



841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A)

MG Chemicals UK Limited

Version No: A-2.00

Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: 07/09/2021

Revision Date: 07/09/2021

L.REACH.GB.EN

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

1.1. Product Identifier

Product name	841ER-A
Synonyms	SDS Code: 841ER-Part A; 841ER-1.17L, 841ER-3.25L UFI:RCK0-60C3-T000-Y657
Other means of identification	Super Shield Nickel Epoxy Conductive Paint (Part A)

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

Relevant identified uses	nickel conductive epoxy resin
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

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

1.4. Emergency telephone number

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

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H336 - Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, H225 - Flammable Liquids Category 2, H318 - Serious Eye Damage/Eye Irritation Category 1, H315 - Skin Corrosion/Irritation Category 2, H317 - Sensitisation (Skin) Category 1, H372 - Specific Target Organ Toxicity - Repeated Exposure Category 1, H351 - Carcinogenicity Category 2, H412 - Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567

2.2. Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H336	May cause drowsiness or dizziness.
H225	Highly flammable liquid and vapour.
H318	Causes serious eye damage.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H372	Causes damage to organs through prolonged or repeated exposure.
H351	Suspected of causing cancer.

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H412	Harmful to aquatic life with long lasting effects.
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Supplementary statement(s)

EUH205	Contains epoxy constituents. May produce an allergic reaction.
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Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe mist/vapours/spray.
P271	Use only a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and 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.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
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.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
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 to authorised hazardous or special waste collection point in accordance with any local regulation.
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2.3. Other hazards

Inhalation, skin contact and/or ingestion may produce health damage*.

Cumulative effects may result following exposure*.

May possibly affect fertility*.

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
1.7440-02-0 2.231-111-4 3.028-002-00-7[028-002-01-4 4.Not Available	53	<u>nickel</u>	Sensitisation (Skin) Category 1, Carcinogenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3; H317, H351, H372, H412 [2]	Not Available
1.78-93-3 2.201-159-0 3.606-002-00-3 4.Not Available	15	<u>methyl ethyl ketone</u> *	Flammable Liquids Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H225, H319, H336, EUH066 [2]	Not Available
1.123-86-4 2.204-658-1 3.607-025-00-1 4.Not Available	10	<u>n-butyl acetate</u> *	Flammable Liquids Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H226, H336, EUH066 [2]	Not Available

Continued...

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1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
1.1675-54-3 2.216-823-5 3.603-073-00-2 603-074-00-8 4.Not Available	8	<u>bisphenol A diglycidyl ether</u>	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Sensitisation (Skin) Category 1; H315, H319, H317 [2]	Not Available
1.71-36-3 2.200-751-6 3.603-004-00-6 4.Not Available	7	<u>n-butanol</u>	Flammable Liquids Category 3, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H226, H302, H315, H318, H335, H336 [2]	Not Available
1.14807-96-6 2.238-877-9 3.Not Available 4.Not Available	3	<u>talc</u>	Acute Toxicity (Inhalation) Category 4, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3; H332, H335 [1]	Not Available
1.68609-97-2 2.271-846-8 3.603-103-00-4 4.Not Available	2	<u>(C12-14)alkylglycidyl ether</u>	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1; H315, H317 [2]	Not Available
Legend:		1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties		

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▶ 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.

Treat symptomatically.

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.

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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.
- ▶ 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

- ▶ **DO NOT** use halogenated fire extinguishing agents.

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.

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▶ 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
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5.3. Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ 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	<ul style="list-style-type: none"> ▶ 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. <p>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.</p> <p>Metal powders, while generally regarded as non-combustible:</p> <ul style="list-style-type: none"> ▶ 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. <p>Note:</p> <ul style="list-style-type: none"> ▶ 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. <p>Combustion products include: carbon dioxide (CO2) other pyrolysis products typical of burning organic material.</p>

SECTION 6 Accidental release measures

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6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none">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.																																																																																																																																		
Major Spills	<p>Environmental hazard - contain spillage.</p> <p>Chemical Class: ester and ethers</p> <p>For release onto land: recommended sorbents listed in order of priority.</p> <table><tr><th>SORBENT TYPE</th><th>RANK</th><th>APPLICATION</th><th>COLLECTION</th><th>LIMITATIONS</th></tr></table> <p>LAND SPILL - SMALL</p> <table><tr><td>cross-linked polymer - particulate</td><td>1</td><td>shovel</td><td>shovel</td><td>R, W, SS</td></tr><tr><td>cross-linked polymer - pillow</td><td>1</td><td>throw</td><td>pitchfork</td><td>R, DGC, RT</td></tr><tr><td>sorbent clay - particulate</td><td>2</td><td>shovel</td><td>shovel</td><td>R,I, P</td></tr><tr><td>wood fiber - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, W, P, DGC</td></tr><tr><td>wood fiber - pillow</td><td>3</td><td>throw</td><td>pitchfork</td><td>R, P, DGC, RT</td></tr><tr><td>treated wood fiber - pillow</td><td>3</td><td>throw</td><td>pitchfork</td><td>DGC, RT</td></tr></table> <p>LAND SPILL - MEDIUM</p> <table><tr><td>cross-linked polymer - 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841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A)

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.
- ▶ 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	<ul style="list-style-type: none"> ▶ 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. ▶ 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	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ CARE: Packing of high density product in light weight metal or plastic packages may result in container collapse with product release ▶ Heavy gauge metal packages / Heavy gauge metal drums ▶ 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.
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Storage incompatibility

n-Butyl acetate:

