



## 8331-B Silver Conductive Epoxy Adhesive

### Mektronics

Version No: 1.2

Safety Data Sheet according to WHS and ADG requirements

Issue Date: 05/03/2020

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

### Product Identifier

Product name	8331-B
Synonyms	SDS Code: 8331-14G, 8331-50ML, 8331-200ML (withdrawn: 8331-429G, 8331-454G)
Other means of identification	Silver Conductive Epoxy Adhesive

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

Relevant identified uses	Electrically conductive epoxy adhesive hardener part for use with resins
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### Details of the supplier of the safety data sheet

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

### Emergency telephone number

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

## SECTION 2 HAZARDS IDENTIFICATION

### Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 1B, Acute Aquatic Hazard Category 1, Serious Eye Damage Category 1, Acute Toxicity (Oral) Category 4, Reproductive Toxicity Category 2, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

### Label elements

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

### Hazard statement(s)

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

### Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.

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P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

## Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P310	Immediately call a POISON CENTER or doctor/physician.
P321	Specific treatment (see advice on this label).
P363	Wash contaminated clothing before reuse.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

## Precautionary statement(s) Storage

P405	Store locked up.
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## 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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## SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

## Substances

See section below for composition of Mixtures

## Mixtures

CAS No	%[weight]	Name
7440-22-4	67	silver
84852-15-3	22	4-nonylphenol, branched
140-31-8	7	N-aminoethylpiperazine
68411-71-2	1	bisphenol A diglycidyl ether diethylenetriamine reaction products
111-40-0	1	diethylenetriamine
80-05-7	1	bisphenol A

## SECTION 4 FIRST AID MEASURES

## Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Immediately hold eyelids apart and flush the eye continuously with running water.</li> <li>▶ 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.</li> <li>▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> <li>▶ <b>DO NOT attempt to remove particles attached to or embedded in eye .</b></li> <li>▶ Lay victim down, on stretcher if available and pad <b>BOTH</b> eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye.</li> <li>▶ Seek urgent medical assistance, or transport to hospital.</li> </ul>
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately flush body and clothes with large amounts of water, using safety shower if available.</li> <li>▶ Quickly remove all contaminated clothing, including footwear.</li> <li>▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
Inhalation	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor, without delay.</li> </ul>

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	<ul style="list-style-type: none"> <li>▶ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema.</li> <li>▶ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).</li> <li>▶ As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested.</li> <li>▶ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered.</li> </ul> <p><b>This must definitely be left to a doctor or person authorised by him/her.</b> (ICSC13719)</p>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ For advice, contact a Poisons Information Centre or a doctor at once.</li> <li>▶ Urgent hospital treatment is likely to be needed.</li> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Transport to hospital or doctor without delay.</li> </ul>

**Indication of any immediate medical attention and special treatment needed**

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

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For acute or short-term repeated exposures to highly alkaline materials:

- ▶ Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- ▶ Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- ▶ Oxygen is given as indicated.
- ▶ The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- ▶ Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- ▶ Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

- ▶ Neutralising agents should never be given since exothermic heat reaction may compound injury.

\* Catharsis and emesis are absolutely contra-indicated.

\* Activated charcoal does not absorb alkali.

\* Gastric lavage should not be used.

Supportive care involves the following:

- ▶ Withhold oral feedings initially.
- ▶ If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- ▶ Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- ▶ Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

- ▶ Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

For acute or short term repeated exposures to phenols/ cresols:

- ▶ Phenol is absorbed rapidly through lungs and skin. [Massive skin contact may result in collapse and death]\*
- ▶ [Ingestion may result in ulceration of upper respiratory tract; perforation of oesophagus and/or stomach, with attendant complications, may occur. Oesophageal stricture may occur.]\*
- ▶ An initial excitatory phase may present. Convulsions may appear as long as 18 hours after ingestion. Hypotension and ventricular tachycardia that require vasopressor and antiarrhythmic therapy, respectively, can occur.
- ▶ Respiratory arrest, ventricular dysrhythmias, seizures and metabolic acidosis may complicate severe phenol exposures so the initial attention should be directed towards stabilisation of breathing and circulation with ventilation, intubation, intravenous lines, fluids and cardiac monitoring as indicated.
- ▶ [Vegetable oils retard absorption; do NOT use paraffin oils or alcohols. Gastric lavage, with endotracheal intubation, should be repeated until phenol odour is no longer detectable; follow with vegetable oil. A saline cathartic should then be given.]\* ALTERNATIVELY: Activated charcoal (1g/kg) may be given. A cathartic should be given after oral activated charcoal.
- ▶ Severe poisoning may require slow intravenous injection of methylene blue to treat methaemoglobinaemia.
- ▶ [Renal failure may require haemodialysis.]\*
- ▶ Most absorbed phenol is biotransformed by the liver to ethereal and glucuronide sulfates and is eliminated almost completely after 24 hours. [Ellenhorn and Barceloux: Medical Toxicology] \*[Union Carbide]

**BIOLOGICAL EXPOSURE INDEX - BEI**

These represent the determinants observed in specimens collected from a healthy worker who has been exposed to the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
1. Total phenol in blood	250 mg/gm creatinine	End of shift	B, NS

B: Background levels occur in specimens collected from subjects **NOT** exposed

NS: Non-specific determinant; also seen in exposure to other materials

**SECTION 5 FIREFIGHTING MEASURES**

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## Extinguishing media

Metal dust fires need to be smothered with sand, inert dry powders.

**DO NOT USE WATER, CO<sub>2</sub> 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 CO<sub>2</sub> may produce flammable and explosive methane.
- ▶ If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- ▶ **DO NOT** use halogenated fire extinguishing agents.

## Special hazards arising from the substrate or mixture

<b>Fire Incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Reacts with acids producing flammable / explosive hydrogen (H<sub>2</sub>) gas</li> <li>▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result</li> </ul>
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## Advice for firefighters

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear full body protective clothing with breathing apparatus.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Use fire fighting procedures suitable for surrounding area.</li> <li>▶ <b>Do not approach containers suspected to be hot.</b></li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ <b>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.</b></li> <li>▶ <b>DO NOT use water or foam as generation of explosive hydrogen may result.</b></li> </ul> <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"> <li>▶ May burn when metal is finely divided and energy input is high.</li> <li>▶ May react explosively with water.</li> <li>▶ May be ignited by friction, heat, sparks or flame.</li> <li>▶ May <b>REIGNITE</b> after fire is extinguished.</li> <li>▶ Will burn with intense heat.</li> </ul> <p>Note:</p> <ul style="list-style-type: none"> <li>▶ Metal dust fires are slow moving but intense and difficult to extinguish.</li> <li>▶ Containers may explode on heating.</li> <li>▶ Dusts or fumes may form explosive mixtures with air.</li> <li>▶ Gases generated in fire may be poisonous, corrosive or irritating.</li> <li>▶ 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.</li> <li>▶ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids</li> <li>▶ 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.</li> </ul> <p>Combustible. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO<sub>2</sub>) other pyrolysis products typical of burning organic material. May emit corrosive fumes.</p>
<b>HAZCHEM</b>	2X

## SECTION 6 ACCIDENTAL RELEASE MEASURES

## Personal precautions, protective equipment and emergency procedures

See section 8

## Environmental precautions

See section 12

## Methods and material for containment and cleaning up

<b>Minor Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Remove all ignition sources.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Use dry clean up procedures and avoid generating dust.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> <li>▶ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.</li> <li>▶ Check regularly for spills and leaks.</li> </ul>
<b>Major Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>· Do not use compressed air to remove metal dusts from floors, beams or equipment</li> <li>· Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.</li> <li>· Use non-sparking handling equipment, tools and natural bristle brushes.</li> <li>· Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations</li> </ul>

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- Cover and reseal partially empty containers.
  - Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.
- If molten:
- ▶ Contain the flow using dry sand or salt flux as a dam.
  - ▶ All tooling (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use.
  - ▶ Allow the spill to cool before remelting scrap.
  - ▶ Clear area of personnel and move upwind.
  - ▶ Alert Fire Brigade and tell them location and nature of hazard.
  - ▶ Wear full body protective clothing with breathing apparatus.
  - ▶ Prevent, by any means available, spillage from entering drains or water course.
  - ▶ Consider evacuation (or protect in place).
  - ▶ Stop leak if safe to do so.
  - ▶ Contain spill with sand, earth or vermiculite.
  - ▶ Collect recoverable product into labelled containers for recycling.
  - ▶ Neutralise/decontaminate residue (see Section 13 for specific agent).
  - ▶ Collect solid residues and seal in labelled drums for disposal.
  - ▶ Wash area and prevent runoff into drains.
  - ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
  - ▶ If contamination of drains or waterways occurs, advise emergency services.

