843ER-A Super Shield™ Silver Coated Copper Epoxy Conductive Coating (Part A)

MG Chemicals UK Limited

Version No: **A-1.02**
Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date:**02/05/2018**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

1.1. Product Identifier

Product name	843ER-A
Synonyms	SDS Code: 843ER-Part A; 843ER-250ML, 843ER-800ML, 843ER-3.25L
Other means of identification	Super Shield™ Silver Coated Copper Epoxy Conductive Coating (Part A)

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

Relevant identified uses	Electrically conductive epoxy coating resin for use with hardeners
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

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

1.4. Emergency telephone number

Association / Organisation	**Verisk 3E (Access code: 335388)**	**Not Available**
Emergency telephone numbers	+(44) 20 35147487	Not Available
Other emergency telephone numbers	+(0) 800 680 0425	Not Available

SECTION 2 HAZARDS IDENTIFICATION

2.1.

Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	H225 - Flammable Liquid Category 2, H315 - Skin Corrosion/Irritation Category 2, H318 - Serious Eye Damage Category 1, H317 - Skin Sensitizer Category 1, H336 - Specific target organ toxicity - single exposure Category 3 (narcotic effects), H410 - Chronic Aquatic Hazard Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

2.2. Label elements

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

Hazard statement(s)

H225	Highly flammable liquid and vapour.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H317	May cause an allergic skin reaction.
H336	May cause drowsiness or dizziness.
H410	Very toxic to aquatic life with long lasting effects.

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Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P271	Use in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.

Precautionary statement(s) Disposal

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

2.3. Other hazards

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.78-93-3 2.201-159-0 3.606-002-00-3 4.01-2119457290-43-XXXX|01-2119943742-35-XXXX	42	methyl ethyl ketone *	Flammable Liquid Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Eye Irritation Category 2; H225, H336, H319, EUH066 [2]
1.7440-50-8 2.231-159-6 3.Not Available 4.01-2119475516-31-XXXX|01-2119480154-42-XXXX|01-2119480184-39-XXXX|01-2120762783-45-XXXX	22	copper	EUH210 [1]
1.25068-38-6 2.500-033-5 3.603-074-00-8 4.01-2119456619-26-XXXX	19	bisphenol A diglycidyl ether resin, solid	Eye Irritation Category 2, Chronic Aquatic Hazard Category 2, Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H319, H411, H317, H315 [2]
1.71-36-3 2.200-751-6 3.603-004-00-6 4.01-2119484630-38-XXXX|01-2120076484-50-XXXX	5	n-butanol	Flammable Liquid Category 3, Acute Toxicity (Oral) Category 4, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation); H226, H302, H336, H315, H318, H335 [2]

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1.67-63-0 2.200-661-7 3.603-117-00-0 4.01-2119457558-25-XXXX	5	isopropanol	Flammable Liquid Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Eye Irritation Category 2; H225, H336, H319 [2]
1.7440-22-4 2.231-131-3 3.Not Available 4.01-2119513211-60-XXXX|01-2119555669-21-XXXX	3	silver	EUH210 [1]
1.14807-96-6 2.238-877-9 3.Not Available 4.01-2120140278-58-XXXX	2	talc	Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Acute Toxicity (Inhalation) Category 4; H335, H332 [1]
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available*		

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

Eye Contact	If this product comes in contact with eyes: ▸ Wash out immediately with water. ▸ If irritation continues, seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

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

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
53ag
To treat poisoning by the higher aliphatic alcohols (up to C7):

- Gastric lavage with copious amounts of water.
- It may be beneficial to instill 60 ml of mineral oil into the stomach.
- Oxygen and artificial respiration as needed.
- Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5)

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for shock.
- Monitor and treat, where necessary, for pulmonary oedema.
- Anticipate and treat, where necessary, for seizures.
- **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Acidosis may respond to hyperventilation and bicarbonate therapy.

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- Haemodialysis might be considered in patients with severe intoxication.
- Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.
Symptomatic and supportive therapy is advised in managing patients.

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

Metal dust fires need to be smothered with sand, inert dry powders.
DO NOT USE WATER, CO2 or FOAM.

- Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- Chemical reaction with CO2 may produce flammable and explosive methane.
- If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- **DO NOT** use halogenated fire extinguishing agents.

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Reacts with acids producing flammable / explosive hydrogen (H2) gas ▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

5.3. Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Consider evacuation (or protect in place). ▸ Fight fire from a safe distance, with adequate cover. ▸ If safe, switch off electrical equipment until vapour fire hazard removed. ▸ Use water delivered as a fine spray to control the 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	▸ **DO NOT** disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ▸ **DO NOT** use water or foam as generation of explosive hydrogen may result. With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present. Metal powders, while generally regarded as non-combustible: ▸ May burn when metal is finely divided and energy input is high. ▸ May react explosively with water. ▸ May be ignited by friction, heat, sparks or flame. ▸ May **REIGNITE** after fire is extinguished. ▸ Will burn with intense heat. Note: ▸ Metal dust fires are slow moving but intense and difficult to extinguish. ▸ Containers may explode on heating. ▸ Dusts or fumes may form explosive mixtures with air. ▸ Gases generated in fire may be poisonous, corrosive or irritating. ▸ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▸ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids ▸ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning. Combustion products include: carbon dioxide (CO2) aldehydes other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions.

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	▸ Remove all ignition sources. ▸ 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 small quantities with vermiculite or other absorbent material. ▸ Wipe up. ▸ Collect residues in a flammable waste container.

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Major Spills

Chemical Class: alcohols and glycols
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R,I, P
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
foamed glass - pillow	4	throw	pichfork	R, P, DGC, RT
LAND SPILL - MEDIUM				
cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
polypropylene - particulate	2	blower	skiploader	W, SS, DGC
sorbent clay - particulate	2	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	3	throw	skiploader	DGC, RT
expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC
polyurethane - mat	4	throw	skiploader	DGC, RT

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

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

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

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- Consider evacuation (or protect in place).
- No smoking, naked lights or ignition sources.
- Increase ventilation.
- Stop leak if safe to do so.
- Water spray or fog may be used to disperse /absorb vapour.

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	▸ Contain spill with sand, earth or vermiculite. ▸ Use only spark-free shovels and explosion proof equipment. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ If contamination of drains or waterways occurs, advise emergency services.