- reacts with water on standing to form acetic acid and n-butyl alcohol
- reacts violently with strong oxidisers and potassium tert-butoxide
- is incompatible with caustics, strong acids and nitrates
- dissolves rubber, many plastics, resins and some coatings

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 acid
- 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

Nickel

- is a strong reducing agent
- may be pyrophoric when dry (dependent on particle size); powders or dusts may ignite spontaneously in air
- reacts with acids, evolving flammable hydrogen gas
- reacts violently with ammonia, ammonium nitrate, fluorine, hydrazine, hydrazoic acid, strong oxidisers, nitric acid, peroxyformic acid, potassium, potassium perchlorate, selenium, sulfur (evolves heat, incandescence), titanium and other materials
- is incompatible with organic solvents, sulfur compounds
- in reducing atmosphere furnace can react with carbon monoxide forming highly toxic nickel carbonyl gas; under fire conditions may also react in similar manner
- Raney alloys, containing aluminium, may react with moisture
- 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
- Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.

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

Epoxides:

- are highly reactive with acids, bases, and oxidising and reducing agents.
- react, possibly violently, with anhydrous metal chlorides, ammonia, amines and group 1 metals.
- may polymerise in the presence of peroxides or heat - polymerisation may be violent
- may react, possibly violently, with water in the presence of acids and other catalysts.
- Esters react with acids to liberate heat along with alcohols and acids.
- Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products.
- Heat is also generated by the interaction of esters with caustic solutions.
- Flammable hydrogen is generated by mixing esters with alkali metals and hydrides.
- Esters may be incompatible with aliphatic amines and nitrates.

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

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
nickel	Inhalation 0.05 mg/m ³ (Systemic, Chronic) Dermal 0.035 mg/cm ² (Local, Chronic) Inhalation 0.05 mg/m ³ (Local, Chronic) Inhalation 11.9 mg/m ³ (Local, Acute) <i>Inhalation 60 ng/m³ (Systemic, Chronic) *</i>	7.1 µg/L (Water (Fresh)) 8.6 µg/L (Water - Intermittent release) 0 µg/L (Water (Marine)) 109 mg/kg sediment dw (Sediment (Fresh Water)) 109 mg/kg sediment dw (Sediment (Marine))

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Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
	<i>Oral 0.011 mg/kg bw/day (Systemic, Chronic) *</i> <i>Dermal 0.035 mg/cm² (Local, Chronic) *</i> <i>Inhalation 60 ng/m³ (Local, Chronic) *</i> <i>Oral 0.37 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 0.8 mg/m³ (Local, Acute) *</i>	29.9 mg/kg soil dw (Soil) 0.33 mg/L (STP) 0.12 mg/kg food (Oral)
methyl ethyl ketone	Dermal 1 161 mg/kg bw/day (Systemic, Chronic) Inhalation 600 mg/m³ (Systemic, Chronic) <i>Dermal 412 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 106 mg/m³ (Systemic, Chronic) *</i> <i>Oral 31 mg/kg bw/day (Systemic, Chronic) *</i>	55.8 mg/L (Water (Fresh)) 55.8 mg/L (Water - Intermittent release) 55.8 mg/L (Water (Marine)) 284.74 mg/kg sediment dw (Sediment (Fresh Water)) 284.7 mg/kg sediment dw (Sediment (Marine)) 22.5 mg/kg soil dw (Soil) 709 mg/L (STP) 1000 mg/kg food (Oral)
n-butyl acetate	Dermal 7 mg/kg bw/day (Systemic, Chronic) Inhalation 48 mg/m³ (Systemic, Chronic) Inhalation 300 mg/m³ (Local, Chronic) Dermal 11 mg/kg bw/day (Systemic, Acute) Inhalation 600 mg/m³ (Systemic, Acute) Inhalation 600 mg/m³ (Local, Acute) <i>Dermal 3.4 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 12 mg/m³ (Systemic, Chronic) *</i> <i>Oral 2 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 35.7 mg/m³ (Local, Chronic) *</i> <i>Dermal 6 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 300 mg/m³ (Systemic, Acute) *</i> <i>Oral 2 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 300 mg/m³ (Local, Acute) *</i>	0.18 mg/L (Water (Fresh)) 0.018 mg/L (Water - Intermittent release) 0.36 mg/L (Water (Marine)) 0.981 mg/kg sediment dw (Sediment (Fresh Water)) 0.098 mg/kg sediment dw (Sediment (Marine)) 0.09 mg/kg soil dw (Soil) 35.6 mg/L (STP)
bisphenol A diglycidyl ether	Dermal 0.75 mg/kg bw/day (Systemic, Chronic) Inhalation 4.93 mg/m³ (Systemic, Chronic) <i>Dermal 89.3 µg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 0.87 mg/m³ (Systemic, Chronic) *</i> <i>Oral 0.5 mg/kg bw/day (Systemic, Chronic) *</i>	0.006 mg/L (Water (Fresh)) 0.001 mg/L (Water - Intermittent release) 0.018 mg/L (Water (Marine)) 0.341 mg/kg sediment dw (Sediment (Fresh Water)) 0.034 mg/kg sediment dw (Sediment (Marine)) 0.065 mg/kg soil dw (Soil) 10 mg/L (STP) 11 mg/kg food (Oral)
n-butanol	Inhalation 310 mg/m³ (Local, Chronic) <i>Dermal 3.125 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 55.357 mg/m³ (Systemic, Chronic) *</i> <i>Oral 1.562 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 155 mg/m³ (Local, Chronic) *</i>	0.082 mg/L (Water (Fresh)) 0.008 mg/L (Water - Intermittent release) 2.25 mg/L (Water (Marine)) 0.324 mg/kg sediment dw (Sediment (Fresh Water)) 0.032 mg/kg sediment dw (Sediment (Marine)) 0.017 mg/kg soil dw (Soil) 2476 mg/L (STP)
talc	Dermal 43.2 mg/kg bw/day (Systemic, Chronic) Inhalation 2.16 mg/m³ (Systemic, Chronic) Dermal 4.54 mg/cm² (Local, Chronic) Inhalation 3.6 mg/m³ (Local, Chronic) Inhalation 2.16 mg/m³ (Systemic, Acute) Inhalation 3.6 mg/m³ (Local, Acute) <i>Dermal 21.6 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 1.08 mg/m³ (Systemic, Chronic) *</i> <i>Oral 160 mg/kg bw/day (Systemic, Chronic) *</i> <i>Dermal 2.27 mg/cm² (Local, Chronic) *</i> <i>Inhalation 1.8 mg/m³ (Local, Chronic) *</i> <i>Inhalation 1.08 mg/m³ (Systemic, Acute) *</i> <i>Oral 160 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 1.8 mg/m³ (Local, Acute) *</i>	597.97 mg/L (Water (Fresh)) 141.26 mg/L (Water - Intermittent release) 597.97 mg/L (Water (Marine)) 31.33 mg/kg sediment dw (Sediment (Fresh Water)) 3.13 mg/kg sediment dw (Sediment (Marine))
(C12-14)alkylglycidyl ether	Dermal 1 mg/kg bw/day (Systemic, Chronic) Inhalation 3.6 mg/m³ (Systemic, Chronic) <i>Dermal 0.5 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 0.87 mg/m³ (Systemic, Chronic) *</i> <i>Oral 0.5 mg/kg bw/day (Systemic, Chronic) *</i>	0.106 mg/L (Water (Fresh)) 0.011 mg/L (Water - Intermittent release) 0.072 mg/L (Water (Marine)) 307.16 mg/kg sediment dw (Sediment (Fresh Water)) 30.72 mg/kg sediment dw (Sediment (Marine)) 1.234 mg/kg soil dw (Soil) 10 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Europe ECHA Occupational exposure limits - Activity list	nickel	Not Available	Not Available	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	nickel	Nickel and its inorganic compounds (except nickel tetracarbonyl): nickel and water-insoluble nickel compounds (as Ni)	0.5 mg/m3	Not Available	Not Available	Sk, Carc (nickel oxides and sulphides) Sen (nickel sulphate)
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