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

## SECTION 7 HANDLING AND STORAGE

## Precautions for safe handling

<b>Safe handling</b>	<p>For molten metals:</p> <ul style="list-style-type: none"> <li>· Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions.</li> <li>· All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use.</li> <li>· Any surfaces that may contact molten metal (e.g. concrete) should be specially coated</li> <li>· Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard.</li> </ul> <p>During melting operations, the following minimum guidelines should be observed:</p> <ul style="list-style-type: none"> <li>· Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage.</li> <li>· Store materials in dry, heated areas with any cracks or cavities pointed downwards.</li> <li>· Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours.</li> </ul> <ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ <b>WARNING: To avoid violent reaction, ALWAYS add material to water and NEVER water to material.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ When handling, <b>DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ Store in a cool, dry, well-ventilated area.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ <b>DO NOT store near acids, or oxidising agents</b></li> <li>▶ No smoking, naked lights, heat or ignition sources.</li> </ul>

## Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Glass container is suitable for laboratory quantities</li> <li>▶ <b>CARE:</b> Packing of high density product in light weight metal or plastic packages may result in container collapse with product release</li> <li>▶ Heavy gauge metal packages / Heavy gauge metal drums</li> <li>▶ Lined metal can, lined metal pail/ can.</li> <li>▶ Plastic pail.</li> <li>▶ Polyliner drum.</li> <li>▶ Packing as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul> <p>For low viscosity materials</p> <ul style="list-style-type: none"> <li>▶ Drums and jerricans must be of the non-removable head type.</li> <li>▶ Where a can is to be used as an inner package, the can must have a screwed enclosure.</li> </ul> <p>For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):</p> <ul style="list-style-type: none"> <li>▶ Removable head packaging;</li> <li>▶ Cans with friction closures and</li> </ul>
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	<ul style="list-style-type: none"> <li>▶ low pressure tubes and cartridges may be used.</li> <li>-</li> </ul> <p>Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.</p>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ <b>WARNING:</b> Avoid or control reaction with peroxides. All <i>transition metal</i> peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.</li> <li>▶ 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.</li> <li>▶ Avoid reaction with borohydrides or cyanoborohydrides</li> <li>▶ Silver or silver salts readily form explosive silver fulminate in the presence of both nitric acid and ethanol. The resulting fulminate is much more sensitive and a more powerful detonator than mercuric fulminate.</li> <li>▶ Silver and its compounds and salts may also form explosive compounds in the presence of acetylene and nitromethane.</li> <li>▶ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air.</li> <li>▶ Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.</li> </ul> <p><b>N-aminoethylpiperazine:</b></p> <ul style="list-style-type: none"> <li>▶ is a strong base in aqueous solutions</li> <li>▶ is incompatible with strong oxidisers, organic anhydrides, acrylates, alcohols, aldehydes, alkylene oxides, substituted allyls, cellulose nitrate, cresols, caprolactam solution, epichlorohydrin, ethylene dichloride, isocyanates, ketones, glycols, nitrates, organic halides, phenols, vinyl acetate</li> <li>▶ decomposes exothermically with maleic anhydride</li> <li>▶ may increase the explosive sensitivity of nitromethane</li> <li>▶ attacks aluminium, copper, magnesium, nickel, zinc, or their alloys, and galvanised steel</li> <li>▶ Phenols are incompatible with strong reducing substances such as hydrides, nitrides, alkali metals, and sulfides.</li> <li>▶ Avoid use of aluminium, copper and brass alloys in storage and process equipment.</li> <li>▶ Heat is generated by the acid-base reaction between phenols and bases.</li> <li>▶ Phenols are sulfonated very readily (for example, by concentrated sulfuric acid at room temperature), these reactions generate heat.</li> <li>▶ Phenols are nitrated very rapidly, even by dilute nitric acid.</li> <li>▶ Nitrated phenols often explode when heated. Many of them form metal salts that tend toward detonation by rather mild shock.</li> <li>▶ Avoid strong acids, bases.</li> <li>▶ Avoid contact with copper, aluminium and their alloys.</li> </ul> <p>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:</p> <ul style="list-style-type: none"> <li>▶ can react exothermically with oxidising acids to form noxious gases.</li> <li>▶ catalyse polymerisation and other reactions, particularly when finely divided</li> <li>▶ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds.</li> </ul> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Safe handling is possible in relatively low concentrations of oxygen in an inert gas.</li> <li>▶ 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.</li> <li>▶ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric.</li> </ul> <p>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.</p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Elemental metals may react with azo/diazo compounds to form explosive products.</li> <li>▶ Some elemental metals form explosive products with halogenated hydrocarbons.</li> </ul>

## SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

## Control parameters

## OCCUPATIONAL EXPOSURE LIMITS (OEL)

## INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	silver	Silver, metal	0.1 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	diethylenetriamine	Diethylene triamine	1 ppm / 4.2 mg/m <sup>3</sup>	Not Available	Not Available	Not Available

## EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
silver	Silver	0.3 mg/m <sup>3</sup>	170 mg/m <sup>3</sup>	990 mg/m <sup>3</sup>
4-nonylphenol, branched	Nonyl phenol, 4- (branched)	3.9 mg/m <sup>3</sup>	43 mg/m <sup>3</sup>	260 mg/m <sup>3</sup>
N-aminoethylpiperazine	Aminoethylpiperazine, N-	6.4 mg/m <sup>3</sup>	71 mg/m <sup>3</sup>	420 mg/m <sup>3</sup>
diethylenetriamine	Diethylenetriamine	3 ppm	8.5 ppm	51 ppm
bisphenol A	Bisphenol A; (4,4'-Isopropylidenediphenol)	15 mg/m <sup>3</sup>	110 mg/m <sup>3</sup>	650 mg/m <sup>3</sup>

Ingredient	Original IDLH	Revised IDLH
silver	10 mg/m <sup>3</sup>	Not Available
4-nonylphenol, branched	Not Available	Not Available
N-aminoethylpiperazine	Not Available	Not Available
bisphenol A diglycidyl ether diethylenetriamine reaction products	Not Available	Not Available
diethylenetriamine	Not Available	Not Available

## 8331-B Silver Conductive Epoxy Adhesive

bisphenol A	Not Available	Not Available
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**OCCUPATIONAL EXPOSURE BANDING**

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
4-nonylphenol, branched	E	≤ 0.1 ppm
N-aminoethylpiperazine	D	> 0.1 to ≤ 1 ppm
bisphenol A diglycidyl ether diethylenetriamine reaction products	D	> 0.1 to ≤ 1 ppm
bisphenol A	E	≤ 0.01 mg/m <sup>3</sup>


**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**

The adopted TLV-TWA for silver dust and fumes is 0.1 mg/m<sup>3</sup> and for the more toxic soluble silver compounds the adopted value is 0.01 mg/m<sup>3</sup>. Cases of argyria (a slate to blue-grey discolouration of epithelial tissues) have been recorded when workers were exposed to silver nitrate at concentrations of 0.1 mg/m<sup>3</sup> (as silver). Exposure to very high concentrations of silver fume has caused diffuse pulmonary fibrosis. Percutaneous absorption of silver compounds is reported to have resulted in allergy. Based on a 25% retention upon inhalation and a 10 m<sup>3</sup>/day respiratory volume, exposure to 0.1 mg/m<sup>3</sup> (TWA) would result in total deposition of no more than 1.5 gms in 25 years.

**Exposure controls**

<p><b>Appropriate engineering controls</b></p>	<p>Metal dusts must be collected at the source of generation as they are potentially explosive.</p> <ul style="list-style-type: none"> <li>▶ Avoid ignition sources.</li> <li>▶ Good housekeeping practices must be maintained.</li> <li>▶ Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions.</li> <li>▶ Do not use compressed air to remove settled materials from floors, beams or equipment</li> <li>▶ Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.</li> <li>▶ 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.</li> <li>▶ Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.</li> <li>▶ 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.</li> <li>▶ Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible.</li> <li>▶ Wet scrubbers are preferable to dry dust collectors.</li> <li>▶ Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.</li> <li>▶ Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states.</li> <li>▶ 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.</li> <li>▶ 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.</li> </ul> <p>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.</p> <table border="1" data-bbox="389 1317 1251 1384"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>welding, brazing fumes (released at relatively low velocity into moderately still air)</td> <td>0.5-1.0 m/s (100-200 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="389 1460 1091 1630"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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.</p>	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.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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<p><b>Personal protection</b></p>															
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.</li> <li>▶ Chemical goggles whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted.</li> <li>▶ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.</li> <li>▶ Alternatively a gas mask may replace splash goggles and face shields.</li> <li>▶ 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</li> </ul>														

