



SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A

On-Crete Australia Pty Ltd

Chemwatch Hazard Alert Code: 3

Version No: 3.13.1.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 22/04/2021

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

Product Identifier

Product name	SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid)
Other means of identification	Not Available

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

Relevant identified uses	Part A of epoxy resin concrete coating system
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Details of the supplier of the safety data sheet

Registered company name	On-Crete Australia Pty Ltd	On-Crete Australia Pty Ltd
Address	4/489 Scottsdale Drive QLD 4227 Australia	4/489 Scottsdale Drive QLD 4227 Australia
Telephone	+61 7 5593 6884	+61 7 5593 6884
Fax		
Website	www.on-crete.com.au	www.on-crete.com.au
Email	sales@on-crete.com.au	sales@on-crete.com.au

Emergency telephone number

Association / Organisation	On-Crete Australia Pty Ltd	On-Crete Australia Pty Ltd
Emergency telephone numbers	1300 292 504	1300 292 504
Other emergency telephone numbers	+613 6121 9073	+613 6121 9073

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

ChemWatch Hazard Ratings

	Min	Max	
Flammability	0		
Toxicity	0		
Body Contact	2		0 = Minimum 1 = Low 2 = Moderate 3 = High 4 = Extreme
Reactivity	2		
Chronic	3		

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

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H319	Causes serious eye irritation.
H411	Toxic to aquatic life with long lasting effects.
H340	May cause genetic defects.
H315	Causes skin irritation.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H317	May cause an allergic skin reaction.
AUH019	May form explosive peroxides.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves/protective clothing/eye protection/face protection/hearing protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.

Precautionary statement(s) Storage

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
25068-38-6	>60	<u>bisphenol A/ diglycidyl ether resin, liquid</u>
9003-36-5	10-30	<u>bisphenol F diglycidyl ether copolymer</u>

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CAS No	%[weight]	Name
108-32-7	<10	<u>propylene carbonate</u>
68609-97-2	<10	<u>(C12-14)alkylglycidyl ether</u>
41556-26-7	<10	<u>bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate</u>
82919-37-7	<1	<u>methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate</u>
22464-99-9	<1	<u>zirconium 2-ethylhexanoate</u>
2530-83-8	<1	<u>gamma-glycidoxypropyltrimethoxysilane</u>
Legend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available		

SECTION 4 First aid measures

Description of first aid measures

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

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> Alert Fire Brigade and tell them location and nature of hazard. Wear full body protective clothing with breathing apparatus. Prevent, by any means available, spillage from entering drains or water course. Use water delivered as a fine spray to control fire and cool adjacent area. Avoid spraying water onto liquid pools. DO NOT approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> Combustible. Slight fire hazard when exposed to heat or flame. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO).

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	<ul style="list-style-type: none"> ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) aldehydes metal oxides other pyrolysis products typical of burning organic material.</p>
HAZCHEM	•3Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> • In the event of a spill of a reactive diluent, the focus is on containing the spill to prevent contamination of soil and surface or ground water. • If irritating vapors are present, an approved air-purifying respirator with organic vapor canister is recommended for cleaning up spills and leaks. • For small spills, reactive diluents should be absorbed with sand. <p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	<p>Environmental hazard - contain spillage.</p> <p>Industrial spills or releases of reactive diluents are infrequent and generally contained. If a large spill does occur, the material should be captured, collected, and reprocessed or disposed of according to applicable governmental requirements. An approved air-purifying respirator with organic-vapor canister is recommended for emergency work.</p> <p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling.
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	<ul style="list-style-type: none"> Work clothes should be laundered separately. Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions. DO NOT allow clothing wet with material to stay in contact with skin
Other information	<ul style="list-style-type: none"> Store in original containers. Keep containers securely sealed. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> Metal can or drum Packaging as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<p>Glycidyl ethers:</p> <ul style="list-style-type: none"> may form unstable peroxides on storage in air, light, sunlight, UV light or other ionising radiation, trace metals - inhibitor should be maintained at adequate levels may polymerise in contact with heat, organic and inorganic free radical producing initiators may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide attack some forms of plastics, coatings, and rubber <p>Reactive diluents are stable under recommended storage conditions, but can decompose at elevated temperatures. In some cases, decomposition can cause pressure build-up in closed systems.</p> <ul style="list-style-type: none"> Avoid cross contamination between the two liquid parts of product (kit). If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur. This excess heat may generate toxic vapour Avoid reaction with amines, mercaptans, strong acids and oxidising agents



X — Must not be stored together

O — May be stored together with specific precautions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

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	zirconium 2-ethylhexanoate	Zirconium compounds (as Zr)	5 mg/m3	10 mg/m3	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
bisphenol A/ diglycidyl ether resin, liquid	90 mg/m3	990 mg/m3	5,900 mg/m3
propylene carbonate	34 mg/m3	370 mg/m3	2,200 mg/m3
gamma-glycidioxypropyltrimethoxysilane	9.3 mg/m3	100 mg/m3	230 mg/m3

Ingredient	Original IDLH	Revised IDLH
bisphenol A/ diglycidyl ether	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
resin, liquid		
bisphenol F diglycidyl ether copolymer	Not Available	Not Available
propylene carbonate	Not Available	Not Available
(C12-14)alkylglycidyl ether	Not Available	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available	Not Available
zirconium 2-ethylhexanoate	25 mg/m3	Not Available
gamma-glycidioxypropyltrimethoxysilane	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
bisphenol A/ diglycidyl ether resin, liquid	E	≤ 0.1 ppm
bisphenol F diglycidyl ether copolymer	E	≤ 0.1 ppm
propylene carbonate	E	≤ 0.1 ppm
(C12-14)alkylglycidyl ether	E	≤ 0.1 ppm
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	D	> 0.1 to ≤ 1 ppm
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	D	> 0.1 to ≤ 1 ppm
gamma-glycidioxypropyltrimethoxysilane	E	≤ 0.1 ppm
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.	

