.
Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L
Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L
Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L
Freshwater species pH <6.5 (all between pH 4.5 and 6.0)
Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L
Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)
Alga: 1 sp NOEC growth 2.0 mg/L
Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates.
Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.
The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4.5.4, with a maximum toxicity occurring around pH 5.0.5.2. The inorganic single unit aluminium species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.
Drinking Water Standards:
aluminium: 200 ug/l (UK max.)
200 ug/l (WHO guideline)
chloride: 400 mg/l (UK max.)
250 mg/l (WHO guideline)
fluoride: 1.5 mg/l (UK max.)
1.5 mg/l (WHO guideline)
nitrate: 50 mg/l (UK max.)
50 mg/l (WHO guideline)
sulfate: 250 mg/l (UK max.)
Soil Guideline: none available.
Air Quality Standards: none available.
DO NOT discharge into sewer or waterways.

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

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 (BCF = 5)

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)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction

Continued...

	▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible 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.

SECTION 14 Transport information

Labels Required

	NOT REGULATED by Ground ADR Special Provision (SP AU01) - ADG Code 7th Ed. NOT REGULATED by Air IATA Special Provision A197 NOT REGULATED by Sea IMDG per 2.10.2.7

Land transport (ADG)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc oxide)
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

UN number	3082
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains zinc oxide)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A97 A158 A197 A215 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 Passenger and Cargo Maximum Qty / Pack: 450 L Passenger and Cargo Limited Quantity Packing Instructions: Y964 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains zinc oxide)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant

Continued...

Special precautions for user	EMS Number	F-A , S-F
	Special provisions	274 335 969
	Limited Quantities	5 L

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

Not Applicable

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

Product name	Group
aluminium oxide	Not Available
zinc oxide	Not Available
linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid	Not Available
tall oil/ triethylenetetramine polyamides	Not Available
diethylene glycol, di(3-aminopropyl) ether	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
triethylenetetramine	Not Available
carbon black	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
aluminium oxide	Not Available
zinc oxide	Not Available
linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid	Not Available
tall oil/ triethylenetetramine polyamides	Not Available
diethylene glycol, di(3-aminopropyl) ether	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
triethylenetetramine	Not Available
carbon black	Not Available

SECTION 15 Regulatory information

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

aluminium oxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

zinc oxide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

tall oil/ triethylenetetramine polyamides is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

diethylene glycol, di(3-aminopropyl) ether is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

propylene glycol monomethyl ether acetate, alpha-isomer is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

triethylenetetramine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

Australian Inventory of Industrial Chemicals (AIIC)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

carbon black is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australian Inventory of Industrial Chemicals (AIIC)	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
Chemical Footprint Project - Chemicals of High Concern List	International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (aluminium oxide; linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid; tall oil/ triethylenetetramine polyamides; propylene glycol monomethyl ether acetate, alpha-isomer; triethylenetetramine; carbon black)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid)
Japan - ENCS	No (linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid; tall oil/ triethylenetetramine polyamides)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid; diethylene glycol, di(3-aminopropyl) ether)
Vietnam - NCI	No (linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid)
Russia - FBEPH	No (linoleic acid/4,7,10-trioxa-1,13-tridecanediamine polyamid; tall oil/ triethylenetetramine polyamides)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	19/01/2022
Initial Date	30/03/2019

SDS Version Summary

Version	Date of Update	Sections Updated
3.6	19/01/2022	Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Chronic Health, Classification, Exposure Standard, First Aid (inhaled), First Aid (skin), First Aid (swallowed), Ingredients, Personal Protection (Respirator), Physical Properties

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

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

Continued...

INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

end of SDS