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	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]
<b>Skin protection</b>	See Hand protection below
<b>Hands/feet protection</b>	<ul style="list-style-type: none"> <li>▶ Elbow length PVC gloves</li> </ul> <b>NOTE:</b> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> <li>▶ Protective gloves eg. Leather gloves or gloves with Leather facing</li> </ul>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ PVC Apron.</li> <li>▶ PVC protective suit may be required if exposure severe.</li> <li>▶ Eyewash unit.</li> <li>▶ Ensure there is ready access to a safety shower.</li> </ul>

## 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
BUTYL	A
NEOPRENE	C
PVC	C
VITON	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

## Respiratory protection

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, ANSI Z88 or national equivalent)

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	P1 Air-line*	-	PAPR-P1
up to 50 x ES	Air-line**	P2	PAPR-P2
up to 100 x ES	-	P3 Air-line*	-
100+ x ES	-	Air-line**	PAPR-P3

\* - Negative pressure demand \*\* - Continuous flow

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(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

## Information on basic physical and chemical properties

Appearance	Silver Grey		
<b>Physical state</b>	Solid	<b>Relative density (Water = 1)</b>	2.4
Odour	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Available	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	>20.5
<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Available
<b>Flash point (°C)</b>	93.3	Taste	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Applicable	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Applicable
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available

## SECTION 10 STABILITY AND REACTIVITY



## 8331-B Silver Conductive Epoxy Adhesive

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>▶ Unstable in the presence of incompatible materials.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 TOXICOLOGICAL INFORMATION

## Information on toxicological effects

<b>Inhaled</b>	<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>Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales.</p> <p>Inhalation of amine vapours may cause irritation of the mucous membranes of the nose and throat and lung irritation with respiratory distress and cough. Single exposures to near lethal concentrations and repeated exposures to sublethal concentrations produces tracheitis, bronchitis, pneumonitis and pulmonary oedema. Aliphatic and alicyclic amines are generally well absorbed from the respiratory tract. Systemic effects include headache, nausea, faintness and anxiety. These effects are thought to be transient and are probably related to the pharmacodynamic action of the amines. Histamine release by aliphatic amines may produce bronchoconstriction and wheezing.</p> <p>Inhalation of epoxy resin amine hardener vapours (including polyamines and amine adducts) may produce bronchospasm and coughing episodes lasting days after cessation of the exposure. Even faint traces of these vapours may trigger an intense reaction in individuals showing 'amine asthma'. The literature records several instances of systemic intoxications following the use of amines in epoxy resin systems.</p> <p>Excessive exposure to the vapours of epoxy amine curing agents may cause both respiratory irritation and central nervous system depression. Signs and symptoms of central nervous system depression, in order of increasing exposure, are headache, dizziness, drowsiness, and incoordination. In short, a single prolonged (measured in hours) or excessive inhalation exposure may cause serious adverse effects, including death.</p> <p>Not normally a hazard due to non-volatile nature of product</p> <p>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p> <p>Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
<b>Ingestion</b>	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation.</p> <p>Nonionic surfactants may produce localised irritation of the oral or gastrointestinal mucosa and induce vomiting and mild diarrhoea.</p> <p>Aliphatic and alicyclic amines are generally well absorbed from the gut. Corrosive action may cause tissue damage throughout the gastrointestinal tract. Detoxification is thought to occur in the liver, kidney and intestinal mucosa with the enzymes, monoamine oxidase and diamine oxidase (histaminase) having a significant role.</p> <p>Ingestion of amine epoxy-curing agents (hardeners) may cause severe abdominal pain, nausea, vomiting or diarrhoea. The vomitus may contain blood and mucous. If death does not occur within 24 hours there may be an improvement in the patients condition for 2-4 days only to be followed by the sudden onset of abdominal pain, board-like abdominal rigidity or hypo-tension; this indicates that delayed gastric or oesophageal corrosive damage has occurred.</p>
<b>Skin Contact</b>	<p>The material can produce severe chemical burns following direct contact with the skin.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants</p>

## 8331-B Silver Conductive Epoxy Adhesive

	<p>Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur.</p> <p>Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions.</p> <p>Individuals exhibiting 'amine dermatitis' may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis.</p> <p>NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided.</p> <p>Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep.</p> <p>Volatile amine vapours produce primary skin irritation and dermatitis. Direct local contact, with the lower molecular weight liquids, may produce skin burns. Percutaneous absorption of simple aliphatic amines is known to produce lethal effects often the same as that for oral administration. Cutaneous sensitisation has been recorded chiefly due to ethyleneamines. Histamine release following exposure to many aliphatic amines may result in 'triple response' (white vasoconstriction, red flare and wheal) in human skin.</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> <p>Phenol and some of its derivatives may produce mild to severe skin irritation on repeated or prolonged contact, producing second and third degree chemical burns. Rapid cutaneous absorption may lead to systemic toxicity affecting the cardiovascular and central nervous system.</p> <p>Absorption through the skin may result in profuse perspiration, intense thirst, nausea, vomiting, diarrhoea, cyanosis (following the formation of methaemoglobin), hyperactivity, stupor, falling blood pressure, hyperpnoea, abdominal pain, haemolysis, convulsions, coma and pulmonary oedema followed by pneumonia. Respiratory failure and kidney damage may follow.</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. Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight.</p> <p>Vapours of volatile amines cause eye irritation with lachrymation, conjunctivitis and minor transient corneal oedema which results in 'halos' around lights (glauropsia, 'blue haze', or 'blue-grey haze'). Vision may become misty and halos may appear several hours after workers are exposed to the substance</p> <p>This effect generally disappears spontaneously within a few hours of the end of exposure, and does not produce physiological after-effects. However oedema of the corneal epithelium, which is primarily responsible for vision disturbances, may take more than one or more days to clear, depending on the severity of exposure. Photophobia and discomfort from the roughness of the corneal surface also may occur after greater exposures.</p> <p>Although no detriment to the eye occurs as such, glauropsia predisposes an affected individual to physical accidents and reduces the ability to undertake skilled tasks such as driving a vehicle.</p> <p>Direct local contact with the liquid may produce eye damage which may be permanent in the case of the lower molecular weight species.</p> <p>Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation.</p> <p>Contact with the eye, by metal dusts, may produce mechanical abrasion or foreign body penetration of the eyeball.</p> <p>Irritation of the eyes may produce a heavy secretion of tears (lachrymation).</p>
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Silver is one of the most physically and physiologically cumulative of the elements. Chronic exposure to silver salts may cause argyria, a permanent ashen-grey discolouration of the skin, conjunctiva and internal organs (due to the deposit of an insoluble albuminate of silver).</p> <p>The respiratory tract may also be a site of local argyria (following chronic inhalation exposures) with a mild chronic bronchitis being the only obvious symptom.</p> <p>The alkyl phenolics (which may occur as breakdown products of some polyethoxylated surfactants) have been implicated in a phenomenon which has apparently occurred since the mid 1960s, namely lower sperm counts and reduced fertility in males. Nonyl phenol acts like an oestrogen hormone which stimulates breast cells to divide in vitro. When pregnant rats are fed nonylphenols at doses comparable to that at which humans might be exposed, male offspring had significantly smaller testicles and lower sperm counts. Although the human foetus is 'bathed' in naturally occurring oestrogens during pregnancy it is suggested that it has developed a protective mechanism against natural oestrogens but is not safe from synthetic variants. These tend to accumulate in body fats which sets them apart from the natural product. During early pregnancy, fats are broken down and may flood the body with concentrated pollutants. Drinking water may be one source of exposure to alkyl phenols as many polyethoxylated surfactants are discharged to water treatment systems where they undergo degradation.</p> <p>Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>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.</p> <p>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.</p> <p>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;</p> <ul style="list-style-type: none"> <li>▶ The route of exposure may affect the dose and site where the metal concentrates, and thus the observed toxic effects;</li> <li>▶ Metal-metal interactions can reduce or enhance toxicity; biotransformation can reduce or enhance toxicity;</li> <li>▶ 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.</li> </ul>