MATERIAL DATA

Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.

OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

For epichlorohydrin

Odour Threshold Value: 0.08 ppm

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

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

Odour Safety Factor (OSF)

OSF=0.54 (EPICHLOROHYDRIN)

Exposure controls

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

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

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

Within each range the appropriate value depends on:

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

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

Personal protection



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

NOTE:

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

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

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

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

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

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

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- dexterity
- Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
 - Contaminated gloves should be replaced.
- As defined in ASTM F-739-96 in any application, gloves are rated as:
- Excellent when breakthrough time > 480 min
 - Good when breakthrough time > 20 min
 - Fair when breakthrough time < 20 min
 - Poor when glove material degrades
- For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
- It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
- Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
- Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:
- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
 - Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential
- Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
- When handling liquid-grade epoxy resins wear chemically protective gloves, boots and aprons.
- The performance, based on breakthrough times, of:
- Ethyl Vinyl Alcohol (EVAL laminate) is generally excellent
 - Butyl Rubber ranges from excellent to good
 - Nitrile Butyl Rubber (NBR) from excellent to fair.
 - Neoprene from excellent to fair
 - Polyvinyl (PVC) from excellent to poor
- As defined in ASTM F-739-96
- Excellent breakthrough time > 480 min
 - Good breakthrough time > 20 min
 - Fair breakthrough time < 20 min
 - Poor glove material degradation
- Gloves should be tested against each resin system prior to making a selection of the most suitable type. Systems include both the resin and any hardener, individually and collectively)
- **DO NOT** use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin).
 - **DO NOT** use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use.
- Replacement time should be considered when selecting the most appropriate glove. It may be more effective to select a glove with lower chemical resistance but which is replaced frequently than to select a more resistant glove which is reused many times

Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

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

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

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

^ - Full-face

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

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compounds(below 65 degC)

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	<p>Reactive diluents are generally colourless to yellow/ amber, low viscosity liquids with mild ether-like odour; solubility in water varies across the family. Substitution on the phenolic rings may generate solids. Reactive diluents may contain trace residuals of epichlorohydrin a known skin irritant.</p> <p>Bisphenol A epoxy resin.</p> <p>Important epoxy resins are produced from combining epichlorohydrin and bisphenol A to give bisphenol A diglycidyl ethers. Increasing the ratio of bisphenol A to epichlorohydrin during manufacture produces higher molecular weight linear polyethers with glycidyl end groups, which are semi-solid to hard crystalline materials at room temperature depending on the molecular weight achieved. As the molecular weight of the resin increases, the epoxide content reduces and the material behaves more and more like a thermoplastic. Very high molecular weight polycondensates (ca. 30 000 – 70 000 g/mol) form a class known as phenoxy resins and contain virtually no epoxide groups (since the terminal epoxy groups are insignificant compared to the total size of the molecule). These resins do however contain hydroxyl groups throughout the backbone, which may also undergo other cross-linking reactions, e.g. with aminoplasts, phenoplasts and isocyanates.</p> <p>White/clear</p>		
Physical state	Liquid	Relative density (Water= 1)	1.08
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	7-8	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	>100	Taste	Not Available
Evaporation rate	Not Available BuAC = 1	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

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SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>In animal testing, exposure to aerosols of some reactive diluents (notably o-cresol glycidyl ether, CAS RN: 2210-79-9) has been reported to affect the adrenal gland, central nervous system, kidney, liver, ovaries, spleen, testes, thymus, and respiratory tract.</p>
Ingestion	<p>Reactive diluents exhibit a range of ingestion hazards. Small amounts swallowed incidental to normal handling operations are not likely to cause injury. However, swallowing larger amounts may cause injury.</p> <p>Male rats exposed to a single oral dose of bisphenol A diglycidyl ether (BADGE) at 750, 1000, and 2000 mg/kg/day showed a significantly increase in the number of immature and maturing sperm on the testis. There were no significant differences with respect to sperm head count, sperm motility, and sperm abnormality in the BADGE treatment groups</p> <p>At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes)</p> <p>At sufficiently high doses the material may be nephrotoxic (i.e. poisonous to the kidney).</p> <p>The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period.</p> <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Bisphenol A diglycidyl ether (BADGE) may produce contact dermatitis characterised by erythema and oedema, with weeping followed by crusting and scaling. A liquid resin with a molecular weight of 350 produced severe skin irritation in rabbits when applied daily for 4 hours over 20 days.</p> <p>Following the initial contact there may be a discrete erythematous lesion, confined to the point of contact, which may persist for 48 hours to 10 days; the erythema may give way to a papular, vesicular rash with scaling.</p> <p>In animals uncured resin produces moderate ante-mortem depression, loss of body weight and diarrhoea. Local irritation, inflammation and death resulting from respiratory system depression are recorded. Higher molecular weight resins generally produce lower toxicity.</p> <p>Skin contact with reactive diluents may cause slight to moderate irritation with local redness. Repeated or prolonged skin contact may cause burns.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury.</p>
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-</p>

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responsive.

Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of

- appropriate animal studies,
- other relevant information

Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.

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

Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity..

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

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

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

Bisphenol A diglycidyl ethers (BADGEs) produce sensitisation dermatitis characterised by a papular, vesicular eczema with considerable itching of the back of the hand, the forearm and face and neck. This lesion may persist for 10-14 days after withdrawal from exposure and recur immediately on re-exposure. This dermatitis may persist for longer periods following each exposure but is unlikely to become more intense. Lesions may develop a brownish colour and scaling occurs frequently. Lower molecular weight species produce sensitisation more readily.