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE

7.1. Precautions for safe handling

Safe handling	▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. **Contains low boiling substance:** Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately. ▸ Check for bulging containers. ▸ Vent periodically ▸ Always release caps or seals slowly to ensure slow dissipation of vapours ▸ 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, heat or ignition sources. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Vapour may ignite on pumping or pouring due to static electricity. ▸ **DO NOT** use plastic buckets. ▸ Earth and secure metal containers when dispensing or pouring product. ▸ Use spark-free tools when handling. ▸ Avoid contact with incompatible materials. ▸ Keep containers securely sealed. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin
Fire and explosion protection	See section 5
Other information	▸ Store in original containers in approved flame-proof area. ▸ No smoking, naked lights, heat or ignition sources. ▸ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped. ▸ Keep containers securely sealed. ▸ Store away from incompatible materials in a cool, dry well ventilated area. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	▸ Packing as supplied by manufacturer. ▸ Plastic containers may only be used if approved for flammable liquid. ▸ Check that containers are clearly labelled and free from leaks. ▸ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▸ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▸ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▸ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▸ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▸ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	Methyl ethyl ketone: ▸ reacts violently with strong oxidisers, aldehydes, nitric acid, perchloric acid, potassium tert-butoxide, oleum ▸ is incompatible with inorganic acids, aliphatic amines, ammonia, caustics, isocyanates, pyridines, chlorosulfonic aid ▸ forms unstable peroxides in storage, or on contact with propanol or hydrogen peroxide ▸ attacks some plastics ▸ may generate electrostatic charges, due to low conductivity, on flow or agitation ▸ 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 ▸ Silver or silver salts readily form explosive silver fulminate in the presence of both nitric acid and ethanol. The resulting fulminate is much more sensitive and a more powerful detonator than mercuric fulminate. ▸ Silver and its compounds and salts may also form explosive compounds in the presence of acetylene and nitromethane. ▸ Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.

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Alcohols
- are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents.
- reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen
- react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium
- should not be heated above 49 deg. C. when in contact with aluminium equipment

Ketones in this group:
- are reactive with many acids and bases liberating heat and flammable gases (e.g., H2).
- react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H2) and heat.
- are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides.
- react violently with aldehydes, HNO3 (nitric acid), HNO3 + H2O2 (mixture of nitric acid and hydrogen peroxide), and HClO4 (perchloric acid).
- may react with hydrogen peroxide to form unstable peroxides; many are heat- and shock-sensitive explosives.

A significant property of most ketones is that the hydrogen atoms on the carbons next to the carbonyl group are relatively acidic when compared to hydrogen atoms in typical hydrocarbons. Under strongly basic conditions these hydrogen atoms may be abstracted to form an enolate anion. This property allows ketones, especially methyl ketones, to participate in condensation reactions with other ketones and aldehydes. This type of condensation reaction is favoured by high substrate concentrations and high pH (greater than 1 wt% NaOH).

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

Metals exhibit varying degrees of activity. Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but:
- can react exothermically with oxidising acids to form noxious gases.
- catalyse polymerisation and other reactions, particularly when finely divided
- react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds.

- Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxide formation on exposure to air.
- Safe handling is possible in relatively low concentrations of oxygen in an inert gas.
- Several pyrophoric metals, stored in glass bottles have ignited when the container is broken on impact. Storage of these materials moist and in metal containers is recommended.
- The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric.

Factors influencing the pyrophoricity of metals are particle size, presence of moisture, nature of the surface of the particle, heat of formation of the oxide, or nitride, mass, hydrogen content, stress, purity and presence of oxide, among others.
- Many metals in elemental form react exothermically with compounds having active hydrogen atoms (such as acids and water) to form flammable hydrogen gas and caustic products.
- Elemental metals may react with azo/diazo compounds to form explosive products.
- Some elemental metals form explosive products with halogenated hydrocarbons.

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	methyl ethyl ketone	Butanone	200 ppm / 600 mg/m3	900 mg/m3 / 300 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	methyl ethyl ketone	Butan-2-one (methyl ethyl ketone)	200 ppm / 600 mg/m3	899 mg/m3 / 300 ppm	Not Available	Sk, BMGV
UK Workplace Exposure Limits (WELs)	copper	Copper fume (as Cu)	0.2 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	n-butanol	Butan-1-ol	Not Available	154 mg/m3 / 50 ppm	Not Available	Sk
UK Workplace Exposure Limits (WELs)	isopropanol	Propan-2-ol	400 ppm / 999 mg/m3	1250 mg/m3 / 500 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	silver	Silver, metallic	0.1 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	talc	Talc, respirable dust	1 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
methyl ethyl ketone	Butanone, 2-; (Methyl ethyl ketone; MEK)	Not Available	Not Available	Not Available

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copper	Copper	3 mg/m3	33 mg/m3	200 mg/m3
bisphenol A diglycidyl ether resin, solid	Epoxy resin includes EPON 1001, 1007, 820, ERL-2795	90 mg/m3	990 mg/m3	5,900 mg/m3
bisphenol A diglycidyl ether resin, solid	Polypropylene glycol, (chloromethyl) oxirane polymer	30 mg/m3	330 mg/m3	2,000 mg/m3
n-butanol	Butyl alcohol, n-; (n-Butanol)	60 ppm	800 ppm	8000 ppm
isopropanol	Isopropyl alcohol	400 ppm	2000 ppm	12000 ppm
silver	Silver	0.3 mg/m3	170 mg/m3	990 mg/m3
talc	Talc	6 mg/m3	66 mg/m3	400 mg/m3

Ingredient	Original IDLH	Revised IDLH
methyl ethyl ketone	3,000 ppm	Not Available
copper	100 mg/m3	Not Available
bisphenol A diglycidyl ether resin, solid	Not Available	Not Available
n-butanol	1,400 ppm	Not Available
isopropanol	2,000 ppm	Not Available
silver	10 mg/m3	Not Available
talc	1,000 mg/m3	Not Available

MATERIAL DATA

For talc (a form of magnesium silicate):
Most health problems associated with occupational exposure to talcs appear to evolve mostly from the nonplatiform content of the talc being mined or milled (being the asbestos-like amphiboles, serpentines (asbestiformes) and other minerals in the form of acicular, prismatic and fibrous crystals including, possibly, asbestos).
Because of severe health effects associated with exposures to asbestos, regulatory agencies tend to regard all elongate mineral crystal particles, whether prismatic, acicular, fibrous, as asbestos - the only provision is the particles have an aspect ratio (length to diameter) of 3:1 or greater.
Consideration is also given to their respirability, their width being less than or equal to 3 um. Only limited data, however, exists on the health effects of elongate mineral particles having prismatic, acicular or fibrous (non-asbestos) forms. Experimental evidence indicates that the carcinogen potential of mineral fibres is related to the size class with diameter of 8 um with shorter, thicker particles having little biological activity.
Dust of nonfibrous talc, consisting entirely of platiform talc crystals and containing no asbestos poses a relatively small respiratory hazard.
Difficulties exist, however, in the determination of asbestos as cleavage fragments of prismatic or acicular crystals, nonasbestos fibres and asbestos fibres are very similar.
Subject to an accurate determination of asbestos and crystalline silica, exposure at or below the recommended TLV-TWA, is thought to protect workers from the significant risk of nonmalignant respiratory effects associated with talc dusts.