## 8331-B Silver Conductive Epoxy Adhesive

	<p>Prolonged exposure to some derivatives of phenol may produce dermatitis, anorexia, weight loss, weakness, muscle aches and pain, liver damage, dark urine, ochronosis, skin eruptions, diarrhoea, nervous disorders with headache, salivation, fainting, increased skin and scleral pigmentation, vertigo and mental disorders. Liver and kidney damage may also ensue. Chronic phenol toxicity was first noted in medical personnel in the late 1800s when 5 and 10% phenol was used as a skin disinfectant. The term carbolic (phenol) marasmus was given to this syndrome.</p> <p>Addition of structurally related phenolic compounds to the diet of Syrian golden hamsters induced forestomach hyperplasia and tumours. These compounds included 2(3)-tert-butyl-4-methoxyphenol (BHA) (CAS RN: 25013-16-5), 2-tert-butyl-4-methylphenol (TBMP) (29759-28-2) and p-tert-butylphenol (PTBP) (98-54-4); less active were catechol (154-23-4), p-methylphenol (331-39-5), methylhydroquinone (MHQ) (95-71-6) and pyrogallol (87-66-1), whilst no activity was seen with resorcinol (108-46-3), hydroquinone (123-31-9), propylparaben (94-13-3) and tert-butylhydroquinone (TBHQ) (1948-33-0).</p> <p>In autoradiographic studies, intake of BHA, TBMP, catechol, PMOP, PTBP and MHQ resulted in a significant increase in the labelling index of the forestomach epithelium, whilst PMOP induced epithelial damage and pyloric regenerative hyperplasia. Catechol, CA and PYMP induced similar but less marked alterations. Both catechol and PMOP increased the labelling index in the glandular stomach. The urinary bladder was free from histo-pathological lesions, but propylparabene, catechol, TBHQ and MHQ increased the labelling index. The authors of this study concluded that long term administration of PTBP and TBMP may be carcinogenic for hamster forestomach and that both 1-hydroxy and tert-butyl substituents may play a role in the induction of forestomach tumours.</p> <p>Hiros, M., et al: Carcinogenesis, Vol 7, pp 1285-1289; 1986</p>														
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<b>Legend:</b>	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														

4-NONYLPHENOL, BRANCHED

551nonphens  
for alkylphenolics category:

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	<p>The alkylphenolics may be divided into three groups.  Group I: ortho-substituted mono-alkylphenols:  Group II para-substituted mono-alkylphenols  Group III: di- and tri-substituted mixed alkyl phenols</p> <p>The subdivision of the category alkylphenols into <i>ortho</i>, <i>para</i> and the di/tri-substituted mixed members is supported by several published investigations. In assessing antimicrobial and antifouling activity of twenty-three alkylphenols, a significant difference was noted between <i>para</i> and <i>ortho</i>-substituted materials. In particular, biological activity was found to vary parabolically with increasing hydrophobicity of the <i>para</i>-substituent while introduction of a bulky substituent at the <i>ortho</i>-position resulted in a very significant decrease in antimicrobial, antifouling, and membrane-perturbation potency. Several alkylphenolic analogs of butylated hydroxytoluene (BHT) were examined for hepatotoxicity in mice depleted of hepatic glutathione. The structural requirement of both hepatic and pulmonary toxicity was a phenol ring having benzylic hydrogen atoms at the para position and an ortho-alkyl group(s) that moderately hinders the phenolic hydroxyl group. It is noteworthy that in this model, neither of the Group III members TTBP (2,4,6-tri-tert-butylphenol) nor 2,6-DTBP (2,6-di-tert-butylphenol) showed either hepatic or pulmonary toxicity. Lastly, important differences were observed in gene activation (recombinant yeast cell assay – Lac-Z reporter gene) between <i>ortho</i>-substituted and <i>para</i>-substituted alkylphenol</p> <p><b>Acute toxicity:</b> The acute (single-dose) toxicity of alkylphenols examined to date shows consistency, with LD50 values ranging from approximately 1000 mg/kg to over 2000 mg/kg. These data demonstrate a very low level of acute systemic toxicity and do not suggest any unique structural specificity, despite the general tendency for the chemicals to be, at least, irritants to skin</p> <p><b>Repeat dose toxicity:</b> The available studies for members drawn from the three groups range from 28-day and 90-day general toxicity studies, through developmental toxicity and reproductive/developmental screening, to multigeneration reproductive studies are available for some category members</p> <p>For the overall category of alkylphenols, the dosage at which the relatively mild general toxicity appears tends only to fall below 100 mg/kg/day with extended treatment, with an overall NOAEL for the category of approximately 20 mg/kg/day. No unusual and no apparent structurally unique toxicity is evident</p> <p>Repeat dose studies on OTBP (o-tert-butylphenol; Group I) and PTBP (p-tert-butylphenol; Group II) suggest the forestomach to be the main organ affected. OTBP also appears to have a mild (though statistically significant) protective effect against benzo[a]pyrene induced forestomach tumors. Long-term treatment with high dietary dose levels of PTBP caused hyperplastic changes in the forestomach epithelium of rats and hamsters, a likely consequence of the irritancy of the material. The relevance of this for human hazard is doubtful, particularly since there is no analogous structure in humans to the forestomach of rodents.</p> <p>There was no evidence of an effect on reproductive function at dosages up to 150 mg/kg. One reproductive screening study reported increased 'breeding loss' and also reduced pup weight gain and survival in early lactation at 750 mg/kg/day. It is reasonable to assume that these effects were secondary to "severe toxic symptoms" reported in the dams at this dosage. Other than an indication of a very mildly oestrogenic effect of PNP (p-nonylphenol; Group II) at a high dose levels (200-300 mg/kg/day) no effect on development was seen in a multigeneration study. By means of the classification method of Verhaar * all the alkylphenols would be classified as Type 2 compounds (polar narcotics). Narcosis, a non-specific mode of toxicity is caused by disruption (perturbation) of the cell membrane. The ability to induce narcosis is dependent on the hydrophobicity of the substance with biochemical activation or reaction involved. Such narcotic effects are also referred to as minimum or base-line toxicity. Polar narcotics such as the category phenols are usually characterised by having hydrogen bond donor activity and are thought to act by a similar mechanism to the inert, narcotic compounds but exhibit above base-line toxicity. In fact, a large number of alkylphenols have been evaluated as intravenous anesthetic agents. While the structure-activity relationships were found to be complex, the anesthetic potency and kinetics appeared to be a function of both the lipophilic character and the degree of steric hindrance exerted by ortho substituents. Less steric hindrance resulted in lower potency, while greater crowding led to complete loss of anesthetic activity and greater lipophilicity resulted in slower kinetics. These data support the notion that the alkylphenols behave as polar narcotics. In addition, the anaesthetic activity/potency differences seen with varying structure and placement of substituents strongly supports the division of alkylphenols category into the ortho, para, and di/tri-substituted groups (i.e. Group I, II and III, respectively).</p> <p><b>Genotoxicity:</b> It reasonable to consider the mutagenic potential of all the alkylphenols together because only functional group is the phenolic, which is not a structural alert for mutagenicity. The data support this, since the results of genotoxicity testing are uniformly negative for all category substances examined</p> <p>* Verhaar, H.J.M. van Leeuwen, C.J. and Hermens, J.L.M., Classifying Environmental Pollutants. 1: Structure-Activity Relationships for Prediction of Aquatic Toxicity, Chemosphere (25), pp 471 – 491 (1992).</p> <p>for nonylphenol:  Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.</p> <p>Nonylphenol was not mutagenic to Salmonella typhimurium, TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA, with or without an exogenous metabolic activation system.</p> <p>Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.</p> <p>Gastrointestinal changes, liver changes, effects on newborn recorded.</p>
N-AMINOETHYLPIPERAZINE	<p>for piperazine:  Exposure to piperazine and its salts has clearly been demonstrated to cause asthma in occupational settings. No NOAEL can be estimated for respiratory sensitisation (asthma).</p> <p>Although the LD50 levels indicate a relatively low level of oral acute toxicity (LD50 1-5 g/kg bw), signs of neurotoxicity may appear in humans after exposure to lower doses. Based on exposure levels of up to 3.4 mg/kg/day piperazine base and a LOAEL of 110 mg/kg, there is no concern for acute toxicity</p> <p>In pigs, piperazine is readily absorbed from the gastrointestinal tract, and the major part of the resorbed compound is excreted as unchanged piperazine during the first 48 hours. The principal route of excretion of piperazine and its metabolites is via urine, with a minor fraction recovered from faeces (16%). In humans the kinetics of the uptake and excretion of piperazine and its metabolites with urine appear to be roughly similar to that in the pig, and the nature and extent of conversion to metabolites has not been determined.</p> <p>Piperazine has demonstrated a low acute toxicity (LD50 = 1-5 g/kg bw) by the oral, dermal, and subcutaneous route of administration to rodents, whereas adequate inhalation toxicity data have not been found. However, there are findings of EEG (electroencephalogram) changes in 37% of 89 children administered 90-130 mg/kg piperazine (two doses during one day), corroborated by a proposed GABA (gamma-aminobutyric acid) receptor agonism exerted by piperazine. Since clinical symptoms of neurotoxicity may occur after exposure to higher doses, a LOAEL of 110 mg/kg piperazine base for acute neurotoxicity in humans after acute exposure is proposed.</p> <p>Piperazine, as concentrated aqueous solution, has strongly irritating properties with regard to skin, and should be regarded as corrosive with respect to the eye. Exposure to piperazine and its salts has been demonstrated to cause allergic dermatitis as well as respiratory sensitisation in humans. As shown by the LLNA, piperazine has a sensitising potential in animals. Although piperazine is clearly sensitising, no NOAEL can be set for this effect from the present database.</p> <p>A NOAEL of 25 mg/kg/day of piperazine for liver toxicity in the beagle dog has been chosen after repeated exposure. A LOAEL of 30 mg/kg/day of piperazine for neurotoxicity is proposed based on documentation of (rare cases) of neurotoxicity from human clinical practice. Neurotoxicity also appears in other species (e.g., rabbits, dogs, cats, tigers, and horses), but not in rodents.</p> <p>For reproductive effects of piperazine, there is a NOAEL of 125 mg/kg/day for effects on fertility, i.e., reduced pregnancy index, decreased</p>

