

SV55 Fastdry Aqueous Epoxy Part A

On-Crete Australia Pty Ltd

Version No: 0.6

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: **17/07/2018**Print Date: **17/01/2019**L.GHS.AUS.EN

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

Product Identifier

Product name	SV55 Fastdry Aqueous Epoxy Part A	
Synonyms	Not Available	
Other means of identification	Not Available	

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

Relevant identified uses Part A of a fastdry, long potlife, waterbourne epoxy coating system for cementitious surfaces

Details of the supplier of the safety data sheet

Registered company name	On-Crete Australia Pty Ltd
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Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	+61 406 948 465
Other emergency telephone numbers	+61 406 102 829

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

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

Poisons Schedule	Not Applicable
Classification ^[1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2

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Legend:

1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from Regulation (EU) No 1272/2008 -

Label elements

Hazard pictogram(s)





SIGNAL WORD

WARNING

Hazard statement(s)

H315	Causes skin irritation.	
H319	Causes serious eye irritation.	
H317	May cause an allergic skin reaction.	
H411	Toxic to aquatic life with long lasting effects.	

Precautionary statement(s) Prevention

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

Precautionary statement(s) Response

ecautionary statement(s) Response		
P362	Take off contaminated clothing and wash before reuse.	
P302+P352	IF ON SKIN: Wash with plenty of soap and water.	
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.	
P391	Collect spillage.	

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

P501

Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
25085-99-8	30-60	bisphenol A/ diglycidyl ether resin, liquid
7732-18-5	30-60	<u>water</u>
55965-84-9	<1	isothiazolinones, mixed
7631-99-4	<1	sodium nitrate
5131-66-8	<10	propylene glycol monobutyl ether - alpha isomer
41556-26-7	<1	bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate
82919-37-7	<1	methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate
112-34-5	<1	diethylene glycol monobutyl ether

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SECTION 4 FIRST AID MEASURES

Description of first aid measures		
Eye Contact	If this product comes in contact with the eyes: • Wash out immediately with fresh running water. • Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. • Seek medical attention without delay; if pain persists or recurs seek medical attention. • Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.	
Skin Contact	If skin contact occurs: ► Immediately remove all contaminated clothing, including footwear. ► Flush skin and hair with running water (and soap if available). ► Seek medical attention in event of irritation.	
Inhalation Inhalation		
Ingestion	 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

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.

Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.

In such an event consider:

- ▶ foam.
- dry chemical powder.
- ▶ carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.		
Advice for firefighters			
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves in the event of a fire. Prevent, by any means available, spillage from entering drains or water courses. Use fire fighting procedures suitable for surrounding area. 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. Equipment should be thoroughly decontaminated after use. 		
Fire/Explosion Hazard	 ➤ Equipment should be thoroughly decontaminated after use. ➤ The material is not readily combustible under normal conditions. ➤ However, it will break down under fire conditions and the organic component may burn. ➤ Not considered to be a significant fire risk. ➤ Heat may cause expansion or decomposition with violent rupture of containers. ➤ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ➤ May emit acrid smoke. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO2) other pyrolysis products typical of burning organic material. May emit corrosive fumes. 		
HAZCHEM	Not Applicable		

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

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Environmental precautions

See section 12

Methods and material for containment and cleaning up

In the event of a spill of a reactive diluent, the focus is on containing the spill to prevent contamination of soil and surface or ground water. If irritating vapors are present, an approved air-purifying respirator with organic vapor canister is recommended for cleaning up spills and leaks. For small spills, reactive diluents should be absorbed with sand. Environmental hazard - contain spillage. **Minor Spills** 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. Environmental hazard - contain spillage. Industrial spills or releases of reactive diluents are infrequent and generally contained. If a large spill does occur, the material should be captured, collected, and reprocessed or disposed of according to applicable governmental requirements. An approved air-purifying respirator with organic-vapor canister is recommended for emergency work. Moderate hazard. Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. **Major Spills** ▶ Prevent, by any means available, spillage from entering drains or water course. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. ► Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. • After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.