The adopted TLV-TWA for silver dust and fumes is 0.1 mg/m3 and for the more toxic soluble silver compounds the adopted value is 0.01 mg/m3. Cases of argyria (a slate to blue-grey discolouration of epithelial tissues) have been recorded when workers were exposed to silver nitrate at concentrations of 0.1 mg/m3 (as silver). Exposure to very high concentrations of silver fume has caused diffuse pulmonary fibrosis. Percutaneous absorption of silver compounds is reported to have resulted in allergy. Based on a 25% retention upon inhalation and a 10 m3/day respiratory volume, exposure to 0.1 mg/m3 (TWA) would result in total deposition of no more than 1.5 gms in 25 years.
For methyl ethyl ketone:
Odour Threshold Value: Variously reported as 2 ppm and 4.8 ppm
Odour threshold: 2 ppm (detection); 5 ppm (recognition) 25 ppm (easy recognition); 300 ppm IRRITATING
Exposures at or below the recommended TLV-TWA are thought to prevent injurious systemic effects and to minimise objections to odour and irritation. Where synergism or potentiation may occur stringent control of the primary toxin (e.g. n-hexane or methyl butyl ketone) is desirable and additional consideration should be given to lowering MEK exposures.
Odour Safety Factor(OSF)
OSF=28 (METHYL ETHYL KETONE)

For n-butanol:
Odour Threshold Value: 0.12-3.4 ppm (detection), 1.0-3.5 ppm (recognition)
NOTE: Detector tubes for n-butanol, measuring in excess of 5 ppm are commercially available.
Exposure at or below the TLV-TWA is thought to provide protection against hearing loss due to vestibular and auditory nerve damage in younger workers and to protect against the significant risk of headache and irritation.
25 ppm may produce mild irritation of the respiratory tract 50 ppm may produce headache and vertigo.
Higher concentrations may produce marked irritation, sore throat, coughing, nausea, shortness of breath, pulmonary injury and central nervous system depression characterised by headache, dizziness, dullness and drowsiness.
6000 ppm may produce giddiness, prostration, narcosis, ataxia, and death.
Odour Safety Factor (OSF)
OSF=60 (n-BUTANOL)

Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)
Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

8.2. Exposure controls

8.2.1. Appropriate engineering controls	Metal dusts must be collected at the source of generation as they are potentially explosive. ▸ Avoid ignition sources. ▸ Good housekeeping practices must be maintained. ▸ Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions. ▸ Do not use compressed air to remove settled materials from floors, beams or equipment ▸ Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. ▸ Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations. ▸ Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. ▸ Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium. ▸ Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible. ▸ Wet scrubbers are preferable to dry dust collectors. ▸ Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.

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- Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states.
- Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec.
- Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts.

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:
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

8.2.2. Personal protection

Eye and face protection

- Safety glasses with side shields.
- 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.

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	When handling liquid-grade epoxy resins wear chemically protective gloves , boots and aprons. The performance, based on breakthrough times ,of: · Ethyl Vinyl Alcohol (EVAL laminate) is generally excellent · Butyl Rubber ranges from excellent to good · Nitrile Butyl Rubber (NBR) from excellent to fair. · Neoprene from excellent to fair · Polyvinyl (PVC) from excellent to poor As defined in ASTM F-739-96 · Excellent breakthrough time > 480 min · Good breakthrough time > 20 min · Fair breakthrough time < 20 min · Poor glove material degradation Gloves should be tested against each resin system prior to making a selection of the most suitable type. Systems include both the resin and any hardener, individually and collectively) · **DO NOT** use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin). · **DO NOT** use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use. Replacement time should be considered when selecting the most appropriate glove. It may be more effective to select a glove with lower chemical resistance but which is replaced frequently than to select a more resistant glove which is reused many times
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower. ▸ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▸ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▸ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

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:
843ER-A Super ShieldTM Silver Coated Copper Epoxy Conductive Coating (Part A)

Material	CPI
PE/EVAL/PE	A
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PVA	C
PVC	C
SARANEX-23	C
TEFLON	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as 'feel' or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

- **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**

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

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9.1. Information on basic physical and chemical properties

Appearance	Light brown metallic

Physical state	Liquid	**Relative density (Water = 1)**	1.19
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	≥343
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	30.00
Initial boiling point and boiling range (°C)	≥80	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	-3	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	10	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1.8	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	0.053	**Gas group**	Not Available
Solubility in water	Partly miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	≥2.1	**VOC g/L**	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

11.1. Information on toxicological effects

Inhaled	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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well. Not normally a hazard due to non-volatile nature of product Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. 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. Acute exposure of humans to high concentrations of methyl ethyl ketone produces irritation to the eyes, nose, and throat. Other effects reported from acute inhalation exposure in humans include central nervous system depression, headache, and nausea. Easy odour recognition and irritant properties of methyl ethyl ketone means that high vapour levels are readily detected and should be avoided by application of control measures; however odour fatigue may occur with loss of warning of exposure. The odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal.
Ingestion	Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In

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	general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased. Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema. Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in he case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent. 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 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. Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products.] Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain is availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively 'inactivates' the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper. Following ingestion, a single exposure to isopropyl alcohol produced lethargy and non-specific effects such as weight loss and irritation. Ingestion of near-lethal doses of isopropanol produces histopathological changes of the stomach, lungs and kidneys, incoordination, lethargy, gastrointestinal tract irritation, and inactivity or anaesthesia. Swallowing 10 ml. of isopropanol may cause serious injury; 100 ml. may be fatal if not promptly treated. The adult single lethal doses is approximately 250 ml. The toxicity of isopropanol is twice that of ethanol and the symptoms of intoxication appear to be similar except for the absence of an initial euphoric effect; gastritis and vomiting are more prominent. Ingestion may cause nausea, vomiting, and diarrhoea. There is evidence that a slight tolerance to isopropanol may be acquired.
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. Dermatitis has been reported in humans following dermal exposure to methyl ethyl ketone. Tests involving acute exposure of rabbits has shown methyl ethyl ketone to have high acute toxicity from dermal exposure. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man. Irritation and skin reactions are possible with sensitive skin Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature. 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. 511ipa The material may produce moderate skin irritation; limited evidence or practical experience suggests, 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	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn). The vapour when concentrated has pronounced eye irritation effects and this gives some warning of high vapour concentrations. If eye irritation occurs seek to reduce exposure with available control measures, or evacuate area. Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea. Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage. Eye contact may cause tearing or blurring of vision.
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.