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	<p>number of implantation sites, and decreased litter sizes in rats. The teratogenic properties have been investigated in rats and rabbits in adequate studies. In rabbit, such effects may be elicited at a dose level that is also toxic to the dam. The LOAEL is 94 mg/kg/day, and the NOAEL 42 mg/kg/day piperazine base (maternal and embryotoxic). In the rat study, there were decreases in body weight of both dams and offspring at the top dose (2,100 mg/kg/day piperazine base), but there were no signs of any malformations.</p> <p>The genotoxic properties have been investigated both <i>in vitro</i> (in the Ames test, in a nonstandard study on <i>Saccharomyces cerevisiae</i> and in Chinese hamster ovary cells) and <i>in vivo</i>, in a micronuclei assay on mice, all with negative results. There are no solid indications of a carcinogenic effect of piperazine, neither in animal studies, nor from the investigation on humans. In view of lack of genotoxic action, it appears unlikely that piperazine poses a carcinogenic risk.</p> <p>There seems to be an additional cancer risk due to the formation of N-mononitrosopiperazine (NPZ) from piperazine. It is possible to calculate a hypothetical additional cancer risk posed by NPZ after exposure to piperazine, but the calculation would depend on several assumptions. We conclude that there seems to be an additional cancer risk due to the formation of NPZ from piperazine, and although it is difficult to estimate, it is probably small.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p><b>BISPHENOL A DIGLYCIDYL ETHER DIETHYLENETRIAMINE REACTION PRODUCTS</b></p>	<p>No significant acute toxicological data identified in literature search.</p> <p>Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative.</p> <p>for 1,2-butylene oxide (ethyloxirane):</p> <p>Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m<sup>3</sup> 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/m<sup>3</sup>) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals. Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic</p>
<p><b>DIETHYLENETRIAMINE</b></p>	<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.</p> <p>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> <p>For alkyl polyamines:</p> <p>The alkyl polyamines cluster consists of organic compounds containing two terminal primary amine groups and at least one secondary amine group. Typically these substances are derivatives of ethylenediamine, propylenediamine or hexanediamine. The molecular weight range for the entire cluster is relatively narrow, ranging from 103 to 232</p> <p>Acute toxicity of the alkyl polyamines cluster is low to moderate via oral exposure and a moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin sensitisers in experimental animals. Repeated exposure in rats via the oral route indicates a range of toxicity from low to high hazard. Most cluster members gave positive results in tests for potential genotoxicity.</p> <p>Limited carcinogenicity studies on several members of the cluster showed no evidence of carcinogenicity. Unlike aromatic amines, aliphatic amines are not expected to be potential carcinogens because they are not expected to undergo metabolic activation, nor would activated intermediates be stable enough to reach target macromolecules.</p> <p>Polyamines potentiate NMDA induced whole-cell currents in cultured striatal neurons</p>
<p><b>BISPHENOL A</b></p>	<p>For bisphenol A (BPA)</p> <p>Following oral administration absorption of BPA is rapid and extensive while dermal absorption is limited. Extensive first pass metabolism occurs following absorption from the gastrointestinal tract with glucuronide conjugation being the major metabolic pathway. Bisphenol A is of low acute toxicity (rodent oral LD50 values from 3300-4100 mg/kg, a rabbit oral LD50 value 2230 mg/kg and a rat acute inhalation 6-hour LC50 value &gt;170 mg/m<sup>3</sup>). Bisphenol A is not a skin irritant, however, it is severely irritating to the eyes. BPA was negative in gene mutation and clastogenicity assays in cultured mammalian cells, as well as in a micronucleus test for clastogenicity <i>in vivo</i>; therefore, BPA is considered not to present a genotoxic concern for human health. BPA results in minimal effects on the liver and kidney (LOAEL from chronic exposure in the diet was 50 mg/kg/day). For reproductive toxicity, data from a three-generation study in the rat, BPA was not a selective reproductive toxicant at doses ranging from 0.001 to 500 mg/kg/day. BPA is not a developmental toxicant in rats or mice.</p> <p>Inconsistent findings are reported in the 'low dose' literature for bisphenol A. The inherent challenge of conducting these types of studies may be exacerbated with bisphenol A because the endpoints of concern are endocrine-mediated and potentially impacted by factors that include phytoestrogen content of the animal feed, extent of bisphenol A exposure from caging or water bottles, and the alleged sensitivity of the animal model to oestrogens.</p> <p>High-dose studies are less susceptible to these types of influences because the toxicologic response should be more robust and less variable. Several large, robust, well designed studies with multiple dose groups using several strains of rats and mice have been conducted and none of these detected any adverse reproductive effects at low to moderate dosage levels of BPA administered via the relevant route of human exposures. Further, none of these studies detected changes in prostate weight, age at puberty (rat), pathology or tumors in any tissue, or reproductive tract malformations.</p> <p>Every chemical that produces low dose cellular and molecular alterations of endocrine function also produces a cascade of effects increasing in severity resulting in clearly adverse alterations at higher doses, albeit the effects can be different from those seen at low doses. With these endocrine disrupters, but not BPA, the low dose effects are often causally linked to the high-dose adverse effects of the chemical. This is true for androgens like testosterone and trenbolone, estrogens like DES, 17beta-oestradiol and ethinyl oestradiol, xenoestrogens like methoxychlor and genistein, and antiandrogens like vinclozolin, for example. Hence, the failure of BPA to produce reproducible adverse effects via a relevant route of exposure, coupled with the lack of robustness of the many of the low dose studies (sample size, dose range, statistical analyses and experimental design, GLP) and the inability to reproduce many of these effects of any adverse effect strains the credibility of some of these study results.</p> <p>The lack of reproducibility of the low dose effects, the absence of toxicity in those low-dose-affected tissues at high-doses, and the uncertain adversity of the reported effects lead to the conclusion that there is 'minimal' concern for reproductive effects.</p> <p>In contrast, the literature on bisphenol A effects on neural and behavioral response is more consistent with respect to the number of 'positive' studies although it should be noted that the high-dose studies that proved to be the most useful for evaluating reproductive effects did not adequately assess neural and behavioral responses. In addition, even though different investigators assessed different neural and behavioral endpoints, an expert Panel concluded that the overall findings suggest that bisphenol A may be associated with neural changes in the brain and behavioral alterations related to sexual dimorphism in rodents. For this reason, the Panel expressed 'some' concern for these effects even though it is not clear the reported effects constitute an adverse toxicological response. In summary:</p> <p>For pregnant women and fetuses, the Expert Panel has different levels of concern for the different developmental endpoints that may be susceptible to bisphenol A disruption, as follows:</p> <ul style="list-style-type: none"> <li>▶ For neural and behavioral effects, the Expert Panel has some concern;</li> </ul>

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	<ul style="list-style-type: none"> <li>▶ For prostate effects, the Expert Panel has minimal concern;</li> <li>▶ For the potential effect of accelerated puberty, the Expert Panel has minimal concern; and</li> <li>▶ For birth defects and malformations, the Expert Panel has negligible concern.</li> </ul> <p>For infants and children, the Expert Panel has the following levels of concern for biological processes that might be altered by Bisphenol A, as follows:</p> <ul style="list-style-type: none"> <li>▶ Some concern for neural and behavioral effects; and</li> <li>▶ Minimal concern for the effect of accelerated puberty.</li> </ul> <p>For adults, the Expert Panel has negligible concern for adverse reproductive effects following exposures in the general population to Bisphenol A. For highly exposed subgroups, such as occupationally exposed populations, the level of concern is elevated to minimal.</p> <p><b>NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Bisphenol A</b>  <b>National Toxicology Program US Department of Health and Human Services September 2008 NTP Publication No 08-5994</b></p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>		
8331-B Silver Conductive Epoxy Adhesive & 4-NONYLPHENOL, BRANCHED & N-AMINOETHYLPIPERAZINE & BISPHENOL A DIGLYCIDYL ETHER DIETHYLENTRIAMINE REACTION PRODUCTS & DIETHYLENTRIAMINE & BISPHENOL A	<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>		
8331-B Silver Conductive Epoxy Adhesive & N-AMINOETHYLPIPERAZINE & BISPHENOL A DIGLYCIDYL ETHER DIETHYLENTRIAMINE REACTION PRODUCTS & DIETHYLENTRIAMINE & BISPHENOL A	<p>The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>		
8331-B Silver Conductive Epoxy Adhesive & BISPHENOL A DIGLYCIDYL ETHER DIETHYLENTRIAMINE REACTION PRODUCTS & BISPHENOL A	<p>The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics. Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities. Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.</p>		
8331-B Silver Conductive Epoxy Adhesive & N-AMINOETHYLPIPERAZINE & DIETHYLENTRIAMINE	<p>Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds.</p> <p>Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines.</p> <p>In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper</p>		
4-NONYLPHENOL, BRANCHED & DIETHYLENTRIAMINE	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>		
4-NONYLPHENOL, BRANCHED & N-AMINOETHYLPIPERAZINE & DIETHYLENTRIAMINE	<p>The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p>		
Acute Toxicity	✓	Carcinogenicity	✗
Skin Irritation/Corrosion	✓	Reproductivity	✓
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✗
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	✗
Mutagenicity	✗	Aspiration Hazard	✗

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

## SECTION 12 ECOLOGICAL INFORMATION

## Toxicity

8331-B Silver Conductive Epoxy Adhesive	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...