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

SECTION 7 HANDLING AND STORAGE

Other information

Precautions for safe handling · Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. • Prevent concentration in hollows and sumps. DO NOT enter confined spaces until atmosphere has been checked. ► **DO NOT** allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, **DO NOT** eat, drink or smoke. Safe handling ▶ Keep containers securely sealed when not in use. Avoid physical damage to containers. Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. 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 are

▶ If contamination of drains or waterways occurs, advise emergency services.

ditions for acts storage including any incompatibilities

maintained.

Conditions for safe stora	age, including any incompatibilities
Suitable container	 Polyethylene or polypropylene container. Packing as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Glycidyl ethers: • may form unstable peroxides on storage in air ,light, sunlight, UV light or other ionising radiation, trace metals - inhibitor should be maintained at adequate levels
	Continued

▶ DO NOT allow clothing wet with material to stay in contact with skin

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- ▶ may polymerise in contact with heat, organic and inorganic free radical producing initiators
- may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines
- react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide
- attack some forms of plastics, coatings, and rubber

Reactive diluents are stable under recommended storage conditions, but can decompose at elevated temperatures. In some cases, decomposition can cause pressure build-up in closed systems.

- ▶ Avoid cross contamination between the two liquid parts of product (kit).
- If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur.
- ▶ This excess heat may generate toxic vapour
- ▶ Avoid reaction with amines, mercaptans, strong acids and oxidising agents

None known















X — Must not be stored together

May be stored together with specific preventions

May be stored together

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
bisphenol A/ diglycidyl ether resin, liquid	Epoxy resin includes EPON 1001, 1007, 820, ERL-2795	90 mg/m3	990 mg/m3	5,900 mg/m3
sodium nitrate	Sodium nitrate	4.1 mg/m3	45 mg/m3	270 mg/m3
diethylene glycol monobutyl ether	Butoxyethoxy)ethanol, 2-(2-; (Diethylene glycol monobutyl ether)	30 ppm	33 ppm	200 ppm

Ingredient	Original IDLH	Revised IDLH
bisphenol A/ diglycidyl ether resin, liquid	Not Available	Not Available
water	Not Available	Not Available
isothiazolinones, mixed	Not Available	Not Available
sodium nitrate	Not Available	Not Available
propylene glycol monobutyl ether - alpha isomer	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
diethylene glycol monobutyl ether	Not Available	Not Available

MATERIAL DATA

For epichlorohydrin

Odour Threshold Value: 0.08 ppm

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

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

Odour Safety Factor (OSF)

OSF=0.54 (EPICHLOROHYDRIN)

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For diethylene glycol monobutyl ether:

CEL TWA: 15.5 ppm, 100 mg/m3 (CEL = Chemwatch Exposure Limit)

In studies involving the inhalation toxicity of diethylene glycol monobutyl ether, exposure for 6 hours daily at 100 mg/m3 had no effect. This concentration is in the range of the saturated vapour concentration.

Local damage was produced following inhalation of concentrations higher than the saturated vapour concentrations, that is, during inhalation of the aerosol (350 mg/m3). Since the only potential effects of inhalation are restricted to local discomfort (in the aerosol concentration range) the substance is classified in category I for the limitation of exposure peaks.

Teratogenicity studies have not revealed prenatal toxic effects at high oral doses and this ether is classified in pregnancy risk group C.

Exposure 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:

contaminant in use.

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

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.

Appropriate engineering

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











- ► 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]

Eye and face protection

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Skin protection

See Hand protection below

NOTE:

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

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

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

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

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

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

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

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

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

Hands/feet protection

See Other protection below

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Other protection

- Overalls.
- ▶ P.V.C. apron.
- ▶ Barrier cream.
- ▶ Skin cleansing cream.
- ► Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the *computer-generated* selection:

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Material	СРІ
BUTYL	Α
NEOPRENE	Α
VITON	A
NATURAL RUBBER	С
PVA	С

^{*} CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	White		
Physical state	Liquid	Relative density (Water = 1)	1.03
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	100	Molecular weight (g/mol)	Not Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	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	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