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Silver is one of the most physically and physiologically cumulative of the elements. Chronic exposure to silver salts may cause argyria, a permanent ashen-grey discolouration of the skin, conjunctiva and internal organs (due to the deposit of an insoluble albuminate of silver).
The respiratory tract may also be a site of local argyria (following chronic inhalation exposures) with a mild chronic bronchitis being the only obvious symptom.
Limited information is available on the chronic (long-term) effects of methyl ethyl ketone in humans. Chronic inhalation studies in animals have reported slight neurological, liver, kidney, and respiratory effects. No information is available on the developmental, reproductive, or carcinogenic effects of methyl ethyl ketone in humans. Developmental effects, including decreased foetal weight and foetal malformations, have been reported in mice and rats exposed to methyl ethyl ketone via inhalation and ingestion.
Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities.
Combinations with chloroform also show increase in toxicity
All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.
Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether.
A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not *n*-butyl glycidyl ether, induced morphological transformation in mammalian cells *in vitro*. *n*-Butyl glycidyl ether induced micronuclei in mice *in vivo* following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations *in vivo* or chromosomal aberrations in animal cells *in vitro*. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in *Drosophila*. The glycidyl ethers were generally mutagenic to bacteria

Chronic copper poisoning is rarely recognised in man although in one instance, at least, symptoms more commonly associated with exposures to mercury, namely infantile acrodynia (pink disease), have been described. Tissue damage of mucous membranes may follow chronic dust exposure. A hazardous situation is exposure of a worker with the rare hereditary condition (Wilson's disease or hereditary hepatolenticular degeneration) to copper exposure which may cause liver, kidney, CNS, bone and sight damage and is potentially lethal. Haemolytic anaemia (a result of red-blood cell damage) is common in cows and sheep poisoned by copper derivatives. Overdosing of copper feed supplements has resulted in pigmentary cirrhosis of the liver. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products]
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.
Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain.

Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals.

There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol.

Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects.
NOTE: Commercial isopropanol does not contain 'isopropyl oil'. An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct 'isopropyl oil'. Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.

843ER-A Super ShieldTM Silver Coated Copper Epoxy Conductive Coating (Part A)

TOXICITY	IRRITATION
Not Available	Not Available

methyl ethyl ketone

TOXICITY	IRRITATION
Dermal (rabbit) LD50: ~6400-8000 mg/kg[2]	Eye (human): 350 ppm -irritant
Inhalation (rat) LC50: 47 mg/l/8H[2]	Eye (rabbit): 80 mg - irritant
Oral (rat) LD50: 2054 mg/kg[1]	Skin (rabbit): 402 mg/24 hr - mild
	Skin (rabbit):13.78mg/24 hr open

copper

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Not Available
Inhalation (rat) LC50: 0.733 mg/l4 h[1]	
Oral (rat) LD50: 300-500 mg/kg[1]	

bisphenol A diglycidyl ether resin, solid

TOXICITY	IRRITATION
dermal (rat) LD50: >1200 mg/kg[2]	Not Available
Oral (rat) LD50: >1000 mg/kg[2]	

n-butanol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 3400 mg/kg[2]	Eye (human): 50 ppm - irritant
Inhalation (rat) LC50: 24 mg/l/4H[2]	Eye (rabbit): 1.6 mg-SEVERE
Oral (rat) LD50: 790 mg/kg[2]	Eye (rabbit): 24 mg/24h-SEVERE
	Skin (rabbit): 405 mg/24h-moderate

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

843ER-A Super ShieldTM Silver Coated Copper Epoxy Conductive Coating (Part A)	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. 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. Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone.. Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review. A 2009 study on Chinese workers in bisphenol A factories found that workers were four times more likely to report erectile dysfunction, reduced sexual desire and overall dissatisfaction with their sex life than workers with no heightened bisphenol A exposure. Bisphenol A workers were also seven times more likely to have ejaculation difficulties. They were also more likely to report reduced sexual function within one year of beginning employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties. Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/ litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades' One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials' One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood. A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells.[whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes. Bisphenol A is the isopropyl adduct of 4,4'-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called 'cytostatic hormones'. Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown to be oestrogenic in vitro; such sealants may represent an additional source of xenoestrogens in humans and may be the cause of additional concerns in children. Concerns have been raised about the possible developmental effects on the foetus/embryo or neonate resulting from the leaching of bisphenol A from epoxy linings in metal cans which come in contact with food-stuffs. Many drugs, including naproxen, salicylic acid, carbamazepine and mefenamic acid can, in vitro, significantly inhibit bisphenol A glucuronidation (detoxification). 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.

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	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
COPPER	for copper and its compounds (typically copper chloride): **Acute toxicity:** There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs. No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation. **Repeat dose toxicity:** In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride. **Genotoxicity:** An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen. **Carcinogenicity:** there was insufficient information to evaluate the carcinogenic activity of copper monochloride. Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day). WARNING: Inhalation of high concentrations of copper fume may cause 'metal fume fever', an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever.
BISPHENOL A DIGLYCIDYL ETHER RESIN, SOLID	CAUTION: Epoxy resin products may contain sensitising glycidyl ethers, even when these are not mentioned in the information given for the product. The likely occurrence of these is greatly reduced in solid grades of the resin.
N-BUTANOL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. for n-butanol **Acute toxicity:** n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC0 of 8000 ppm (24000 mg/m3) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA's localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser. The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed. **Repeat dose toxicity:** An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAc) to BA. Hydrolysis of BAc in blood and brain was estimated to be 99 percent complete within 2.7 minutes (elimination t1/2 = 0.41 minute). Thus, organisms exposed to BAc can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAc can be used as supplemental, surrogate data to provide information on the toxicity of BA. A thirteen-week, subchronic exposure to BAc, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m3) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOAEL) of 500 ppm (2395 mg/m3) was reported for systemic effects in rats, and a NOAEL of 3000 ppm (14370 mg/m3) was reported for post exposure neurotoxicity in rats. **Reproductive toxicity:** Several studies indicate that BA is not a reproductive toxicant. Female rats exposed to 6000 ppm (18000 mg/m3) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m3) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity. **Developmental toxicity:** BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m3) throughout gestation. **Genotoxicity:** An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic. **Carcinogenicity:** Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.
ISOPROPANOL	For isopropanol (IPA): **Acute toxicity:** Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat. Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred. **Repeat dose studies**: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and