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

4-nonylphenol, branched	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.017mg/L	4
	EC50	48	Crustacea	0.0844mg/L	2
	EC50	96	Algae or other aquatic plants	0.027mg/L	2
	BCF	24	Fish	0.193mg/L	4
	EC10	96	Algae or other aquatic plants	0.012mg/L	4
NOEC	168	Crustacea	0.001mg/L	2	

N-aminoethylpiperazine	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	2-190mg/L	2
	EC50	48	Crustacea	32mg/L	2
	EC50	72	Algae or other aquatic plants	>1-mg/L	2
	EC100	48	Crustacea	100mg/L	2
	NOEC	96	Fish	1-30mg/L	2

bisphenol A diglycidyl ether diethylenetriamine reaction products	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

diethylenetriamine	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-14mg/L	2
	EC50	48	Crustacea	=16mg/L	1
	EC50	72	Algae or other aquatic plants	1-164mg/L	2
	NOEC	504	Crustacea	=5.6mg/L	1

bisphenol A	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	3.275mg/L	3
	EC50	48	Crustacea	3.9mg/L	2
	EC50	96	Algae or other aquatic plants	1mg/L	5
	BCF	288	Fish	0.556mg/L	4
	NOEC	Not Available	Crustacea	0.001-mg/L	2

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

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

Very toxic to aquatic organisms, 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.

Surfactants are in general toxic to aquatic organisms due to their surface-active properties. Historically, synthetic surfactants were often composed of branched alkyl chains resulting in poor biodegradability which led to concerns about their environmental effects. Today however, many of them, for example those used in large amounts, globally, as detergents, are linear and therefore readily biodegradable and considered to be of rather low risk to the environment. A linear structure of the hydrophobic chain facilitates the approach of microorganism while branching, in particular at the terminal position, inhibits biodegradation. Also, the bioaccumulation potential of surfactants is usually low due to the hydrophilic units. Linear surfactants are not always preferred however, as some branching (that ideally does not hinder ready biodegradability) is often preferable from a performance point of view. The reduction in waste water of organic contaminants such as surfactants can either be a consequence of adsorption onto sludge or aerobic biodegradation in the biological step. Similar sorption and degradation processes occur in the environment as a consequence of direct release of surfactants into the environment from product use, or through effluent discharge from sewage treatment plants in surface waters or the application of sewage sludge on land. However, a major part of surfactants in waste water will be efficiently eliminated in the sewage treatment plant. Although toxic to various organisms, surfactants in general only have a limited effect on the bacteria in the biological step. There are occasions however, where adverse effects have been noticed due to e.g. large accidental releases of softeners from laundry companies.

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.

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A metal ion is considered infinitely persistent because it cannot degrade further.

The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.

The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.

Environmental processes may enhance bioavailability.

For bisphenol A and related bisphenols:

Environmental fate:

Biodegradability (28 d) 89% - Easily biodegradable

Bioconcentration factor (BCF) 7.8 mg/l

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

Substance does not meet the criteria for PBT or vPvB according to Regulation (EC) No 1907/2006, Annex XIII

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

Ecotoxicity:

Fish LC50 (96 h): 4.6 mg/l (freshwater fish); 11 mg/l (saltwater fish); NOEC 0.016 mg/l (freshwater fish- 144 d); 0.064 mg/l (saltwater fish 164 d)

Fresh water invertebrates EC50 (48 h): 10.2 mg/l; NOEC 0.025 mg/l - 328 d)

Marine water invertebrate EC50 (96 h): 1.1 mg/l; NOEC 0.17 mg/l (28 d)

Freshwater algae (96 h): 2.73 mg/l

Marine water algae (96 h): 1.1 mg/l

Fresh water plant EC50 (7 d): 20 mg/l; NOEC 7.8 mg/l

In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms.

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

A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations.

A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.

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

Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F ((bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem, Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe<sup>3+</sup> ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.

Environmental toxicity is a function of the n-octanol/ water partition coefficient (log Pow, log Kow). Phenols with log Pow >7.4 are expected to exhibit low toxicity to aquatic organisms. However the toxicity of phenols with a lower log Pow is variable, ranging from low toxicity (LC50 values >100 mg/l) to highly toxic (LC50 values <1 mg/l) dependent on log Pow, molecular weight and substitutions on the aromatic ring. Dinitrophenols are more toxic than predicted from QSAR estimates. Hazard information for these groups is not generally available.

For ethyleneamines:

Adsorption of the ethyleneamines correlates closely with both the cation exchange capacity (CEC) and organic content of the soil. Soils with increased CEC and organic content exhibited higher affinities for these amines. This dependence of adsorption on CEC and organic content is most likely due to the strong electrostatic interaction between the positively charged amine and the negatively charged soil surface.

For silver and its compounds:

**Environmental fate:**

Silver is a rare but naturally occurring metal, often found deposited as a mineral ore in association with other elements. Emissions from smelting operations, manufacture and disposal of certain photographic and electrical supplies, coal combustion, and cloud seeding are some of the anthropogenic sources of silver in the biosphere. The global biogeochemical movements of silver are characterized by releases to the atmosphere, water, and land by natural and anthropogenic sources, long-range transport of fine particles in the atmosphere, wet and dry deposition, and sorption to soils and sediments.

In general, accumulation of silver by terrestrial plants from soils is low, even if the soil is amended with silver-containing sewage sludge or the plants are grown on tailings from silver mines, where silver accumulates mainly in the root systems.

The ability to accumulate dissolved silver varies widely between species. Some reported bioconcentration factors for marine organisms (calculated as milligrams of silver per kilogram fresh weight organism divided by milligrams of silver per litre of medium) are 210 in diatoms, 240 in brown algae, 330 in mussels, 2300 in scallops, and 18 700 in oysters, whereas bioconcentration factors for freshwater organisms have been reported to range from negligible in bluegills (*Lepomis macrochirus*) to 60 in daphnids; these values represent uptake of bioavailable silver in laboratory experiments. Laboratory studies with the less toxic silver compounds, such as silver sulfide and silver chloride, reveal that accumulation of silver does not necessarily lead to adverse effects. At concentrations normally encountered in the environment, food-chain biomagnification of silver in aquatic systems is unlikely. Elevated silver concentrations in biota occur in the vicinities of sewage outfalls, electroplating plants, mine waste sites, and silver iodide-seeded areas. Maximum concentrations recorded in field collections, in milligrams total silver per kilogram dry weight (tissue), were 1.5 in marine mammals (liver) (except Alaskan beluga whales *Delphinapterus leucas*, which had concentrations 2 orders of magnitude higher than those of other marine mammals), 6 in fish (bone), 14 in plants (whole), 30 in annelid worms (whole), 44 in birds (liver), 110 in mushrooms (whole), 185 in bivalve molluscs (soft parts), and 320 in gastropods (whole).

**Ecotoxicity:**

In general, silver ion was less toxic to freshwater aquatic organisms under conditions of low dissolved silver ion concentration and increasing water pH, hardness, sulfides, and dissolved and particulate organic loadings; under static test conditions, compared with flow-through regimens; and when animals were adequately nourished instead of being starved. Silver ions are very toxic to microorganisms. However, there is generally no strong inhibitory effect on microbial activity in sewage treatment plants because of reduced bioavailability due to rapid complexation and adsorption. Free silver ion was lethal to representative species of sensitive aquatic plants, invertebrates, and teleosts at nominal water concentrations of 1-5 ug/litre. Adverse effects occur on development of trout at concentrations as low as 0.17 ug/litre and on phytoplankton species composition and succession at 0.3-0.6 ug/litre.