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	n-butyl acetate	n-Butyl acetate	50 ppm / 241 mg/m3	723 mg/m3 / 150 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	n-butyl acetate	Butyl acetate	150 ppm / 724 mg/m3	966 mg/m3 / 200 ppm	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)	talc	Talc, respirable dust	1 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
nickel	4.5 mg/m3	50 mg/m3	99 mg/m3
methyl ethyl ketone	Not Available	Not Available	Not Available
n-butyl acetate	Not Available	Not Available	Not Available
bisphenol A diglycidyl ether	39 mg/m3	430 mg/m3	2,600 mg/m3
bisphenol A diglycidyl ether	90 mg/m3	990 mg/m3	5,900 mg/m3
n-butanol	60 ppm	800 ppm	8000** ppm

Ingredient	Original IDLH	Revised IDLH
nickel	10 mg/m3	Not Available
methyl ethyl ketone	3,000 ppm	Not Available
n-butyl acetate	1,700 ppm	Not Available
bisphenol A diglycidyl ether	Not Available	Not Available
n-butanol	1,400 ppm	Not Available
talc	1,000 mg/m3	Not Available
(C12-14)alkylglycidyl ether	Not Available	Not Available

Occupational Exposure Banding

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

MATERIAL DATA

For talc (a form of magnesium silicate):

Most health problems associated with occupational exposure to talcs appear to evolve mostly from the nonplatform 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 µm. 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 µm with shorter, thicker particles having little biological activity.

Dust of nonfibrous talc, consisting entirely of platform 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.

For n-butyl acetate

Odour Threshold Value: 0.0063 ppm (detection), 0.038-12 ppm (recognition)

Exposure at or below the recommended TLV-TWA is thought to prevent significant irritation of the eyes and respiratory passages as well as narcotic effects. In light of the lack of substantive evidence regarding teratogenicity and a review of acute oral data a STEL is considered inappropriate.

Odour Safety Factor(OSF)

OSF=3.8E2 (n-BUTYL ACETATE)

For epichlorohydrin

Odour Threshold Value: 0.08 ppm

NOTE: Detector tubes for epichlorohydrin, measuring in excess of 5 ppm, are commercially available.

Exposure at or below the recommended TLV-TWA is thought to minimise the potential for adverse respiratory, liver, kidney effects. Epichlorohydrin has been implicated as a human skin sensitiser, hence individuals who are hypersusceptible or otherwise unusually responsive to certain chemicals may NOT be adequately protected from adverse health effects.