Continued...

	oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney. **Reproductive toxicity**: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful. **Developmental toxicity:** The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity **Genotoxicity:** All genotoxicity assays reported for isopropanol have been negative **Carcinogenicity:** rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment
TALC	For talc (a form of magnesium silicate) The overuse of talc in nursing infants has resulted in pulmonary oedema, pneumonia and death within hours of inhaling talcum powder.The powder dries the mucous membranes of the bronchioles, disrupts pulmonary clearance, clogs smaller airways. Victims display wheezing, rapid or difficult breathing, increased pulse, cyanosis, fever. Mild exposure may cause relatively minor inflammatory lung disease. Long term exposure may show wheezing, weakness, productive cough, limited chest expansion, scattered rales, cyanosis.
843ER-A Super ShieldTM Silver Coated Copper Epoxy Conductive Coating (Part A) & BISPHENOL A DIGLYCIDYL ETHER RESIN, SOLID	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. 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.
843ER-A Super ShieldTM Silver Coated Copper Epoxy Conductive Coating (Part A) & METHYL ETHYL KETONE	Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities. Combinations with chloroform also show increase in toxicity
METHYL ETHYL KETONE & N-BUTANOL & TALC	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.
METHYL ETHYL KETONE & N-BUTANOL	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.
BISPHENOL A DIGLYCIDYL ETHER RESIN, SOLID & ISOPROPANOL	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 RESIN, SOLID & TALC	No significant acute toxicological data identified in literature search.
ISOPROPANOL & TALC	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.

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

12.1. Toxicity

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843ER-A Super ShieldTM Silver Coated Copper Epoxy Conductive Coating (Part A)

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

methyl ethyl ketone

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	2-993mg/L	2
EC50	48	Crustacea	5-91mg/L	2
EC50	72	Algae or other aquatic plants	1-972mg/L	2
EC0	96	Fish	1-848mg/L	2
NOEC	96	Fish	1-170mg/L	2

copper

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	0.001-0.09mg/L	2
EC50	48	Crustacea	0.001mg/L	2
EC50	72	Algae or other aquatic plants	0.013335mg/L	4
BCF	960	Fish	200mg/L	4
EC25	6	Algae or other aquatic plants	0.00150495mg/L	4
NOEC	96	Crustacea	0.0008mg/L	4

bisphenol A diglycidyl ether resin, solid

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
EC50	48	Crustacea	ca.2mg/L	2

n-butanol

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	1-376mg/L	2
EC50	48	Crustacea	1-328mg/L	2
EC50	96	Algae or other aquatic plants	225mg/L	2
BCF	24	Fish	921mg/L	4
EC0	48	Crustacea	1-260mg/L	2
NOEC	504	Crustacea	4.1mg/L	2

isopropanol

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	9-640mg/L	2
EC50	48	Crustacea	12500mg/L	5
EC50	96	Algae or other aquatic plants	993.232mg/L	3
EC0	24	Crustacea	5-102mg/L	2
NOEC	5760	Fish	0.02mg/L	4

silver

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>0.001-0.93mg/L	2
EC50	48	Crustacea	0.00024mg/L	4
EC50	72	Algae or other aquatic plants	0.000016mg/L	2
BCF	336	Crustacea	0.02mg/L	4
NOEC	72	Algae or other aquatic plants	0.000003mg/L	2

talc

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	89-581.016mg/L	2
EC50	96	Algae or other aquatic plants	7-202.7mg/L	2
NOEC	720	Crustacea	1-459.798mg/L	2

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

Toxic to aquatic organisms.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. A significant

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proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms.
When released to dry soil most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. Environmental processes may also be important in changing solubilities.
Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects.
A metal ion is considered infinitely persistent because it cannot degrade further.
The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.
The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.
Environmental processes may enhance bioavailability.
For bisphenol A and related bisphenols:
Environmental fate:
Biodegradability (28 d) 89% - Easily biodegradable
Bioconcentration factor (BCF) 7.8 mg/l
Bisphenol A, its derivatives and analogues, can be released from polymers, resins and certain substances by metabolic products
Substance does not meet the criteria for PBT or vPvB according to Regulation (EC) No 1907/2006, Annex XIII
As an environmental contaminant, bisphenol A interferes with nitrogen fixation at the roots of leguminous plants associated with the bacterial symbiont Sinorhizobium meliloti. Despite a half-life in the soil of only 1-10 days, its ubiquity makes it an important pollutant. According to Environment Canada, 'initial assessment shows that at low levels, bisphenol A can harm fish and organisms over time. Studies also indicate that it can currently be found in municipal wastewater.' However, a study conducted in the United States found that 91-98% of bisphenol A may be removed from water during treatment at municipal water treatment plants.
Ecotoxicity:
Fish LC50 (96 h): 4.6 mg/l (freshwater fish); 11 mg/l (saltwater fish): NOEC 0.016 mg/l (freshwater fish- 144 d); 0.064 mg/l (saltwater fish 164 d)
Fresh water invertebrates EC50 (48 h): 10.2 mg/l: NOEC 0.025 mg/l - 328 d)
Marine water invertebrate EC50 (96 h): 1.1 mg/l; NOEC 0.17 mg/l (28 d)
Freshwater algae (96 h): 2.73 mg/l
Marine water algae (96 h): 1.1 mg/l
Fresh water plant EC50 (7 d): 20 mg/l: NOEC 7.8 mg/l
In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms.
Among freshwater organisms, fish appear to be the most sensitive species. Evidence of endocrine-related effects in fish, aquatic invertebrates, amphibians and reptiles has been reported at environmentally relevant exposure levels lower than those required for acute toxicity. There is a widespread variation in reported values for endocrine-related effects, but many fall in the range of 1 ug/L to 1 mg/L
A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations. A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.
Although abundant data are available on the toxicity of bisphenol-A (2,2-bis (4-hydroxydiphenyl)propane;(BPA) A variety of BPs were examined for their acute toxicity against Daphnia magna, mutagenicity, and oestrogenic activity using the Daphtoxkit (Creasel Ltd.), the umu test system, and the yeast two-hybrid system, respectively, in comparison with BPA. BPA was moderately toxic to D. magna (48-h EC50 was 10 mg/l) according to the current U.S. EPA acute toxicity evaluation standard, and it was weakly oestrogenic with 5 orders of magnitude lower activity than that of the natural estrogen 17 beta-oestradiol in the yeast screen, while no mutagenicity was observed. All seven BPs tested here showed moderate to slight acute toxicity, no mutagenicity, and weak oestrogenic activity as well as BPA. Some of the BPs showed considerably higher oestrogenic activity than BPA, and others exhibited much lower activity. Bisphenol S (bis(4-hydroxydiphenyl)sulfone) and bis(4-hydroxyphenyl)sulfide) showed oestrogenic activity.
Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F ([bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem,
Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe3+ ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.