In mice technical grades of bisphenol A diglycidyl ether produced epidermal tumours and a small increase in the incidence kidney tumours in males and of lymphoreticular/ haematopoietic tumours in females. Subcutaneous injection produced a small number of fibrosarcomas in rats.

BADGE is listed as an IARC Group 3 carcinogen, meaning it is "not classifiable as to its carcinogenicity to humans". Concern has been raised over this possible carcinogenicity because BADGE is used in epoxy resins in the lining of some tin cans for foodstuffs, and unreacted BADGE may end up in the contents of those cans.

For some reactive diluents, prolonged or repeated skin contact may result in absorption of potentially harmful amounts or allergic skin reactions

Exposure to some reactive diluents (notably neopentylglycol diglycidyl ether, CAS RN:17557-23-2) has caused cancer in some animal testing.

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Bisphenol F, bisphenol A, fluorine-containing bisphenol A (bisphenol AF), and other diphenylalkanes were found to be oestrogenic in a bioassay with MCF7 human breast cancer cells in culture. Bisphenol F (4,4'-dihydroxydiphenylmethane) has been reported to exhibit oestrogen agonistic properties in the uterotrophic assay. Bisphenol F (BPF) is present in the environment and as a contaminant of food. Humans may, therefore, be exposed to BP. BPF has been shown to have genotoxic and endocrine-disruptor properties in a human hepatoma cell line (HepG2), which is a model system for studies of xenobiotic toxicity. BPF was largely metabolised into the corresponding sulfate by the HepG2 cell line. BPF was metabolised into both sulfate and glucuronide by human hepatocytes, but with differences between individuals. The metabolism of BPF in both HepG2 cells and human hepatocytes suggests the existence of a detoxification pathway

Bisphenol F was orally administered at doses 0, 20, 100 and 500 mg/kg per day for at least 28 days, but no clear endocrine-mediated changes were detected, and it was concluded to have no endocrine-mediated effects in young adult rats. On the other hand, the main effect of bisphenol F was concluded to be liver toxicity based on clinical biochemical parameters and liver weight, but without histopathological changes. The no-observed-effect level for bisphenol F is concluded to be under 20 mg/kg per day since decreased body weight accompanied by decreased serum total cholesterol, glucose, and albumin values were observed in the female rats given 20 mg/kg per day or higher doses of bisphenol F.

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Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. The presence of the p-hydroxy group on the benzene rings is thought to be responsible for the oestradiol mimicry.

Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review.

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

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

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

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

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

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

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

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

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

SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A	TOXICITY	IRRITATION
	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >17.094 mg/kg ^[2] Oral(Mouse) LD50; >500 mg/kg ^[2]	Eye (rabbit): 100mg - Mild
bisphenol F diglycidyl ether copolymer	TOXICITY	IRRITATION
	dermal (rat) LD50: >400 mg/kg ^[2] Oral(Rat) LD50; >2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1]
propylene carbonate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >=2000 mg/kg ^[1]	Eye (rabbit): 60 mg - moderate

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	Oral(Rat) LD50; >5000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin (human): 100 mg/3d-I moderate
		Skin (rabbit): 500 mg moderate
		Skin: no adverse effect observed (not irritating) ^[1]
(C12-14)alkylglycidyl ether	TOXICITY	IRRITATION
	Oral(Rat) LD50; 21.818 mg/kg ^[2]	Eye (rabbit): mild [Ciba]
		Eye: adverse effect observed (irritating) ^[1]
		Skin (guinea pig): sensitiser
		Skin (human): Irritant
		Skin (human): non- sensitiser
		Skin (rabbit): moderate
		Skin : Moderate
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	TOXICITY	IRRITATION
	Oral(Rat) LD50; 23693920 mg/kg ^[2]	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	TOXICITY	IRRITATION
	Not Available	Not Available
zirconium 2-ethylhexanoate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Inhalation(Rat) LC50; >4.3 mg/l4h ^[1]	
	Oral(Rat) LD50; 2043 mg/kg ^[1]	
gamma-glycidioxypropyltrimethoxysilane	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3.71 mg/kg ^[1]	Not Available
	Inhalation(Rat) LC50; >5.3 mg/l4h ^[1]	
	Oral(Rat) LD50; >4.673 mg/kg ^[1]	