Odour Safety Factor (OSF)

OSF=0.54 (EPICHLOROHYDRIN)

For methyl ethyl ketone:

Odour Threshold Value: Various 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)

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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)

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.
- ▶ 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.

For esters:

- ▶ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.

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The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

- Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

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 and 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:

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Material	CPI
PE/EVAL/PE	A
TEFLON	A

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the 'Exposure Standard' (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
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Continued...

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PVA	B
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PVC	C
SARANEX-23	C
VITON/BUTYL	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.

up to 10 x ES	A-AUS	-	A-PAPR-AUS / Class 1
up to 50 x ES	-	A-AUS / Class 1	-
up to 100 x ES	-	A-2	A-PAPR-2 ^

^ - Full-face

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

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

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	grey		
Physical state	Liquid	Relative density (Water = 1)	1.8
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	200
Initial boiling point and boiling range (°C)	80	Molecular weight (g/mol)	Not Available
Flash point (°C)	-9	Taste	Not Available
Evaporation rate	Not Available BuAC = 1	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	11	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.7	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (%)	Not Available
Vapour density (Air = 1)	>4	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

Continued...

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SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression, headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures.</p> <p>Human subjects exposed to 24 ppm n-butanol experienced mild irritation which became objectionable. Headaches were reported at 50 ppm. Exposure by mice to 6600 ppm produced signs of marked central nervous system (CNS) depression, including prostration after 2 hours, narcosis after 3 hours and some deaths.</p> <p>Although n-butanol is odorous and generally possesses adequate warning properties, the olfactory senses may become fatigued. 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.</p> <p>Not normally a hazard due to non-volatile nature of product</p> <p>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.</p> <p>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.</p> <p>Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Regular exposure to nickel fume, as the oxide, may result in 'metal fume fever' a sometimes debilitating upper respiratory tract condition resembling influenza.</p> <p>Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in closed or poorly ventilated areas. Pulmonary oedema, pulmonary fibrosis and asthma has been reported in welders using nickel alloys; level of exposure are generally not available and case reports are often confounded by mixed exposures to other agents.</p> <p>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p>
Ingestion	<p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Reactive diluents exhibit a range of ingestion hazards. Small amounts swallowed incidental to normal handling operations are not likely to cause injury. However, swallowing larger amounts may cause injury.</p> <p>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.</p> <p>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 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.</p> <p>Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality</p> <p>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</p>

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	<p>small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.</p> <p>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 the 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.</p> <p>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 in the testis. There were no significant differences with respect to sperm head count, sperm motility, and sperm abnormality in the BADGE treatment groups</p> <p>Swallowing of n-butanol may cause breathing difficulty, headache, nausea, vomiting, upper respiratory tract irritation, mucous membrane irritation, central nervous system depression.</p> <p>The material has NOT been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Nickel is poorly absorbed from the gastrointestinal tract. It is transported in the plasma bound to serum albumin and various small organic ligands. Excretion in the urine is substantially complete in 4-5 days. Serum nickel is influenced by environmental nickel or nickel concentrations in the air with faecal nickel about 100 times urinary nickel. Parenterally administered nickel is rapidly distributed to kidney, pituitary, lung, skin, adrenal and ovary and testis. In vivo binding with metallothionein has been demonstrated. A nickel binding protein has also been identified in plasma; it has been tentatively identified as an alpha-1-glycoprotein with a serum alpha-1-macroglobulin complex.</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Bisphenol A diglycidyl ether (BADGE) may produce contact dermatitis characterised by erythema and oedema, with weeping followed by crusting and scaling. A liquid resin with a molecular weight of 350 produced severe skin irritation in rabbits when applied daily for 4 hours over 20 days.</p> <p>Following the initial contact there may be a discrete erythematous lesion, confined to the point of contact, which may persist for 48 hours to 10 days; the erythema may give way to a papular, vesicular rash with scaling.</p> <p>In animals uncured resin produces moderate ante-mortem depression, loss of body weight and diarrhoea. Local irritation, inflammation and death resulting from respiratory system depression are recorded. Higher molecular weight resins generally produce lower toxicity.</p> <p>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.</p> <p>Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Workers exposed to 200 ppm n-butanol showed ocular symptoms including corneal inflammation, burning sensation, blurring of vision, lachrymation, and photophobia. 100 ppm produced no systemic effects and reports of irritation of the eyes was rare.</p>
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances than can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>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.</p> <p>Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity..</p> <p>Testicular abnormalities (including testicular atrophy with decreased spermatogenic activity) following exposure to glycidyl ethers have been reported. Haemopoietic abnormalities following exposure to glycidyl ethers, including alteration of the leukocyte count, atrophy of lymphoid tissue, and bone marrow cytotoxicity have also been reported. These abnormalities were usually observed along with pneumonia and/or toxemia, and</p>

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therefore may be secondary effects. However, especially in light of the generalized reduction in leukocytes and the atrophy of lymphoid tissues, the observed haemopoietic abnormalities may have been predisposing factors to pneumonia. While none of the individual research reports are conclusive with respect to the ability of glycidyl ethers to produce permanent changes to the testes or haemopoietic system in laboratory animals, the pattern of displayed effects is reason for concern

Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether.