Copper is unlikely to accumulate in the atmosphere due to a short residence time for airborne copper aerosols. Airborne coppers, however, may be transported over large distances. Copper accumulates significantly in the food chain.
Drinking Water Standards:
3000 ug/l (UK max)
2000 ug/l (WHO provisional Guideline)
1000 ug/l (WHO level where individuals complain)
Soil Guidelines: Dutch Criteria
36 mg/kg (target)
190 mg/kg (intervention)
Air Quality Standards: no data available.
The toxic effect of copper in the aquatic biota depends on the bio-availability of copper in water which, in turn, depends on its physico-chemical form (ie.speciation). Bioavailability is decreased by complexation and adsorption of copper by natural organic matter, iron and manganese hydrated oxides, and chelating agents excreted by algae and other aquatic organisms. Toxicity is also affected by pH and hardness. Total copper is rarely useful as a predictor of toxicity. In natural sea water, more than 98% of copper is organically bound and in river waters a high percentage is often organically bound, but the actual percentage depends on the river water and its pH.
Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper with EC50 (96 hour) values as low as 47 ug/litre dissolved copper whilst for other algal species EC50 values of up to 481 ug/litre have been reported. However many of the reportedly high EC50 values may arise in experiments conducted with a culture media containing copper-complexing agents such as silicate, iron, manganese and EDTA which reduce bioavailability.
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).

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For methyl ethyl ketone:
log Kow : 0.26-0.69
log Koc : 0.69
Koc : 34
Half-life (hr) air : 2.3
Half-life (hr) H2O surface water : 72-288
Henry's atm m3 /mol: 1.05E-05
BOD 5 : 1.5-2.24, 46%
COD : 2.2-2.31, 100%
ThOD : 2.44
BCF : 1

Environmental fate:

TERRESTRIAL FATE: Measured Koc values of 29 and 34 were obtained for methyl ethyl ketone in silt loams. Methyl ethyl ketone is expected to have very high mobility in soil. Volatilisation of methyl ethyl ketone from dry soil surfaces is expected based upon an experimental vapor pressure of 91 mm Hg at 25 deg C. Volatilization from moist soil surfaces is also expected given the measured Henry's Law constant of 4.7x10-5 atm-cu m/mole. The volatilisation half-life of methyl ethyl ketone from silt and sandy loams was measured as 4.9 days. Methyl ethyl ketone is expected to biodegrade under both aerobic and anaerobic conditions as indicated by numerous screening tests.
AQUATIC FATE: Based on Koc values, methyl ethyl ketone is not expected to adsorb to suspended solids and sediment in water. Methyl ethyl ketone is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated half-lives for a model river and model lake are 19 and 197, hours respectively. Biodegradation of this compound is expected based upon numerous screening tests. An estimated BCF value of 1 based on an experimental log Kow of 0.29, suggests that bioconcentration in aquatic organisms is low.
ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, methyl ethyl ketone, which has an experimental vapor pressure of 91 mm Hg at 25 deg C, will exist solely as a vapor in the ambient atmosphere. Vapour-phase methyl ethyl ketone is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 14 days. Methyl ethyl ketone is also expected to undergo photodecomposition in the atmosphere by natural sunlight. Photochemical degradation of methyl ethyl ketone by natural sunlight is expected to occur at approximately 1/5 the rate of degradation by photochemically produced hydroxyl radicals.

Ecotoxicity:

Fish LC50 (24 h): bluegill sunfish (Lepomis macrochirus) 1690-5640 mg/l; guppy (Lebistes reticulatus) 5700 mg/l; goldfish (Carassius auratus) >5000 mg/l
Fish LC50 (96 h): fathead minnow (Pimephales promelas) 3200 mg/l; bluegill sunfish (Lepomis macrochirus) 4467 mg/l; mosquito fish (Gambusia affinis) 5600 mg/l
Daphnia magna LC50 (48 h):<520-1382 mg/l
Daphnia magna LC50 (24 h): 8890 mg/l
Brine shrimp (Artemia salina) LC50 (24 h): 1950 mg/l

Toxic effects arising following exposure by aquatic species to copper are typically:

Algae EC50 (96 h)	Daphnia magna LC50 (48-96 h)	Amphipods LC50 (48-96 h)	Gastropods LC50 (48-96 h)	Crab larvae LC50 (48-96 h)
47-481 *	7-54 *	37-183 *	58-112 *	50-100 *

* ug/litre

Exposure to concentrations ranging from one to a few hundred micrograms per litre has led to sublethal effects and effects on long-term survival. For high bioavailability waters, effect concentrations for several sensitive species may be below 10 ug Cu/litre.

In fish, the acute lethal concentration of copper ranges from a few ug/litre to several mg/litre, depending both on test species and exposure conditions. Where the value is less than 50 ug Cu/litre, test waters generally have a low dissolved organic carbon (DOC) level, low hardness and neutral to slightly acidic pH. Exposure to concentrations ranging from one to a few hundred micrograms per litre has led to sublethal effects and effects on long-term survival. Lower effect concentrations are generally associated with test waters of high bioavailability.