	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
Inhaled	that suitable control measures be used in an occupational setting. 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, an respiratory tract.
Ingestion	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 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. 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.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic leve there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Bisphenol A diglycidyl ether (BADGE) may produce contact dermatitis characterised by erythema and oedema, with weeping followed by crusting and scaling. A liquid resin with a molecular weight of 350 produced severe skin irritation in rabbits when applied daily for 4 hours over 20 days. Following the initial contact there may be a discrete erythematous lesion, confined to the point of contact, which may persist for 48 hours to 10 days; the erythema may give way to a papular, vesicular rash with scaling. In animals uncured resin produces moderate ante-mortem depression, loss of body weight and diarrhoea. Local irritation, inflammation and death resulting from respiratory system depression are recorded. Higher molecular weight resins generally produce lower toxicity. Skin contact with reactive diluents may cause slight to moderate irritation with local redness. Repeated or prolonged skin contact may cause burns. Open cuts,
Еуе	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury.
Chronic	Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.

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> 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

> 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.

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

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.

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

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

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

SV55 Fastdry Aqueous	TOXICITY	IRRITAT	TON				
Epoxy Part A	Not Available	ailable					
	TOXICITY		IRRITATION				
bisphenol A/ diglycidyl ether resin, liquid	dermal (rat) LD50: >1200 mg/kg ^[2]	Eye (rabbit): 100	mg - Mild				
	Oral (rat) LD50: >1000 mg/kg ^[2]						
water	TOXICITY		II	RRITATION			
water	Oral (rat) LD50: >90000 mg/kg ^[2]	<u> </u>	Not Available				
	TOXICITY		IRRITATION				
isothiazolinones, mixed	dermal (rat) LD50: >1008 mg/kg ^[1]		Not Available				
	Oral (rat) LD50: 53 mg/kg ^[2]						
	TOXICITY			IRRITATION			
sodium nitrate	dermal (rat) LD50: >5000 mg/kg ^[1]		Not Available				
	Oral (rat) LD50: 1267 mg/kg ^[2]						
	TOXICITY		IRRITATION				
propylene glycol	dermal (rat) LD50: >2000 mg/kg ^[1]		Eye (rabbit): 15 m	ng SEVERE			
monobutyl ether - alpha isomer	Inhalation (rat) LC50: >1997.718 mg/l/8hE ^[2]		Skin (rabbit0: 500	mg OPEN - mild			
	Oral (rat) LD50: >2000 mg/kg ^[1]						

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bis(1,2,2,6,6-pentamethyl- 4-piperidyl)sebacate	TOXICITY Oral (rat) LD50: 3100 mg/kg ^[2]				
methyl 1,2,2,6,6- pentamethyl-4-piperidyl sebacate	TOXICITY Not Available	IRRITATION Not Available			
diethylene glycol monobutyl ether	TOXICITY Dermal (rabbit) LD50: >2000 mg/kg ^[2] Oral (rat) LD50: =4500 mg/kg ^[2]	IRRITATION Eye (rabbit): 20 n Eye (rabbit): 5 m			
Legend:	Value obtained from Europe ECHA Registered Sub- Unless otherwise specified data extracted from RTE	_			

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

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

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

Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review.

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

likely to report reduced sexual function within one year of beginning employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties.

Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/ litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital

masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the

70's. They also suggested that "it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades"

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

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

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

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

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

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

Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative. for 1,2-butylene oxide (ethyloxirane):

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

BISPHENOL A/ DIGLYCIDYL ETHER **RESIN, LIQUID**

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.

Foetoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity; NOEL (maternal 60 ma/ka

ISOTHIAZOLINONES, MIXED

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating

None are skin sensitisers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB -13 wk) and 450 mg/kg-d (DPnB - 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk

PROPYLENE GLYCOL MONOBUTYL ETHER -**ALPHA ISOMER**

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study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs,

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

For diethylene glycol monoalkyl ethers and their acetates:

have been reported. Commercially available PGEs showed no teratogenicity.

This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates.

Acute toxicity: There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals and/or humans were negative.

Repeat dose toxicity: Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than continuous exposure regimens.

Mutagenicity: DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in *S. typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in E. coli WP2uvrA, with and without metabolic activation. *In vitro* cytogenicity and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus or cytogenicity tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic.