A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not n-butyl glycidyl ether, induced morphological transformation in mammalian cells in vitro. n-Butyl glycidyl ether induced micronuclei in mice in vivo following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations in vivo or chromosomal aberrations in animal cells in vitro. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in *Drosophila*. The glycidyl ethers were generally mutagenic to bacteria.

Bisphenol A diglycidyl ethers (BADGEs) produce sensitisation dermatitis characterised by a papular, vesicular eczema with considerable itching of the back of the hand, the forearm and face and neck. This lesion may persist for 10-14 days after withdrawal from exposure and recur immediately on re-exposure. This dermatitis may persist for longer periods following each exposure but is unlikely to become more intense. Lesions may develop a brownish colour and scaling occurs frequently. Lower molecular weight species produce sensitisation more readily. In mice technical grades of bisphenol A diglycidyl ether produced epidermal tumours and a small increase in the incidence kidney tumours in males and of lymphoreticular/ haematopoietic tumours in females. Subcutaneous injection produced a small number of fibrosarcomas in rats. BADGE is listed as an IARC Group 3 carcinogen, meaning it is 'not classifiable as to its carcinogenicity to humans'. Concern has been raised over this possible carcinogenicity because BADGE is used in epoxy resins in the lining of some tin cans for foodstuffs, and unreacted BADGE may end up in the contents of those cans.

For some reactive diluents, prolonged or repeated skin contact may result in absorption of potentially harmful amounts or allergic skin reactions. Exposure to some reactive diluents (notably neopentylglycol diglycidyl ether, CAS RN:17557-23-2) has caused cancer in some animal testing. 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

Serious systemic effects from exposure to n-butanol in the form of auditory and vestibular nerve damage have been reported amongst workers in France and Mexico. Audiologic impairment was produced in workers exposed to 80 ppm n-butanol with unprotected noise exposure. Workers exposed over a 15 year period (1929-1944) exhibited severe vertigo and vertiges graves. Workers exposed from 3-11 years without personal protective equipment from noise experienced greater hearing loss (hypoacusia) in direct relation to exposure time when compared to a control group exposed to industrial noise of 90-100 dB but with n-butanol exposure. Average hearing loss was not large but the workers had central frequencies of 21.98 dB (11.59 dB minimum and 32.30 dB maximum) with a mean widening of the break between 3000 and 4000 Hz of 42.22 dB. There was a tendency of the averages to decrease as the frequencies moved away from the central zone. Affected workers were aged from 20-39 years. [ACGIH Documentation of TLVs]

Metallic dusts generated by the industrial process give rise to a number of potential health problems. The larger particles, above 5 micron, are nose and throat irritants. Smaller particles however, may cause lung deterioration. Particles of less than 1.5 micron can be trapped in the lungs and, dependent on the nature of the particle, may give rise to further serious health consequences.

Metals are widely distributed in the environment and are not biodegradable. Biologically, many metals are essential to living systems and are involved in a variety of cellular, physiological, and structural functions. They often are cofactors of enzymes, and play a role in transcriptional control, muscle contraction, nerve transmission, blood clotting, and oxygen transport and delivery. Although all metals are potentially toxic at some level, some are highly toxic at relatively low levels. Moreover, in some cases the same metal can be essential at low levels and toxic at higher levels, or it may be toxic via one route of entry but not another. Toxic effects of some metals are associated with disruption of functions of essential metals. Metals may have a range of effects, including cancer, neurotoxicity, immunotoxicity, cardiotoxicity, reproductive toxicity, teratogenicity, and genotoxicity. Biological half lives of metals vary greatly, from hours to years. Furthermore, the half life of a given metal varies in different tissues. Lead has a half life of 14 days in soft tissues and 20 years in bone.

In considering how to evaluate the toxicity of metals of potential concern, a number of aspects of metal toxicity should be kept in mind:

Different species vary in their responses to different metals; in some cases, humans are more sensitive than rodents. Thus, there is a need for broad-based testing of metals;

- ▶ The route of exposure may affect the dose and site where the metal concentrates, and thus the observed toxic effects;
- ▶ Metal-metal interactions can reduce or enhance toxicity; biotransformation can reduce or enhance toxicity;
- ▶ It is difficult to predict the toxicity of one metal based on the adverse effects of another; in trying to evaluate the toxicity of one particular metal compound, predictions based on similar compounds of the same metal may be valid.

841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A)	TOXICITY Not Available	IRRITATION Not Available
nickel	TOXICITY Oral(Rat) LD50; >9000 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
methyl ethyl ketone	TOXICITY Dermal (rabbit) LD50: ~6400-8000 mg/kg ^[2] Inhalation(Mouse) LC50; 32 mg/L4h ^[2] Oral(Rat) LD50; 2054 mg/kg ^[1]	IRRITATION Eye (human): 350 ppm -irritant Eye (rabbit): 80 mg - irritant Skin (rabbit): 402 mg/24 hr - mild Skin (rabbit):13.78mg/24 hr open
n-butyl acetate	TOXICITY Dermal (rabbit) LD50: >14100 mg/kg ^[2] Inhalation(Rat) LC50; 0.74 mg/l4h ^[2] Oral(Rat) LD50; >3200 mg/kg ^[2]	IRRITATION Eye (human): 300 mg Eye (rabbit): 20 mg (open)-SEVERE Eye (rabbit): 20 mg/24h - moderate