In summary:

Responses expected for high concentration ranges of copper *

Total dissolved Cu concentration range (ug/litre)	Effects of high availability in water
1-10	Significant effects are expected for diatoms and sensitive invertebrates, notably cladocerans. Effects on fish could be significant in freshwaters with low pH and hardness.
10-100	Significant effects are expected on various species of microalgae, some species of macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Survival of sensitive fish will be affected and a variety of fish show sublethal effects.
100-1000	Most taxonomic groups of macroalgae and invertebrates will be severely affected. Lethal levels for most fish species will be reached.
>1000	Lethal concentrations for most tolerant organisms are reached.

* Sites chosen have moderate to high bioavailability similar to water used in most toxicity tests.

In soil, copper levels are raised by application of fertiliser, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Generally, vegetation rooted in soils reflects the soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned.

Typical foliar levels of copper are:

Uncontaminated soils (0.3-250 mg/kg)	Contaminated soils (150-450 mg/kg)	Mining/smelting soils
6.1-25 mg/kg	80 mg/kg	300 mg/kg

Plants rarely show symptoms of toxicity or of adverse growth effects at normal soil concentrations of copper. Crops are often more sensitive to copper than the native flora, so protection levels for agricultural crops range from 25 mg Cu/kg to several hundred mg/kg, depending on country. Chronic and or acute effects on sensitive species occur at copper levels occurring in some soils as a result of human activities such as copper fertiliser addition, and addition of sludge.

When soil levels exceed 150 mg Cu/kg, native and agricultural species show chronic effects. Soils in the range 500-1000 mg Cu/kg act in a strongly selective fashion allowing the survival of only copper-tolerant species and strains. At 2000 Cu mg/kg most species cannot survive. By 3500 mg Cu/kg areas are largely devoid of vegetation cover. The organic content of the soil appears to be a key factor affecting the bioavailability of copper.

On normal forest soils, non-rooted plants such as mosses and lichens show higher copper concentrations. The fruiting bodies and mycorrhizal sheaths of soil fungi associated with higher plants in forests often accumulate copper to much higher levels than plants at the same site. International Programme on Chemical Safety (IPCS): Environmental Health Criteria 200
For silver and its compounds:

Environmental fate:

Silver is a rare but naturally occurring metal, often found deposited as a mineral ore in association with other elements. Emissions from smelting operations, manufacture and disposal of certain photographic and electrical supplies, coal combustion, and cloud seeding are some of the anthropogenic sources of silver in the biosphere. The global biogeochemical movements of silver are characterized by releases to the atmosphere, water, and land by natural and anthropogenic sources, long-range transport of fine particles in the atmosphere, wet and dry deposition, and sorption to soils and sediments.
In general, accumulation of silver by terrestrial plants from soils is low, even if the soil is amended with silver-containing sewage sludge or the plants are grown on tailings from silver mines, where silver accumulates mainly in the root systems.
The ability to accumulate dissolved silver varies widely between species. Some reported bioconcentration factors for marine organisms (calculated as milligrams of silver per kilogram fresh weight organism divided by milligrams of silver per litre of medium) are 210 in diatoms, 240 in brown algae, 330 in mussels, 2300 in scallops, and 18 700 in oysters, whereas bioconcentration factors for freshwater organisms have been reported to range from negligible in bluegills (*Lepomis macrochirus*) to 60 in daphnids; these values represent uptake of bioavailable silver in laboratory experiments. Laboratory studies with the less toxic silver compounds, such as silver sulfide and silver chloride, reveal that accumulation of silver does not necessarily lead to adverse effects. At concentrations normally encountered in the environment, food-chain biomagnification of silver in aquatic systems is unlikely. Elevated silver concentrations in biota occur in the vicinities of sewage outfalls, electroplating plants, mine waste sites, and silver iodide-seeded areas. Maximum concentrations recorded in field collections, in milligrams total silver per kilogram dry weight (tissue),

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were 1.5 in marine mammals (liver) (except Alaskan beluga whales *Delphinapterus leucas*, which had concentrations 2 orders of magnitude higher than those of other marine mammals), 6 in fish (bone), 14 in plants (whole), 30 in annelid worms (whole), 44 in birds (liver), 110 in mushrooms (whole), 185 in bivalve molluscs (soft parts), and 320 in gastropods (whole).
Ecotoxicity:
In general, silver ion was less toxic to freshwater aquatic organisms under conditions of low dissolved silver ion concentration and increasing water pH, hardness, sulfides, and dissolved and particulate organic loadings; under static test conditions, compared with flow-through regimens; and when animals were adequately nourished instead of being starved. Silver ions are very toxic to microorganisms. However, there is generally no strong inhibitory effect on microbial activity in sewage treatment plants because of reduced bioavailability due to rapid complexation and adsorption. Free silver ion was lethal to representative species of sensitive aquatic plants, invertebrates, and teleosts at nominal water concentrations of 1-5 ug/litre. Adverse effects occur on development of trout at concentrations as low as 0.17 ug/litre and on phytoplankton species composition and succession at 0.3-0.6 ug/litre.

A knowledge of the speciation of silver and its consequent bioavailability is crucial to understanding the potential risk of the metal. Measurement of free ionic silver is the only direct method that can be used to assess the likely effects of the metal on organisms. Speciation models can be used to assess the likely proportion of the total silver measured that is bioavailable to organisms. Unlike some other metals, background freshwater concentrations in pristine and most urban areas are well below concentrations causing toxic effects. Levels in most industrialized areas border on the effect concentration, assuming that conditions favour bioavailability. On the basis of available toxicity test results, it is unlikely that bioavailable free silver ions would ever be at sufficiently high concentrations to cause toxicity in marine environments.
No data were found on effects of silver on wild birds or mammals. Silver was harmful to poultry (tested as silver nitrate) at concentrations as low as 100 mg total silver/litre in drinking-water or 200 mg total silver/kg in diets. Sensitive laboratory mammals were adversely affected at total silver concentrations (added as silver nitrate) as low as 250 ug/litre in drinking-water (brain histopathology), 6 mg/kg in diet (high accumulations in kidneys and liver), or 13.9 mg/kg body weight (lethality).