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

SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A	<p>The various members of the bisphenol family produce hormone like effects, seemingly as a result of binding to estrogen receptor-related receptors (ERRs; not to be confused with estrogen receptors)</p> <p>A suspected estrogen-related receptors (ERR) binding agent:</p> <p>Estrogen-related receptors (ERR, oestrogen-related receptors) are so named because of sequence homology with estrogen receptors but do not appear to bind estrogens or other tested steroid hormones. The ERR family have been demonstrated to control energy homeostasis, oxidative metabolism and mitochondrial biogenesis, while effecting mammalian physiology in the heart, brown adipose tissue, white adipose tissue, placenta, macrophages, and demonstrated additional roles in diabetes and cancer.</p> <p>ERRs bind enhancers throughout the genome where they exert effects on gene regulation</p> <p>Although their overall functions remain uncertain, they also share DNA-binding sites, co-regulators, and target genes with the conventional estrogen receptors ERalpha and ERbeta and may function to modulate estrogen signaling pathways.</p> <ul style="list-style-type: none"> ERR-alpha has wide tissue distribution but it is most highly expressed in tissues that preferentially use fatty acids as energy sources such as kidney, heart, brown adipose tissue, cerebellum, intestine, and skeletal muscle. ERRalpha has been detected in normal adrenal cortex tissues, in which its expression is possibly related to adrenal development, with a possible role in fetal adrenal function, in dehydroepiandrosterone (DHEA) production in adrenarche, and also in steroid production of post-adrenarche/adult life. DHEA and other adrenal androgens such as androstenedione, although relatively weak androgens, are responsible for the androgenic effects of adrenarche, such as early pubic and axillary hair growth, adult-type body odor, increased oiliness of hair and skin, and mild acne. ERR-beta is a nuclear receptor. Its function is unknown; however, a similar protein in mouse plays an essential role in placental development ERR-gamma is a nuclear receptor that behaves as a constitutive activator of transcription. There is evidence that bisphenol A functions as an endocrine disruptor by binding strongly to ERRgamma BPA as well as its nitrated and chlorinated metabolites seems to binds strongly to ERR-gamma (dissociation constant = 5.5 nM), but not to the estrogen receptor (ER). BPA binding to ERR-gamma preserves its basal constitutive activity. Different expression of ERR-gamma in different parts of the body may account for variations in bisphenol
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	A effects. For instance, ERR-gamma has been found in high concentration in the placenta, explaining reports of high bisphenol A accumulation there
BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	<p>Foetotoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity; NOEL (maternal 60 mg/kg</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
BISPHENOL F DIGLYCIDYL ETHER COPOLYMER	Data for liquid polymer, ie for molecular weights generally less than 700 CAUTION: Epoxy resin products may contain sensitising glycidyl ethers, even when these are not mentioned in the information given for the product. Limited animal studies have indicated that bisphenol A diglycidyl ethers may be potential carcinogens. [CISDOC Patty]
PROPYLENE CARBONATE	<p>for propylene carbonate:</p> <p>Numerous adequate and reliable acute toxicity tests are available on propylene carbonate. Oral and dermal tests meet OECD and EPA test guidelines. Propylene carbonate is practically nontoxic following acute exposures; the oral LD50 is >.5000 mg/kg and the dermal LD50 is >3000 mg/kg. No further testing is recommended.</p> <p>Subchronic studies (13- 14 weeks) of propylene carbonate by inhalation (aerosol) and oral (gavage) routes were conducted in rats according to current guidelines. The oral study indicated low systemic toxicity from propylene carbonate (NOAEL = 5000 mg/kg/day). In the inhalation study, no systemic toxicity was seen at concentrations up to 1000 mg/m³; however, there was periocular irritation and swelling in a few males at 500 and 1000 mg/m³. A dermal carcinogenicity study in mice did not indicate tumorigenic potential or systemic toxicity from 2 years of exposure to propylene carbonate. No further testing is recommended.</p> <p>There is a negative Ames in vitro mutagenicity assay of propylene carbonate. A single intraperitoneal injection of 1666 mg/kg propylene carbonate did not induce an increase in micronuclei when examined after 30,48 and 72 hours. The mutagenicity battery is satisfactorily filled; no further mutagenicity testing is recommended.</p> <p>Gavage administration of propylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 and 5000 mg/kg/day, including mortality (not seen in 13 week study of non-pregnant rats). The NOAEL for maternal toxicity was 1000 mg/kg/day. This indicates that pregnant rats are more susceptible to propylene carbonate than are non-pregnant rats. There were no significant differences in live litter size, average fetal weight, percentage of males, or malformed fetuses.</p> <p>No studies of the effect of propylene carbonate on reproduction are available. However, no adverse effects on testis, ovaries, or accessory sex organs were noted in rats following oral or inhalation of propylene carbonate for 13 weeks. Therefore, reproductive effects from propylene carbonate are unlikely</p>
ZIRCONIUM 2-ETHYLHEXANOATE	<p>For aliphatic fatty acids (and salts)</p> <p>Acute oral (gavage) toxicity:</p> <p>The acute oral LD50 values in rats for both were greater than >2000 mg/kg bw Clinical signs were generally associated with poor condition following administration of high doses (salivation, diarrhoea, staining, piloerection and lethargy). There were no adverse effects on body weight in any study In some studies, excess test substance and/or irritation in the gastrointestinal tract was observed at necropsy.</p> <p>Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length</p> <p>According to several OECD test regimes the animal skin irritation studies indicate that the C6-10 aliphatic acids are severely irritating or corrosive, while the C12 aliphatic acid is irritating, and the C14-22 aliphatic acids generally are not irritating or mildly irritating.</p> <p>Human skin irritation studies using more realistic exposures (30-minute, 1-hour or 24-hours) indicate that the aliphatic acids have sufficient, good or very good skin compatibility.</p> <p>Animal eye irritation studies indicate that among the aliphatic acids, the C8-12 aliphatic acids are irritating to the eye while the C14-22 aliphatic acids are not irritating.</p> <p>Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes.</p> <p>Dermal absorption:</p> <p>The in vitro penetration of C10, C12, C14, C16 and C18 fatty acids (as sodium salt solutions) through rat skin decreases with increasing chain length. At 86.73 ug C16/cm² and 91.84 ug C18/cm², about 0.23% and less than 0.1% of the C16 and C18 soap solutions is absorbed after 24 h exposure, respectively.</p> <p>Sensitisation:</p> <p>No sensitisation data were located.</p> <p>Repeat dose toxicity:</p> <p>Repeated dose oral (gavage or diet) exposure to aliphatic acids did not result in systemic toxicity with NOAELs greater than the limit dose of 1000 mg/kg bw. .</p> <p>Mutagenicity</p> <p>Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo</p> <p>Carcinogenicity</p> <p>No data were located for carcinogenicity of aliphatic fatty acids.</p> <p>Reproductive toxicity</p> <p>No effects on fertility or on reproductive organs, or developmental effects were observed in studies on aliphatic acids and the NOAELs correspond to the maximum dose tested. The weight of evidence supports the lack of reproductive and developmental toxicity potential of the aliphatic acids category.</p> <p>Given the large number of substances in this category, their closely related chemical structure, expected trends in physical chemical properties, and similarity of toxicokinetic properties, both mammalian and aquatic endpoints were filled using read-across to the closest structural analogue, and selecting the most conservative supporting substance effect level.</p>

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Structure-activity relationships are not evident for the mammalian toxicity endpoints. That is, the low mammalian toxicity of this category of substances limits the ability to discern structural effects on biological activity. Regardless, the closest structural analogue with the most conservative effect value was selected for read across. Irritation is observed for chain lengths up to a "cut-off" at or near 12 carbons).