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		Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating) ^[1]
bisphenol A diglycidyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 2 mg/24h - SEVERE
	Oral(Rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg - mild
		Skin: adverse effect observed (irritating) ^[1]
n-butanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: ~3430 mg/kg ^[1]	Eye (human): 50 ppm - irritant
	Inhalation(Rat) LC50: >17.76 mg/14h ^[2]	Eye (rabbit): 1.6 mg-SEVERE
	Oral(Mouse) LD50: 100 mg/kg ^[2]	Eye (rabbit): 24 mg/24h-SEVERE
		Eye: adverse effect observed (irreversible damage) ^[1]
		Skin (rabbit): 405 mg/24h-moderate
talc	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50: >2.1 mg/14h ^[1]	Skin (human): 0.3 mg/3d-I mild
	Oral(Rat) LD50: >5000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
(C12-14)alkylglycidyl ether	TOXICITY	IRRITATION
	Oral(Rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): mild [Ciba]
		Eye: adverse effect observed (irritating) ^[1]
		Skin (guinea pig): sensitiser
		Skin (human): Irritant
		Skin (human): non- sensitiser
		Skin (rabbit): moderate
		Skin : Moderate
		Skin: adverse effect observed (irritating) ^[1]
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	

841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A)	<p>Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).</p> <p>Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.</p> <p>Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.</p>
NICKEL	<p>Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m3/24H/17W-C</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]</p>
BISPHENOL A DIGLYCIDYL ETHER	<p>Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. The presence of the p-hydroxy group on the benzene rings is thought to be responsible for the oestradiol mimicry.</p> <p>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.</p> <p>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.</p> <p>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</p>

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and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadias and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades'. One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials'.

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

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

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

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

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

BPA belongs to the list of compounds having this property as the rodent models have shown that BPA exposure is linked with increased body weight (obesogens). Several mechanisms can help explain the effect of BPA on body weight increase. A possible mechanism leading to triglyceride accumulation is the decreased production of the hormone adiponectin from all human adipose tissue tested when exposed to very low levels (below nanomolar range) of BPA in cell or explant culture settings. The expression of leptin as well as several enzymes and transcription factors is also affected by BPA exposure in vivo as well as in vitro. Together, the altered expression and activity of these important mediators of fat metabolism could explain the increase in weight following BPA exposure in rodent models. These results also suggest that, together with other obesogens, low, environmentally relevant levels of BPA may contribute to the human obesity phenomenon.

All glycidyl ethers show genotoxic potential due to their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.

Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity.. Testicular abnormalities (including testicular atrophy with decreased spermatogenic activity) following exposure to glycidyl ethers have been reported. Haemopoietic abnormalities following exposure to glycidyl ethers, including alteration of the leukocyte count, atrophy of lymphoid tissue, and bone marrow cytotoxicity have also been reported. These abnormalities were usually observed along with pneumonia and/or toxemia, and therefore may be secondary effects. However, especially in light of the generalized reduction in leukocytes and the atrophy of lymphoid tissues, the observed haemopoietic abnormalities may have been predisposing factors to pneumonia. While none of the individual research reports are conclusive with respect to the ability of glycidyl ethers to produce permanent changes to the testes or haemopoietic system in laboratory animals, the pattern of displayed effects is reason for concern.

Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether.

A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not n-butyl glycidyl ether, induced morphological transformation in mammalian cells in vitro. n-Butyl glycidyl ether induced micronuclei in mice in vivo following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations in vivo or chromosomal aberrations in animal cells in vitro. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in *Drosophila*. The glycidyl ethers were generally mutagenic to bacteria.

55badger

N-BUTANOL

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.

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TALC	<p>No significant acute toxicological data identified in literature search.</p> <p>For talc (a form of magnesium silicate)</p> <p>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.</p> <p>Long term exposure may show wheezing, weakness, productive cough, limited chest expansion, scattered rales, cyanosis.</p>
841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A) & METHYL ETHYL KETONE & N-BUTANOL & TALC	<p>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.</p>
841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A) & NICKEL & BISPHENOL A DIGLYCIDYL ETHER & (C12-14)ALKYLGlycidyl ETHER	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A) & N-BUTYL ACETATE	<p>Generally, linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized</p> <p>Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw</p> <p>Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic.</p> <p>The JEFCA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods</p> <p>International Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA) Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids., 1998</p>
841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A) & BISPHENOL A DIGLYCIDYL ETHER	<p>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).</p> <p>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.</p> <p>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).</p> <p>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).</p> <p>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).</p> <p>Immunotoxicity: Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs</p> <p>-</p> <p>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 weight/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.</p>
841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A) & BISPHENOL A DIGLYCIDYL ETHER & (C12-14)ALKYLGlycidyl ETHER	<p>Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative.</p>
841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A) & METHYL ETHYL KETONE	<p>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</p>

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METHYL ETHYL KETONE & N-BUTYL ACETATE & 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.
N-BUTYL ACETATE & N-BUTANOL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
BISPHENOL A DIGLYCIDYL ETHER & (C12-14)ALKYLGLYCIDYL ETHER	for 1,2-butylene oxide (ethyloxirane): Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic
BISPHENOL A DIGLYCIDYL ETHER & 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