Reproductive and developmental toxicity: Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGPE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA. Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m3) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during destation; however a transient decrease in body weight was observed, which reversed by Day 21 In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus

DIETHYLENE GLYCOL MONOBUTYL ETHER

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SV55 Fastdry Aqueous
Epoxy Part A &
BISPHENOL A/
DIGLYCIDYL ETHER
RESIN, LIQUID &
ISOTHIAZOLINONES,
MIXED & BIS(1,2,2,6,6PENTAMETHYL4-PIPERIDYL)SEBACATE &
METHYL 1,2,2,6,6PENTAMETHYL4-PIPERIDYL SEBACATE

The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg).

Reproductive and Developmental Toxicity: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg.

Carcinogenicity: IARC concluded that "there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals." Its overall evaluation was "Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3).

In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997).

Genotoxicity: In S. typhimurium strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg).

Immunotoxicity: Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs

SV55 Fastdry Aqueous
Epoxy Part A &
BISPHENOL A/
DIGLYCIDYL ETHER
RESIN, LIQUID

Consumer exposure to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/ kg/body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weigh/day (male rats) from the 2-year carcinogenicity study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs.

The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics

Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities.

Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.

WATER & ISOTHIAZOLINONES, MIXED & METHYL

No significant acute toxicological data identified in literature search.

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1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE

ISOTHIAZOLINONES, **MIXED & SODIUM NITRATE**

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

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

🗶 – Data either not available or does not fill the criteria for classification

✓ – Data available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

SV55 Fastdry Aqueous	ENDPOINT		TEST DURATION (HR)		SPECIES	VALU	E	so	URCE
Epoxy Part A	Not Available		Not Available	t Available No		ot Available Not Available		le Not Available	
bisphenol A/ diglycidyl	ENDPOINT	F	TEST DURATION (HR)		SPECIES		VALUE		SOURCE
ether resin, liquid	EC50		48		Crustacea		ca.2mg/L		2
	ENDPOINT	TES	T DURATION (HR)	SPECIE	5		VALUE		SOURCE
water	LC50	96		Fish			897.520	mg/L	3
	EC50	96		Algae	or other aquatic plan	ts	8768.87	4mg/L	3
sothiazolinones, mixed	ENDPOINT	TES	T DURATION (HR)	SPECIES			VALUE	/ALUE SOUI	
	LC50	96	96 Fish		Fish		0.129m	0.129mg/L	
	EC50	48		Crusta	Crustacea		0.007mg/L		2
	EC50	72 Alga		Algae	Algae or other aquatic plants		0.0063	0.0063mg/L	
	NOEC	48	48 Algae or other aquatic plants				0.0004	0.00049mg/L 2	
	ENDPOINT	TES	T DURATION (HR)	SPECIE			VALUE		SOURCE
	LC50	96 F		Fish	Fish		1-559mg/L		2
sodium nitrate	EC50	48		Crustacea			3-581mg/L		2
	EC50	96		Algae or other aquatic plants			1181.887mg/L		3
	NOEC	288	0	Fish			1.6mg/L	1.6mg/L	
	ENDPOINT	TES	ST DURATION (HR)	SPECIES			VALUE		SOURCE
propylene glycol	LC50	96		Fish				0mg/L	2
nonobutyl ether - alpha	EC50	48		Crus	rustacea		>1-	mg/L	2
isomer	EC50	96		Alga	e or other aquatic pl	ants	>1-	mg/L	2
	NOEC	96		Fish 180mg)mg/L	2	

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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 propylene glycol ethers:

Environmental fate:

Most are liquids at room temperature and all are water-soluble.

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM)

Environmental fate: Log octanol-water partition coefficients (log Kow's) range from 0.309 for TPM to 1.523 for DPnB. Calculated BCFs range from 1.47 for DPnB to 3.16 for DPMA and TPM, indicating low bioaccumulation. Henry's Law Constants, which indicate propensity to partition from water to air, are low for all category members, ranging from 5.7 x 10-9 atm-m3/mole for TPM to 2.7 x10-9 atm-m3/mole for PnB. Fugacity modeling indicates that most propylene glycol ethers are likely to partition roughly equally into the soil and water compartments in the environment with small to negligible amounts remaining in other environmental compartments (air, sediment, and aquatic biota). Propylene glycol ethers are unlikely to persist in the environment. Once in air, the half-life of the category members due to direct reactions with photochemically generated hydroxyl radicals, range from 2.0 hours for TPM to 4.6 hours for PnB. In water, most members of this family are "readily biodegradable" under aerobic conditions. (DPMA degraded within 28 days (and within the specified 10-day window) but only using pre-adapted or "acclimated" inoculum.). In soil, biodegradation is rapid for PM and PMA.