Metabolism:

The aliphatic acids share a common degradation pathway in which they are metabolized to acetyl-CoA or other key metabolites in all living systems. Common biological pathways result in structurally similar breakdown products, and are, together with the physico-chemical properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

Differences in metabolism or biodegradability of even and odd numbered carbon chain compounds or saturated/unsaturated compounds are not expected; even- and odd-numbered carbon chain compounds, and the saturated and unsaturated compounds are naturally occurring and are expected to be metabolized and biodegraded in the same manner.

The acid and alkali salt forms of the homologous aliphatic acid are expected to have many similar physicochemical and toxicological properties when they become bioavailable; therefore, data read across is used for those instances where data are available for the acid form but not the salt, and vice versa. In the gastrointestinal tract, acids and bases are absorbed in the undissociated (non-ionised) form by simple diffusion or by facilitated diffusion. It is expected that both the acids and the salts will be present in (or converted to) the acid form in the stomach. This means that for both aliphatic acid or aliphatic acid salt, the same compounds eventually enter the small intestine, where equilibrium, as a result of increased pH, will shift towards dissociation (ionised form).

Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated.

Note that the saturation or unsaturation level is not a factor in the toxicity of these substances and is not a critical component of the read across process..

Toxicokinetics:

The turnover of the [¹⁴C] surfactants in the rat showed that there was no significant difference in the rate or route of excretion of ¹⁴C given by intraperitoneal or subcutaneous administration. The main route of excretion was as ¹⁴CO₂ in the expired air at 6 h after administration. The remaining material was incorporated in the body. Longer fatty acid chains are more readily incorporated than shorter chains. At ca. 1.55 and 1.64 mg/kg bw, 71% of the C₁₆:0 and 56% of the C₁₈:0 was incorporated and 21% and 38% was excreted as ¹⁴CO₂, respectively.

Glycidyl fatty acid esters (GEs), one of the main contaminants in processed oils, are mainly formed during the deodorisation step in the refining process of edible oils and therefore occur in almost all refined edible oils. GEs are potential carcinogens, due to the fact that they readily hydrolyze into the free form glycidol in the gastrointestinal tract, which has been found to induce tumours in various rat tissues. Therefore, significant effort has been devoted to inhibit and eliminate the formation of GEs.

GEs contain a common terminal epoxide group but exhibit different fatty acid compositions. This class of compounds has been reported in edible oils after overestimation of 3-monochloropropane-1,2-diol (3-MCPD) fatty acid esters analysed by an indirect method, 3-MCPD esters have been studied as food processing contaminants and are found in various food types and food ingredients, particularly in refined edible oils. 3-Monochloropropane-1,2-diol (3-MCPD) and 2-monochloropropane-1,3-diol (2-MCPD) are chlorinated derivatives of glycerol (1,2,3-propanetriol). 3- and 2-MCPD and their fatty acid esters are among non-volatile chloropropanols. Glycidol is associated with the formation and decomposition of 3- and 2-MCPD. It forms monoesters with fatty acids (GE) during the refining of vegetable oils. Chloropropanols are formed in HVP during the hydrochloric acid-mediated hydrolysis step of the manufacturing process. In food production, chloropropanols form from the reaction of endogenous or added chloride with glycerol or acylglycerol.

Although harmful effects on humans and animals have not been demonstrated, the corresponding hydrolysates, 3-MCPD and glycidol, have been identified as rodent genotoxic carcinogens, ultimately resulting in the formation of kidney tumours (3-MCPD) and tumours at other tissue sites (glycidol). Therefore, 3-MCPD and glycidol have been categorised as "possible human carcinogens" (group 2B) and "probably carcinogenic to humans" (group 2A), respectively, by the International Agency for Research on Cancer (IARC).

Diacylglyceride (DAG) based oils produced by one company were banned from the global market due to "high levels" of GEs.

Several reports have also suggested that a bidirectional transformation process may occur not only between glycidol and 3-MCPD but also their esterified forms in the presence of chloride ions. The transformation rate of glycidol to 3-MCPD was higher than that of 3-MCPD to glycidol under acidic conditions in the presence of chloride ion.

Precursors of GEs in refined oils have been identified as partial acylglycerols, that is, DAGs and monoacylglycerides (MAGs); however, whether they also originate from triacylglycerides (TAGs) is still a topic of controversial debates. Several authors noted that pure TAGs were stable during heat treatment (such as 235 deg C) for 3 h and were therefore not involved in the formation of GEs. However, experimental results have shown that small amounts of GEs are present in a heat-treated oil model consisting of almost 100% TAGs. The formation of GEs from TAGs can be attributed to the pyrolysis of TAGs to DAGs and MAGs. In contrast, 3-MCPD esters in refined oils can be obtained from TAG. Presently, the mechanism for the formation of GE intermediates and the relationship between GEs and 3-MCPD esters are still unknown.