A knowledge of the speciation of silver and its consequent bioavailability is crucial to understanding the potential risk of the metal. Measurement of free ionic silver is the only direct method that can be used to assess the likely effects of the metal on organisms. Speciation models can be used to assess the likely proportion of the total silver measured that is bioavailable to organisms. Unlike some other metals, background freshwater concentrations in pristine and most urban areas are well below concentrations causing toxic effects. Levels in most industrialized areas border on the effect concentration, assuming that conditions favour bioavailability. On the basis of available toxicity test results, it is unlikely that bioavailable free silver ions would ever be at sufficiently high concentrations to cause toxicity in marine environments.

No data were found on effects of silver on wild birds or mammals. Silver was harmful to poultry (tested as silver nitrate) at concentrations as low as 100 mg total silver/litre in drinking-water or 200 mg total silver/kg in diets. Sensitive laboratory mammals were adversely affected at total silver concentrations (added as silver nitrate) as low as 250 ug/litre in drinking-water (brain histopathology), 6 mg/kg in diet (high accumulations in kidneys and liver), or 13.9 mg/kg body weight (lethality).

Silver and Silver Compounds; Concise International Chemical Assessment Document (CICAD) 44 IPCS InChem (WHO)

'The transport of silver through estuarine and coastal marine systems is dependent on biological uptake and incorporation. Uptake by phytoplankton is rapid, in proportion to silver

Continued...



## 8331-B Silver Conductive Epoxy Adhesive

concentration and inversely proportional to salinity. In contrast to studies performed with other toxic metals, silver availability appears to be controlled by both the free silver ion concentration and the concentration of other silver complexes. Silver incorporated by phytoplankton is not lost as salinity increases; as a result silver associated with cellular material is largely retained within the estuary. Phytoplankton exhibit a variable sensitivity to silver. Sensitive species exhibit a marked delay in the onset of growth in response to silver at low concentrations, even though maximum growth rates are similar to controls. A delay in the onset of growth reduces the ability of a population to respond to short-term favourable conditions and to succeed within the community.

James G. Saunders and George R Abbe: Aquatic Toxicology and Environmental Fate; ASTM STP 1007, 1989, pp 5-18

For surfactants:

**Environmental fate:**

Octanol/water partition coefficients cannot easily be determined for surfactants because one part of the molecule is hydrophilic and the other part is hydrophobic. Consequently they tend to accumulate at the interface and are not extracted into one or other of the liquid phases. As a result surfactants are expected to transfer slowly, for example, from water into the flesh of fish. During this process, readily biodegradable surfactants are expected to be metabolised rapidly during the process of bioaccumulation. This was emphasised by the OECD Expert Group stating that chemicals are not to be considered to show bioaccumulation potential if they are readily biodegradable.

Surfactants show a complex solubility behaviour due to aggregation. The monomer concentration, and hence the thermodynamic activity, reaches a limiting value at the critical micelle concentration (CMC). It remains approximately constant as the total concentration is further increased. For ecotoxicological models requiring a solubility value, the critical micelle concentration is therefore the appropriate parameter describing water solubility of surface active materials.

Surfactants can form dispersions or emulsions in which the bioavailability for aquatic toxicity studies is difficult to ascertain, even with careful solution preparation. Micelle formation can result in an overestimation of the bioavailable fraction even when "solutions" are apparently formed. This presents significant problems of interpretation of aquatic toxicity test results for surface active materials. The so-called critical micelle concentration (CMC) is related to surface tension produced by the substance and is the key value for actual water solubility of the substance.

Several anionic and nonionic surfactants have been investigated to evaluate their potential to bioconcentrate in fish. BCF values (BCF - bioconcentration factor) ranging from 1 to 350 were found. These are absolute maximum values, resulting from the radiolabelling technique used. In all these studies, substantial oxidative metabolism was found resulting in the highest radioactivity in the gall bladder. This indicates liver transformation of the parent compound and biliary excretion of the metabolised compounds, so that 'real' bioconcentration is overstated. After correction it can be expected that 'real' parent BCF values are one order of magnitude less than those indicated above, i.e. 'real' BCF is <100. Therefore the usual data used for classification by EU directives to determine whether a substance is 'Dangerous to the Environment' has little bearing on whether the use of the surfactant is environmentally acceptable.

**Ecotoxicity:**

Surfactant should be considered to be toxic (EC50 and LC50 values of < 10 mg/L) to aquatic species under conditions that allow contact of the chemicals with the organisms. The water solubility of the chemicals does not impact the toxicity except as it relates to the ability to conduct tests appropriately to obtain exposure of the test species. The acute aquatic toxicity generally is considered to be related to the effects of the surfactant properties on the organism and not to direct chemical toxicity.

for alkylphenols and their ethoxylates, or propoxylates:

**Environmental fate:** Alkylphenols are ubiquitous in the environment after the introduction, generally as wastes, of their alkoxylated forms (ethoxylates and propoxylates, for example); these are extensively used throughout industry and in the home.

Alkylphenol ethoxylates are widely used surfactants in domestic and industrial products, which are commonly found in wastewater discharges and in sewage treatment plant (STP) effluents. Degradation of APES in wastewater treatment plants or in the environment generates more persistent shorter-chain APES and alkylphenols (APs) such as nonylphenol (NP), octylphenol (OP) and AP mono- to triethoxylates (NPE1, NPE2 and NPE3). There is concern that APE metabolites (NP, OP, NPE1-3) can mimic natural hormones and that the levels present in the environment may be sufficient to disrupt endocrine function in wildlife and humans. The physicochemical properties of the APE metabolites (NP, NPE1-4, OP, OPE1-4), in particular the high Kow values, indicate that they will partition effectively into sediments following discharge from STPs. The aqueous solubility data for the APE metabolites indicate that the concentration in water combined with the high partition coefficients will provide a significant reservoir (load) in various environmental compartments. Data from studies conducted in many regions across the world have shown significant levels in samples of every environmental compartment examined. In the US, levels of NP in air ranged from 0.01 to 81 ng/m<sup>3</sup>, with seasonal trends observed. Concentrations of APE metabolites in treated wastewater effluents in the US ranged from < 0.1 to 369 ug/l, in Spain they were between 6 and 343 ug/l and concentrations up to 330 ug/l were found in the UK. Levels in sediments reflected the high partition coefficients with concentrations reported ranging from < 0.1 to 13,700 ug/kg for sediments in the US. Fish in the UK were found to contain up to 0.8 ug/kg NP in muscle tissue. APES degraded faster in the water column than in sediment. Aerobic conditions facilitate easier further biotransformation of APE metabolites than anaerobic conditions.

Nonylphenols are susceptible to photochemical degradation. Using natural, filtered, lake water it was found that nonylphenol had a half-life of approximately 10-15 h under continuous, noon, summer sun in the surface water layer, with a rate approximately 1.5 times slower at depths 20-25 cm. Photolysis was much slower with ethoxylated nonylphenol, and so it is unlikely to be a significant event in removal of the ethoxylates.

**Air:** Alkylphenols released to the atmosphere will exist in the vapour phase and is thought to be degraded by reaction with photochemically produced hydroxyl radicals, with a calculated half-life, for nonylphenol, of 0.3 days.

**Water:** Abiotic degradation of alkylphenol is negligible. Biodegradation does not readily take place. The half-life in surface water may be around 30 days.

**Degradation:** Alkylphenol ethoxylates (APES) may abiotically degrade into the equivalent alkylphenol. During degradation ethylene oxide units are cleaved off the ethylene oxide chain until only short-chain alkylphenol ethoxylates remain, typically mono- and diethylene oxides. Oxidation of these oligomers creates the corresponding carboxylic acids. This leaves several degradation products: short-chain ethoxylates, their carboxylic acids, and alkylphenols.

**Biodegradation:** Alkylphenols are not readily biodegradable. Several mechanisms of microbial aromatic ring degradation have been reported, the most common being formation of catechol from phenol, followed by ring scission between or adjacent to the two hydroxyl groups.

The full breakdown pathway for APES has not yet been determined, and all studies have so far focused on identification of intermediates in bacterial culture media, rather than studying cell-free systems or purified enzymes. It is, however, likely that microbial metabolism usually starts by an attack on the ethoxylate chain, rather than on the ring or the hydrophobic chain. The ethoxylate groups are progressively removed, either by ether cleavage, or by terminal alcohol oxidation followed by cleavage of the resulting carboxylic acid. Biodegradation of APES produces less biodegradable products: alkylphenol mono- and di-ethoxylates, alkylphenoxy acetic and alkylphenoxypolyethoxy acetic acids, and alkylphenols. These metabolites frequently persist through sewage treatment and in rivers. Anaerobic conditions generally lead to the accumulation of alkylphenols. The rate of biodegradation seems to decrease with increasing length of the ethylene oxide chain.