Silver and Silver Compounds; Concise International Chemical Assessment Document (CICAD) 44 IPCS InChem (WHO)
'The transport of silver through estuarine and coastal marine systems is dependent on biological uptake and incorporation. Uptake by phytoplankton is rapid, in proportion to silver concentration and inversely proportional to salinity. In contrast to studies performed with other toxic metals, sliver availability appears to be controlled by both the free silver ion concentration and the concentration of other silver complexes. Silver incorporated by phytoplankton is not lost as salinity increase; as a result silver associated with cellular material is largely retained within the estuary. Phytoplankton exhibit a variable sensitivity to silver. Sensitive species exhibit a marked delay in the onset of growth in response to silver at low concentrations, even though maximum growth rates are similar to controls. A delay in the onset of growth reduces the ability of a population to respond to short-term favourable conditions and to succeed within th community.
James G. Saunders and George R Abbe: Aquatic Toxicology and Environmental Fate; ASTM STP 1007, 1989, pp 5-18
For ketones:
Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds
Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrateThe higher molecular weight ketones do no form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions
Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (*e.g.,* ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable.
Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
bisphenol A diglycidyl ether resin, solid	HIGH	HIGH
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
methyl ethyl ketone	LOW (LogKOW = 0.29)
bisphenol A diglycidyl ether resin, solid	LOW (LogKOW = 2.6835)
n-butanol	LOW (BCF = 0.64)
isopropanol	LOW (LogKOW = 0.05)

12.4. Mobility in soil

Ingredient	Mobility
methyl ethyl ketone	MEDIUM (KOC = 3.827)
bisphenol A diglycidyl ether resin, solid	LOW (KOC = 51.43)
n-butanol	MEDIUM (KOC = 2.443)
isopropanol	HIGH (KOC = 1.06)

12.5.Results of PBT and vPvB assessment

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

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

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13.1. 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 ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

Limited quantity: 843ER-250ML, 843ER-800ML, 843ER-3.25L kits

Land transport (ADR)

14.1. **UN number**	1139
14.2. **UN proper shipping name**	Not Applicable
14.3. **Transport hazard class(es)**	Class: 3 Subrisk: Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Hazard identification (Kemler): 33 Classification code: F1 Hazard Label: 3 Special provisions: 640C; 640D Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	1139
14.2. **UN proper shipping name**	Coating solution (includes surface treatments or coatings used for industrial or other purposes such as vehicle undercoating, drum or barrel lining)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A3 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L Passenger and Cargo Packing Instructions: 353 Passenger and Cargo Maximum Qty / Pack: 5 L

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	Passenger and Cargo Limited Quantity Packing Instructions	Y341
	Passenger and Cargo Limited Maximum Qty / Pack	1 L

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	1139
14.2. **UN proper shipping name**	COATING SOLUTION (includes surface treatments or coatings used for industrial or other purposes such as vehicle under-coating, drum or barrel lining)
14.3. **Transport hazard class(es)**	IMDG Class: 3 IMDG Subrisk: Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-E , S-E Special provisions: Not Applicable Limited Quantities: 5 L

Inland waterways transport (ADN)

14.1. **UN number**	1139
14.2. **UN proper shipping name**	COATING SOLUTION (includes surface treatments or coatings used for industrial or other purposes such as vehicle under coating, drum or barrel lining) (vapour pressure at 50°C more than 110 kPa); COATING SOLUTION (includes surface treatments or coatings used for industrial or other purposes such as vehicle under coating, drum or barrel lining) (vapour pressure at 50°C not more than 110 kPa)
14.3. **Transport hazard class(es)**	3 Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Classification code: F1 Special provisions: 640C|640D Limited quantity: 5 L Equipment required: PP, EX, A Fire cones number: 1

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

METHYL ETHYL KETONE(78-93-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)
Europe European Customs Inventory of Chemical Substances - ECICS (Slovak)
Europe European Customs Inventory of Chemical Substances ECICS (Bulgarian)
Europe European Customs Inventory of Chemical Substances ECICS (Czech)
Europe European Customs Inventory of Chemical Substances ECICS (Romanian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
GESAMP/EHS Composite List - GESAMP Hazard Profiles
IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
UK Workplace Exposure Limits (WELs)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

COPPER(7440-50-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
Europe European Customs Inventory of Chemical Substances - ECICS (Slovak)
Europe European Customs Inventory of Chemical Substances ECICS (Bulgarian)
Europe European Customs Inventory of Chemical Substances ECICS (Czech)
Europe European Customs Inventory of Chemical Substances ECICS (Romanian)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
UK Workplace Exposure Limits (WELs)

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

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways
Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
International Air Transport Association (IATA) Dangerous Goods Regulations
International FOSFA List of Banned Immediate Previous Cargoes
International Maritime Dangerous Goods Requirements (IMDG Code)
Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

N-BUTANOL(71-36-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)
Europe European Customs Inventory of Chemical Substances - ECICS (Slovak)
Europe European Customs Inventory of Chemical Substances ECICS (Bulgarian)
Europe European Customs Inventory of Chemical Substances ECICS (Czech)
Europe European Customs Inventory of Chemical Substances ECICS (Romanian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
GESAMP/EHS Composite List - GESAMP Hazard Profiles
IMO IBC Code Chapter 17: Summary of minimum requirements
IMO IBC Code Chapter 18: List of products to which the Code does not apply
IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
UK Workplace Exposure Limits (WELs)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

ISOPROPANOL(67-63-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways
Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
GESAMP/EHS Composite List - GESAMP Hazard Profiles
IMO IBC Code Chapter 18: List of products to which the Code does not apply
IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO
IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
UK Workplace Exposure Limits (WELs)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

SILVER(7440-22-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
UK Workplace Exposure Limits (WELs)

TALC(14807-96-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Europe EC Inventory
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
UK Workplace Exposure Limits (WELs)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2015/830; Regulation (EC) No 1272/2008 as updated through ATPs.

Continued...

15.2. Chemical safety assessment

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

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (bisphenol A diglycidyl ether resin, solid; talc; n-butanol; copper; isopropanol; silver; methyl ethyl ketone)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (copper; silver)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Legend:	*Yes = All ingredients are on the inventory* *No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Revision Date	18/03/2020
Initial Date	02/05/2018

Full text Risk and Hazard codes

H226	Flammable liquid and vapour.
H302	Harmful if swallowed.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.
H411	Toxic to aquatic life with long lasting effects.

Other information

Ingredients with multiple cas numbers

Name	CAS No
copper	7440-50-8, 133353-46-5, 133353-47-6, 195161-80-9, 65555-90-0, 72514-83-1
bisphenol A diglycidyl ether resin, solid	25068-38-6, 25085-99-8

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index

Reason For Change

A-1.02 - Update to the emergency phone number information.

end of SDS