Fatty acid salts are of low acute toxicity. Their skin and eye irritation potential is chain length dependent and decreases with increasing chain length - they are poorly absorbed through the skin nor are they skin sensitizers. The available repeated dose toxicity data demonstrate the low toxicity of the fatty acids and their salts. Also, they are not considered to be mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. Accidental ingestion of fatty acid salt containing detergent products is not expected to result in any

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	<p>significant adverse health effects. This assessment is based on toxicological data demonstrating the low acute oral toxicity of fatty acid salts and the fact that not a single fatality has been reported in the UK following accidental ingestion of detergents containing fatty acid salts. Also in a report published by the German Federal Institute for Health Protection of Consumers and Veterinary Medicine, detergent products were not mentioned as dangerous products with a high incidence of poisoning. The estimated total human exposure to fatty acid salts, from the different exposure scenarios for the handling and use of detergent products containing fatty acid salts, showed a margin of exposure (MOE) of 258,620. This extremely large MOE is large enough to be reassuring with regard to the relatively small variability of the hazard data on which it is based. Also, in the UK, the recommended dietary fatty acid intake by the Department of Health is about 100 g of fatty acids per day or 1.7 g (1700 mg) of fatty acids per kilogram body weight per day. This exposure is several orders of magnitude above that resulting from exposure to fatty acid salts in household cleaning products. Based on the available data, the use of fatty acid salts in household detergent and cleaning products does not raise any safety concerns with regard to consumer</p>
<p>GAMMA-GLYCIDIOXYPROPYLTRIMETHOXYISILANE</p>	<p>For alkoxysilanes:</p> <p>Low molecular weight alkoxysilanes (including alkyl orthosilicates) are a known concern for lung toxicity, due to inhalation of vapours or aerosols causing irreversible lung damage at low doses.</p> <p>Alkoxysilane groups that rapidly hydrolyse when in contact with water, result in metabolites that may only cause mild skin irritation. Although there appears to be signs of irritation under different test conditions, based on the available information, the alkoxysilanes cannot be readily classified as a skin irritant.</p> <p>The trimethoxysilane group of chemicals have previously been associated with occupational eye irritation in exposed workers who experienced severe inflammation of the cornea. Based on the collective information, these substances are likely to be severe irritants to the eyes.</p> <p>Methoxysilanes are generally reported to possess higher reactivity and toxicity compared to ethoxysilanes; some methoxysilanes appear to be carcinogenic. In the US, alkoxysilanes with alkoxy groups greater than C2 are classified as moderate concern.</p> <p>Based on available information on methoxysilanes, the possibility that this family causes skin sensitisation cannot be ruled out. Amine-functional methoxysilanes have previously been implicated as a cause of occupational contact dermatitis, often as a result of repeated skin exposure with workers involved in the manufacture or use of the resins containing the chemical during fibreglass production.</p> <p>For gamma-glycidopropyltrimethoxysilane (GPTMS)</p> <p>GPTMS is subject to rapid hydrolysis, and the observed toxicity is expected to be due primarily to methanol and silanetriols. GPTMS has been tested for acute toxicity by the oral, dermal, and inhalation routes of exposure. Reported acute oral LD50s in rats range from 7010 to 16900 mg/kg bw and > 5 ml/kg bw to 22.6 ml/kg bw. The dermal LD50s are 6800 mg/kg bw and 4.0 ml/kg bw. The 4-hour inhalation LC50 was greater than 2.7 mg/L in one study and greater than 5.3 mg/L in another study. GPTMS is mildly irritating to the skin and eyes and is not a known skin sensitiser in humans or in animals.</p> <p>Following inhalation exposures of rats to target aerosol concentrations of 0, 75, 225 and 750 mg/m³ (actual concentrations were 0, 77, 226, 707 mg/m³ (males) and 0, 73, 226, 734 mg/m³ (females)), GPTMS in 9 repeated exposures administered over two weeks, 6 animals in the high dose group died or were sacrificed from three to five days after initiation of the study. These animals had signs of inanition but no acute tissue toxicity. At both the mid and high doses, rats exhibited some clinical signs including a dose-related decrease in body weight. Under the conditions of this study, the No Observed Adverse Effect Concentration is 225 mg/m³. Repeated exposure of rats by gavage to GPTMS doses of 40, 400 and 1000 mg/kg bw/day for 5 days/week for 4 weeks resulted in no test substance-related organ weights effects or gross or microscopic pathological changes. Under the conditions of this study, the NOAEL for the test substance was found to be 1000 mg/kg bw/day.</p> <p>Genotoxicity: GPTMS did not induce chromosomal damage in mouse bone marrow cells by gavage at doses of 500, 1670 and 5000 mg/kg bw/day, or when administered by intraperitoneal (i.p.) injection at 1600 mg/kg bw/day. However, chromosomal damage was induced in mouse bone marrow cells when administered by i.p. in water at doses of 500, 1000 and 2000 mg/kg bw/day. GPTMS induced gene mutations in bacteria. GPTMS induced gene mutations in mouse lymphoma L1578Y TK cells but did not induce forward mutations in CHO cells. GPTMS induced SCE in vitro. There are no in vivo gene mutation data.</p> <p>Carcinogenicity: GPTMS was not considered tumourigenic when applied to the clipped skin of mice (25 µl dose of 25% GPTMS in acetone) three times per week for approximately 78 weeks. Note that there was only one dose level, and this dose was relatively low.</p> <p>Reproductive toxicity: In a one-generation reproduction toxicity study in rats, no reproductive effects were observed at any of the doses tested (250, 500, or 1000 mg/kg bw/day). At 1000 mg/kg bw/day, treatment with GPTMS resulted in the following signs in parental animals: discomfort after dosing (noted for females from early/mid gestation onwards), decreased body weight gain (males), increased mean relative liver and kidney weights (noted for males and females), and histopathological effects on livers and kidneys (males). Based on these data, a NOAEL for parental animals was established at 500 mg/kg bw/day. A NOAEL for reproductive effects was established at 1000 mg/kg bw/day.</p> <p>Developmental toxicity: Three developmental studies have been conducted using GPTMS. In a rabbit study, the maternal NOAEL was 200 mg/kg bw/day and the developmental NOAEL was 400 mg/kg bw/day (the highest dose tested). In a rat study, the NOAELs for both maternal and developmental toxicity were also at the highest dose tested (1000 mg/kg bw/day). In another rat study, developmental effects were observed at the maternally toxic dose of 3000 mg/kg bw/day (again, the highest dose tested).</p>
<p>SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID &</p>	<p>The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed</p>