**Bioaccumulation:** Metabolites of APES accumulate in organisms, with bioconcentration factors varying from ten to several thousand, depending on species, metabolite and organ. The metabolites of APES are generally more toxic than the original compounds. APES have LC50s above about 1.5 mg/l, whereas alkylphenols, such as nonylphenol, have LC50s are generally around 0.1 mg/l.

**Oestrogenic activity:** The role of alkyl chain length and branching, substituent position, number of alkylated groups, and the requirement of a phenolic ring structure was assessed in fish. The results showed that most alkylphenols were oestrogenic, although with 3-300 thousand times lower potency than the endogenous estrogen 17beta-estradiol.

Mono-substituted tertiary alkylphenols with moderate (C4-C5) and long alkyl chain length (C8-C9) in the para position exhibited the highest oestrogenic potency. Substitution with multiple alkyl groups, presence of substituents in the ortho- and meta-position and lack of a hydroxyl group on the benzene ring reduced the oestrogenic activity, although several oestrogenic alkylated non-phenolics were identified.

**Human exposure:** Alkylphenols were first found to be oestrogenic (oestrogen-mimicking) in the 1930s, but more recent research has highlighted the implications of these effects. The growth of cultured human breast cancer cells is affected by nonylphenol at concentrations as low as 1 uM (220 ug/l) or concentrations of octylphenol as low as 0.1 uM (20 ug/l). Oestrogenic effects have also been shown on rainbow trout hepatocytes, chicken embryo fibroblasts and a mouse oestrogen receptor.

The insecticide chlordecone (Kepone) shows similar behaviour to alkylphenols, accumulating in liver and adipose tissue, and eliciting oestrogenic activity. Workers exposed to this insecticide can suffer reproductive effects such as low sperm counts and sterility. In addition, the oestrogenic effects of chlordecone on MCF7 cells occur at similar concentrations to those of alkylphenols, suggesting that alkylphenols will be a similar health hazard if target cells are exposed to uM levels of these compounds.

By comparing environmental concentrations, bioconcentration factors and *in vitro* oestrogenic effect levels, current environmental levels of alkylphenolic compounds are probably high enough to affect the hormonal control systems of some organisms. It is also possible that human health could be being affected.

Prevent, by any means available, spillage from entering drains or water courses.

**DO NOT discharge into sewer or waterways.**

**Persistence and degradability**

Ingredient	Persistence: Water/Soil	Persistence: Air
4-nonylphenol, branched	HIGH	HIGH
N-aminoethylpiperazine	HIGH	HIGH
diethylenetriamine	LOW	LOW
bisphenol A	HIGH (Half-life = 360 days)	LOW (Half-life = 0.31 days)

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## Bioaccumulative potential

Ingredient	Bioaccumulation
4-nonylphenol, branched	LOW (BCF = 271)
N-aminoethylpiperazine	LOW (LogKOW = -1.5677)
diethylenetriamine	LOW (BCF = 1.7)
bisphenol A	LOW (BCF = 100)

## Mobility in soil

Ingredient	Mobility
4-nonylphenol, branched	LOW (KOC = 56010)
N-aminoethylpiperazine	LOW (KOC = 171.7)
diethylenetriamine	LOW (KOC = 87.53)
bisphenol A	LOW (KOC = 75190)


## SECTION 13 DISPOSAL CONSIDERATIONS

## Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> <li>▶ Containers may still present a chemical hazard/ danger when empty.</li> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Recycle wherever possible.</li> <li>▶ 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.</li> <li>▶ Treat and neutralise at an approved treatment plant.</li> <li>▶ Treatment should involve: Mixing or slurring in water; Neutralisation with suitable dilute acid followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material).</li> <li>▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.</li> </ul>
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## SECTION 14 TRANSPORT INFORMATION

## Labels Required

		Limited quantity: 8331-14G, 8331-50ML, 8331-200ML
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## Land transport (ADG)

UN number	3263				
UN proper shipping name	CORROSIVE SOLID, BASIC, ORGANIC, N.O.S. (contains silver, N-aminoethylpiperazine and 4-nonylphenol, branched)				
Transport hazard class(es)	<table style="width: 100%; border-collapse: collapse;"> <tr> <td style="border-right: 1px dashed black; padding-right: 5px;">Class</td> <td style="padding-left: 5px;">8</td> </tr> <tr> <td style="border-right: 1px dashed black; padding-right: 5px;">Subrisk</td> <td style="padding-left: 5px;">Not Applicable</td> </tr> </table>	Class	8	Subrisk	Not Applicable
Class	8				
Subrisk	Not Applicable				
Packing group	II				
Environmental hazard	Environmentally hazardous				
Special precautions for user	<table style="width: 100%; border-collapse: collapse;"> <tr> <td style="border-right: 1px dashed black; padding-right: 5px;">Special provisions</td> <td style="padding-left: 5px;">274</td> </tr> <tr> <td style="border-right: 1px dashed black; padding-right: 5px;">Limited quantity</td> <td style="padding-left: 5px;">1 kg</td> </tr> </table>	Special provisions	274	Limited quantity	1 kg
Special provisions	274				
Limited quantity	1 kg				

## Air transport (ICAO-IATA / DGR)

UN number	3263						
UN proper shipping name	Corrosive solid, basic, organic, n.o.s. * (contains silver, N-aminoethylpiperazine and 4-nonylphenol, branched)						
Transport hazard class(es)	<table style="width: 100%; border-collapse: collapse;"> <tr> <td style="border-right: 1px dashed black; padding-right: 5px;">ICAO/IATA Class</td> <td style="padding-left: 5px;">8</td> </tr> <tr> <td style="border-right: 1px dashed black; padding-right: 5px;">ICAO / IATA Subrisk</td> <td style="padding-left: 5px;">Not Applicable</td> </tr> <tr> <td style="border-right: 1px dashed black; padding-right: 5px;">ERG Code</td> <td style="padding-left: 5px;">8L</td> </tr> </table>	ICAO/IATA Class	8	ICAO / IATA Subrisk	Not Applicable	ERG Code	8L
ICAO/IATA Class	8						
ICAO / IATA Subrisk	Not Applicable						
ERG Code	8L						
Packing group	II						
Environmental hazard	Environmentally hazardous						

## 8331-B Silver Conductive Epoxy Adhesive

<b>Special precautions for user</b>	Special provisions	A3 A803
	Cargo Only Packing Instructions	863
	Cargo Only Maximum Qty / Pack	50 kg
	Passenger and Cargo Packing Instructions	859
	Passenger and Cargo Maximum Qty / Pack	15 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Y844
	Passenger and Cargo Limited Maximum Qty / Pack	5 kg

**Sea transport (IMDG-Code / GGVSee)**

<b>UN number</b>	3263	
<b>UN proper shipping name</b>	CORROSIVE SOLID, BASIC, ORGANIC, N.O.S. (contains silver, N-aminoethylpiperazine and 4-nonylphenol, branched)	
<b>Transport hazard class(es)</b>	IMDG Class	8
	IMDG Subrisk	Not Applicable
<b>Packing group</b>	II	
<b>Environmental hazard</b>	Marine Pollutant	
<b>Special precautions for user</b>	EMS Number	F-A , S-B
	Special provisions	274
	Limited Quantities	1 kg

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

Not Applicable

**SECTION 15 REGULATORY INFORMATION****Safety, health and environmental regulations / legislation specific for the substance or mixture****SILVER IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards  
Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2  
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**4-NONYLPHENOL, BRANCHED IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2  
Chemical Footprint Project - Chemicals of High Concern List

**N-AMINOETHYLPIPERAZINE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
Australia Inventory of Chemical Substances (AICS)

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

**BISPHENOL A DIGLYCIDYL ETHER DIETHYLENTRIAMINE REACTION PRODUCTS IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)

**DIETHYLENTRIAMINE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards  
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C  
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

**BISPHENOL A IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2  
Chemical Footprint Project - Chemicals of High Concern List

**National Inventory Status**

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (N-aminoethylpiperazine; bisphenol A diglycidyl ether diethylenetriamine reaction products; bisphenol A; diethylenetriamine; silver)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (bisphenol A diglycidyl ether diethylenetriamine reaction products; silver)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes

Continued...

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USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (bisphenol A diglycidyl ether diethylenetriamine reaction products)
Vietnam - NCI	Yes
Russia - ARIPS	No (bisphenol A diglycidyl ether diethylenetriamine reaction products)
<b>Legend:</b>	<i>Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)</i>

## SECTION 16 OTHER INFORMATION

<b>Revision Date</b>	12/05/2020
<b>Initial Date</b>	06/03/2020

## Other information

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

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

## Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average  
 PC—STEL: Permissible Concentration-Short Term Exposure Limit  
 IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit.  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 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