11.2.1. Endocrine Disruption Properties

Not Available

SECTION 12 Ecological information

12.1. Toxicity

841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
nickel	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	72h	Algae or other aquatic plants	0.18mg/l	1
	EC50	72h	Algae or other aquatic plants	0.18mg/l	1
	LC50	96h	Fish	0.168mg/L	4
	EC50	48h	Crustacea	>100mg/l	1
	EC50	96h	Algae or other aquatic plants	0.36mg/l	2
methyl ethyl ketone	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Crustacea	68mg/l	2
	EC50	72h	Algae or other aquatic plants	1972mg/l	2
	LC50	96h	Fish	>324mg/L	4
	EC50	48h	Crustacea	308mg/l	2
	EC50	96h	Algae or other aquatic plants	>500mg/l	4
n-butyl acetate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Fish	18mg/l	2
	EC50	72h	Algae or other aquatic plants	246mg/l	2
	LC50	96h	Fish	18mg/l	2
	EC50	48h	Crustacea	32mg/l	1
bisphenol A diglycidyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	9.4mg/l	2
	LC50	96h	Fish	1.2mg/l	2
	EC50	48h	Crustacea	1.1mg/l	2
	NOEC(ECx)	504h	Crustacea	0.3mg/l	2

Continued...

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n-butanol	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	4.1mg/l	2
	EC50	72h	Algae or other aquatic plants	>500mg/l	1
	LC50	96h	Fish	100-500mg/l	4
	EC50	48h	Crustacea	>500mg/l	1
	EC50	96h	Algae or other aquatic plants	225mg/l	2
talc	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	89581.016mg/l	2
	NOEC(ECx)	720h	Algae or other aquatic plants	918.089mg/l	2
	EC50	96h	Algae or other aquatic plants	7202.7mg/l	2
(C12-14)alkylglycidyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	48h	Crustacea	6.07mg/l	2
	LC50	96h	Fish	>5000mg/l	2
	EC50	48h	Crustacea	6.07mg/l	2
Legend: <i>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</i>					

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

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

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

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and/or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and/or delayed, to the structure and/or functioning of natural ecosystems.

Liquid epoxy resins and some reactive diluents are not readily biodegradable, although its epoxy functional groups are hydrolysed in contact with water, they have the potential to bio-accumulate and are moderately toxic to aquatic organisms. They are generally classified as dangerous for the environment according to the European Union classification criteria. Uncured solid resins on the other hand are not readily bio-available, not toxic to aquatic and terrestrial organisms, not readily biodegradable, but hydrolysable. They present no significant hazard for the environment.

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 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.

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 ($t_{1/2\text{water}} : t_{1/2\text{soil}} : t_{1/2\text{sediment}} = 1 : 1 : 4$) (Boethling 1995). According to these values, it can be concluded that ethyloxirane does not meet the persistence criteria in water and soil (half-lives = 182 days) and sediments (half-life = 365 days).

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

Ecotoxicity:

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

* Persistence and Bioaccumulation Regulations (Canada 2000).

For 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:

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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 4.7×10^{-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

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 substrate. The higher molecular weight ketones do not 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.

For n-butyl acetate:

Half-life (hr) air : 144

Half-life (hr) H2O surface water : 178-27156

Henry's atm m³ /mol: 3.20E-04

BOD 5 if unstated: 0.15-1.02,7%

COD : 78%

ThOD : 2.207

BCF : 4-14

Environmental Fate:

TERRESTRIAL FATE: An estimated Koc value of 200 determined from a measured log Kow of 1.78 indicates that n-butyl acetate is expected to have moderate mobility in soil.

Volatilisation of n-butyl acetate is expected from moist soil surfaces given its Henry's Law constant of 2.8×10^{-4} atm-cu m/mole. Volatilisation from dry soil surfaces is expected based on a measured vapor pressure of 11.5 mm Hg. Using a standard BOD dilution technique and a sewage inoculum, theoretical BODs of 56 % to 86 % were observed during 5-20 day incubation periods, which suggests that n-butyl acetate may biodegrade in soil.

AQUATIC FATE: An estimated Koc value indicates that n-butyl acetate is not expected to adsorb to suspended solids and sediment in water. Butyl acetate is expected to volatilise from water surfaces based on a Henry's Law constant of 2.8×10^{-4} atm-cu m/mole. Estimated half-lives for a model river and model lake are 7 and 127, hours respectively. An estimated BCF value of 10 based on the log Kow, suggests that bioconcentration in aquatic organisms is low. Using a filtered sewage seed, 5-day and 20-day theoretical BODs of 58 % and 83 % were measured in freshwater dilution tests; 5-day and 20-day theoretical BODs of 40 % and 61 % were measured in salt water. A 5-day theoretical BOD of 56.8 % and 51.8 % were measured for n-butyl acetate in distilled water and seawater, respectively. Hydrolysis may be an important environmental fate for this compound based upon experimentally determined hydrolysis half-lives of 114 and 11 days at pH 8 and 9 respectively.

ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, n-butyl acetate, which has a vapour pressure of 11.5 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapour-phase n-butyl acetate 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 4 days

Environmental fate:

Fish LC50 (96 h, 23 C): island silverside (*Menidia beryllina*) 185 ppm (static bioassay in synthetic seawater, mild aeration applied after 24 h); bluegill sunfish (*Lepomis macrochirus*)

100 ppm (static bioassay in fresh water, mild aeration applied after 24 h)

Fish EC50 (96 h): fathead minnow (*Pimephales promelas*) 18 mg/l (affected fish lost equilibrium prior to death)

Daphnia LC50 (48 h): 44 ppm

Algal LC50 (96 h): *Scenedesmus* 320 ppm

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)
n-butyl acetate	LOW	LOW
bisphenol A diglycidyl ether	HIGH	HIGH
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
methyl ethyl ketone	LOW (LogKOW = 0.29)
n-butyl acetate	LOW (BCF = 14)
bisphenol A diglycidyl ether	MEDIUM (LogKOW = 3.8446)
n-butanol	LOW (BCF = 0.64)

12.4. Mobility in soil

Ingredient	Mobility
methyl ethyl ketone	MEDIUM (KOC = 3.827)
n-butyl acetate	LOW (KOC = 20.86)
bisphenol A diglycidyl ether	LOW (KOC = 1767)

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Ingredient	Mobility
n-butanol	MEDIUM (KOC = 2.443)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗
PBT Criteria fulfilled?	No		
vPvB	No		

12.6. Endocrine Disruption Properties

Not Available

12.7. Other adverse effects

Not Available


SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Waste Management</p> <p>Production waste from epoxy resins and resin systems should be treated as hazardous waste in accordance with National regulations. Fire retarded resins containing halogenated compounds should also be treated as special waste. Accidental spillage of resins, curing agents and their formulations should be contained and absorbed by special mineral absorbents to prevent them from entering the environment.</p> <p>Contaminated or surplus product should not be washed down the sink, but preferably be fully reacted to form cross-linked solids which is non-hazardous and can be more easily disposed.</p> <p>Finished articles made from fully cured epoxy resins are hard, infusible solids presenting no hazard to the environment. However, finished articles from flame-retarded material containing halogenated resins should be considered hazardous waste, and disposed as required by National laws.</p> <p>Articles made from epoxy resins, like other thermosets, can be recycled by grinding and used as fillers in other products. Another way of disposal and recovery is combustion with energy recovery.</p> <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> Reduction Reuse Recycling Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> DO NOT allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. Recycle wherever possible. 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

	 <p>Limited Quantity: 841ER-250ML, 841ER-1.17L, 841ER-3.25L</p>
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Land transport (ADR-RID)

14.1. UN number	1263
14.2. UN proper shipping name	PAINT or PAINT RELATED MATERIAL

Continued...

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14.3. Transport hazard class(es)	Class	3
	Subrisk	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Hazard identification (Kemler)	33
	Classification code	F1
	Hazard Label	3
	Special provisions	163 367 640C 650 640D
	Limited quantity	5 L
	Tunnel Restriction Code	2 (D/E)

Air transport (ICAO-IATA / DGR)

14.1. UN number	1263	
14.2. UN proper shipping name	PAINT or PAINT RELATED MATERIAL	
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	Not Applicable	
14.6. Special precautions for user	Special provisions	A3 A72 A192
	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
	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	1263	
14.2. UN proper shipping name	PAINT or PAINT RELATED MATERIAL	
14.3. Transport hazard class(es)	IMDG Class	3
	IMDG Subrisk	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	F-E , S-E
	Special provisions	163 367
	Limited Quantities	5 L

Inland waterways transport (ADN)

14.1. UN number	1263	
14.2. UN proper shipping name	PAINT or PAINT RELATED MATERIAL	
14.3. Transport hazard class(es)	3	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Classification code	F1
	Special provisions	163; 367; 640C; 640D; 650
	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

Continued...

841ER-A Super Shield Nickel Epoxy Conductive Paint (Part A)

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

Product name	Group
nickel	Not Available
methyl ethyl ketone	Not Available
n-butyl acetate	Not Available
bisphenol A diglycidyl ether	Not Available
n-butanol	Not Available
talc	Not Available
(C12-14)alkylglycidyl ether	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
nickel	Not Available
methyl ethyl ketone	Not Available
n-butyl acetate	Not Available
bisphenol A diglycidyl ether	Not Available
n-butanol	Not Available
talc	Not Available
(C12-14)alkylglycidyl ether	Not Available

SECTION 15 Regulatory information

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

nickel is found on the following regulatory lists

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

Europe EC Inventory

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

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

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

methyl ethyl ketone is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

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

Europe EC Inventory

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

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

n-butyl acetate is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

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

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

bisphenol A diglycidyl ether is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

Europe EC Inventory

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

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

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

n-butanol is found on the following regulatory lists

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

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

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

talc is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
Europe EC Inventory

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

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

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

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Chemical Footprint Project - Chemicals of High Concern List

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

Europe EC Inventory

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

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

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

15.2. Chemical safety assessment

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

National Inventory Status

National Inventory	Status
Australia - AIIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (nickel; methyl ethyl ketone; n-butyl acetate; bisphenol A diglycidyl ether; n-butanol; talc; (C12-14)alkylglycidyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (nickel; (C12-14)alkylglycidyl ether)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (bisphenol A diglycidyl ether; (C12-14)alkylglycidyl ether)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</p>

SECTION 16 Other information

Revision Date	07/09/2021
Initial Date	12/06/2018

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.

Other information

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

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average

PC—STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit.

IDLH: Immediately Dangerous to Life or Health Concentrations

ES: Exposure Standard

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

AIIIC: Australian Inventory of Industrial Chemicals

Continued...

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DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory

NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act

TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

Reason For Change

A-2.00 - Add UFI number and modified safety data sheet