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<p>BISPHENOL F DIGLYCIDYL ETHER COPOLYMER & (C12-14)ALKYLGLYCIDYL ETHER & BIS(1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL)SEBACATE & METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE</p>	<p>type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
<p>SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID</p>	<p>In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg).</p> <p>Reproductive and Developmental Toxicity: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg.</p> <p>Carcinogenicity: IARC concluded that "there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals." Its overall evaluation was "Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3).</p> <p>In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997).</p> <p>Genotoxicity: In <i>S. typhimurium</i> strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg).</p> <p>Immunotoxicity: Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs</p> <p>-</p> <p>Consumer exposure to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/ kg/body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weight/day (male rats) from the 2-year carcinogenicity study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELS from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs.</p>
<p>SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & BISPHENOL F DIGLYCIDYL ETHER COPOLYMER</p>	<p>The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics.</p> <p>Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities.</p> <p>Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.</p> <p>In vitro cell models were used to evaluate the ability of 22 bisphenols (BPs) to induce or inhibit estrogenic and androgenic activity. BPA, Bisphenol AF (BPAF), bisphenol Z (BPZ), bisphenol C (BPC), tetramethyl bisphenol A (TMBPA), bisphenol S (BPS), bisphenol E (BPE), 4,4-bisphenol F (4,4-BPF), bisphenol AP (BPAP), bisphenol B (BPB), tetrachlorobisphenol A (TCBPA), and benzylparaben (PHBB) induced estrogen receptor (ER)alpha and/or</p>

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	ERbeta-mediated activity. With the exception of BPS, TCBPA, and PHBB, these same BPs were also androgen receptor (AR) antagonists. Only 3 BPs were found to be ER antagonists. Bisphenol P (BPP) selectively inhibited ERbeta-mediated activity and 4-(4-phenylmethoxyphenyl)sulfonylphenol (BPS-MPE) and 2,4-bisphenol S (2,4-BPS) selectively inhibited ERalpha-mediated activity. None of the BPs induced AR-mediated activity.
SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A & BISPHENOL F DIGLYCIDYL ETHER COPOLYMER & (C12-14)ALKYLGLYCIDYL ETHER & GAMMA-GLYCIDOXYPROPYLTRIMETHOXYSILANE	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.
BISPHENOL F DIGLYCIDYL ETHER COPOLYMER & METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE & ZIRCONIUM 2-ETHYLHEXANOATE	No significant acute toxicological data identified in literature search.
BISPHENOL F DIGLYCIDYL ETHER COPOLYMER & PROPYLENE CARBONATE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
(C12-14)ALKYLGLYCIDYL ETHER & GAMMA-GLYCIDOXYPROPYLTRIMETHOXYSILANE	for 1,2-butylene oxide (ethyloxirane): Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m ³ 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 ³) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals. Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic

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

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

SECTION 12 Ecological information

Toxicity

SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	48	Crustacea	~2mg/l	2
	EC50	48	Crustacea	~2mg/l	2
bisphenol F diglycidyl ether copolymer	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
propylene carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96	Fish	>1000mg/l	2
	EC50	48	Crustacea	>1000mg/l	1

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	EC50(ECx)	72	Algae or other aquatic plants	>900mg/l	1
	EC50	72	Algae or other aquatic plants	>900mg/l	1
(C12-14)alkylglycidyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48	Crustacea	6.07mg/l	2
	EC50(ECx)	48	Crustacea	6.07mg/l	2
	LC50	96	Fish	>5000mg/l	2
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC0(ECx)	24	Crustacea	<10mg/l	1
	LC50	96	Fish	0.34mg/l	1
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
zirconium 2-ethylhexanoate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48	Crustacea	>0.17mg/l	2
	LC50	96	Fish	>100mg/l	2
	EC50(ECx)	48	Crustacea	>0.17mg/l	2
	EC50	72	Algae or other aquatic plants	49.3mg/l	2
gamma-glycidoxypropyltrimethoxysilane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48	Crustacea	473mg/l	2
	LC50	96	Fish	4.9mg/l	2
	NOEC(ECx)	96	Fish	1.5mg/l	2
	EC50	72	Algae or other aquatic plants	>420mg/l	2
	EC50	96	Algae or other aquatic plants	250mg/l	2

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

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

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

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

For bisphenol A and related bisphenols:

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.

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

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

Reactive diluents generally have a low to moderate potential for bioconcentration (tendency to accumulate in the food chain) and a high to very high potential for mobility in soil. Small amounts that escape to the atmosphere will photodegrade.

They would not be expected to persist in the environment.

Most reactive diluents should be considered slightly to moderately toxic to aquatic organisms on an acute basis while some might also be considered harmful to the environment.

Environmental toxicity is a function of the n-octanol/water partition coefficient (log Pow, log Kow). Compounds with log Pow >5 act as neutral organics, but at a lower log Pow, the toxicity of epoxide-containing polymers is greater than that predicted for simple narcotics.

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

for 1,2-butylene oxide (ethyloxirane):

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

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

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

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

Ecotoxicity:

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

* *Persistence and Bioaccumulation Regulations* (Canada 2000).

Reactive diluents which are only slightly soluble in water and do not evaporate quickly are expected to sink to the bottom or float to the top, depending on the density, where they would be expected to biodegrade slowly.

for propylene carbonate:

Environmental fate:

Data on the transport of propylene carbonate between environmental compartments has been estimated using EPIWIN; propylene carbonate will partition mostly to water and soil; 1% to air; 46% to water; 53% to soil.

Does not absorb UV and therefore is unlikely to be photodegradable.

Biodegradation: OECD Test Guideline 301B (Modified Sturm Test)

Propylene carbonate was readily biodegradable by activated domestic sludge under aerobic conditions. More than 85% degraded after 29 days

Ecotoxicity

Invertebrate EC50: >1000 mg/l

Aquatic plant EC50: >=929 mg/l

Acute toxicity studies on fish and daphnia on a propylene carbonate analog, butylene carbonate, were conducted according to OECD Guidelines, following GLP guidelines. Butylene carbonate (CAS # 4437-85-8) is considered an acceptable surrogate for propylene carbonate because of similar physical-chemical properties. In rainbow trout the LC50 for butylene carbonate was 480 mg/l and in *Daphnia* the EC50 was >1000 mg/l. Based on similarity to butylene carbonate, propylene carbonate is not expected to be significantly more toxic to aquatic organisms. The low toxicity of butylene carbonate provides adequate information to conclude that propylene carbonate is not likely to be more than slightly toxic to aquatic organisms.

Toxicity to Aquatic plants (e.g., Algae): OECD Test Guideline 201 Propylene carbonate did not affect cell density, area under the growth curve, or growth rate of unicellular green alga, *Selenastrum capricornutum*, after 72 and 96 hours of exposure at levels up to 929 mg/l, measured concentration.

Biodegradation: OECD Test Guideline 301B (Modified Sturm Test)

Propylene carbonate was readily biodegradable by activated domestic sludge under aerobic conditions. More than 85% degraded after 29 days.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
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Continued...

SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A

Ingredient	Persistence: Water/Soil	Persistence: Air
bisphenol A/ diglycidyl ether resin, liquid	HIGH	HIGH
propylene carbonate	HIGH	HIGH
gamma-glycidoxypropyltrimethoxysilane	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
bisphenol A/ diglycidyl ether resin, liquid	LOW (LogKOW = 2.6835)
propylene carbonate	LOW (LogKOW = -0.41)
gamma-glycidoxypropyltrimethoxysilane	LOW (LogKOW = -0.9152)

Mobility in soil

Ingredient	Mobility
bisphenol A/ diglycidyl ether resin, liquid	LOW (KOC = 51.43)
propylene carbonate	LOW (KOC = 14.85)
gamma-glycidoxypropyltrimethoxysilane	LOW (KOC = 90.22)

SECTION 13 Disposal considerations



Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> Reduction Reuse Recycling Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> DO NOT allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. <p>Removal of bisphenol A (BPA) from aqueous solutions was accomplished by adsorption of enzymatically generated quinone derivatives on chitosan beads. The use of chitosan in the form of beads was found to be more effective because heterogeneous removal of BPA with chitosan beads was much faster than homogeneous removal of BPA with chitosan solutions, and the removal efficiency was enhanced by increasing the amount of chitosan beads dispersed in the BPA solutions and BPA was completely removed by quinone adsorption in the presence of chitosan beads more than 0.10 cm³/cm³. In addition, a variety of bisphenol derivatives were completely or effectively removed by the procedure constructed in this study, although the enzyme dose or the amount of chitosan beads was further increased as necessary for some of the bisphenol derivatives used.</p> <p>M. Suzuki, and E. Musashi J Appl Polym Sci, 118(2):721 - 732; October 2010</p> <ul style="list-style-type: none"> Recycle wherever possible or consult manufacturer for recycling options. Consult State Land Waste Authority for disposal. Bury or incinerate residue at an approved site. Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A

Labels Required

	
Marine Pollutant	
HAZCHEM	*3Z

Land transport (ADG)

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

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082

are not subject to this Code when transported by road or rail in;

(a) packagings;

(b) IBCs; or

(c) any other receptacle not exceeding 500 kg(L).

- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

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

Sea transport (IMDG-Code / GGVSee)

UN number	3082	
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid)	
Transport hazard class(es)	IMDG Class	9
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Marine Pollutant	

Continued...

SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A

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

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

Not Applicable

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

Product name	Group
bisphenol A/ diglycidyl ether resin, liquid	Not Available
bisphenol F diglycidyl ether copolymer	Not Available
propylene carbonate	Not Available
(C12-14)alkylglycidyl ether	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available
zirconium 2-ethylhexanoate	Not Available
gamma-glycidoxypropyltrimethoxysilane	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
bisphenol A/ diglycidyl ether resin, liquid	Not Available
bisphenol F diglycidyl ether copolymer	Not Available
propylene carbonate	Not Available
(C12-14)alkylglycidyl ether	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available
zirconium 2-ethylhexanoate	Not Available
gamma-glycidoxypropyltrimethoxysilane	Not Available

SECTION 15 Regulatory information

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

bisphenol A/ diglycidyl ether resin, liquid is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

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

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

bisphenol F diglycidyl ether copolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

propylene carbonate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

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

Continued...

SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

zirconium 2-ethylhexanoate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

gamma-glycidoxypolytrimethoxysilane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (bisphenol A/ diglycidyl ether resin, liquid; bisphenol F diglycidyl ether copolymer; propylene carbonate; (C12-14)alkylglycidyl ether; bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate; zirconium 2-ethylhexanoate; gamma-glycidoxypolytrimethoxysilane)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (bisphenol F diglycidyl ether copolymer; (C12-14)alkylglycidyl ether)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No ((C12-14)alkylglycidyl ether; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate; zirconium 2-ethylhexanoate; gamma-glycidoxypolytrimethoxysilane)
Vietnam - NCI	Yes
Russia - FBEPH	No (methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate)
Legend:	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)</p>

SECTION 16 Other information

Revision Date	22/04/2021
Initial Date	10/08/2015

SDS Version Summary

Version	Date of Update	Sections Updated
2.13.1.1	22/04/2021	Physical Properties, Name

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.

Continued...

SV100 Epoxy Resin Part A & SV100 High Build Epoxy Resin Part A**Definitions and abbreviations**

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

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