Ecotoxicity:

Acute aquatic toxicity testing indicates low toxicity for both ethers and acetates. For ethers, effect concentrations are > 500 mg/L. For acetates, effect concentrations are > 151 mg/L.

For bisphenol A and related bisphenols:

Environmental fate:

Biodegradability (28 d) 89% - Easily biodegradable

Bioconcentration factor (BCF) 7.8 mg/l

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

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

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

Ecotoxicity:

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

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

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

Freshwater algae (96 h): 2.73 mg/l Marine water algae (96 h): 1.1 mg/l

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

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

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

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

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

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

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

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.

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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:

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

* Persistence and Bioaccumulation Regulations (Canada 2000).

For glycol ethers:

Environmental fate:

Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity:

Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.

Glycols exert a high oxygen demand for decomposition and once released to the environments cause the death of aquatic organisms if dissolved oxygen is depleted.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
bisphenol A/ diglycidyl ether resin, liquid	HIGH	HIGH
water	LOW	LOW
sodium nitrate	LOW	LOW
propylene glycol monobutyl ether - alpha isomer	LOW	LOW
diethylene glycol monobutyl ether	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
bisphenol A/ diglycidyl ether resin, liquid	LOW (LogKOW = 2.6835)
water	LOW (LogKOW = -1.38)
sodium nitrate	LOW (LogKOW = 0.209)
propylene glycol monobutyl ether - alpha isomer	LOW (LogKOW = 0.9842)
diethylene glycol monobutyl ether	LOW (BCF = 0.46)

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Ingredient	Mobility
bisphenol A/ diglycidyl ether resin, liquid	LOW (KOC = 51.43)
water	LOW (KOC = 14.3)
sodium nitrate	LOW (KOC = 14.3)
propylene glycol monobutyl ether - alpha isomer	HIGH (KOC = 1.289)
diethylene glycol monobutyl ether	LOW (KOC = 10)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

- ► Containers may still present a chemical hazard/ danger when empty.
- ▶ Return to supplier for reuse/ recycling if possible.

Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- ▶ Reduction
- ▶ Reuse
- ▶ Recycling
- ► Disposal (if all else fails)

Product / Packaging disposal

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- ▶ **DO NOT** allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- ▶ Where in doubt contact the responsible authority.
- ▶ Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material).
- ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant



HAZCHEM

Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

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BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID(25085-99-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)

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

WATER(7732-18-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

ISOTHIAZOLINONES, MIXED(55965-84-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

SODIUM NITRATE(7631-99-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)

PROPYLENE GLYCOL MONOBUTYL ETHER - ALPHA ISOMER(5131-66-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

BIS(1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL)SEBACATE(41556-26-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous

Chemicals

Australia Inventory of Chemical Substances (AICS)

METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE(82919-37-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

DIETHYLENE GLYCOL MONOBUTYL ETHER(112-34-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous

Chemicals Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)

Australia Standard for the Uniform Scheduling of Medicines and Poisons

(SUSMP) - Schedule 5

National Inventory Status

National Inventory	Status
Australia - AICS	No (isothiazolinones, mixed)
Canada - DSL	Yes
Canada - NDSL	No (propylene glycol monobutyl ether - alpha isomer; bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; diethylene glycol monobutyl ether; bisphenol A/ diglycidyl ether resin, liquid; isothiazolinones, mixed; water; sodium nitrate; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (isothiazolinones, mixed)
Japan - ENCS	No (isothiazolinones, mixed)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (isothiazolinones, mixed)
Legend:	Yes = All ingredients are on the inventory No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

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Other information

Ingredients with multiple cas numbers

Name	CAS No
bisphenol A/ diglycidyl ether resin, liquid	25068-38-6, 25085-99-8
isothiazolinones, mixed	55965-84-9, 96118-96-6

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

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

Definitions and abbreviations

PC-TWA: Permissible Concentration-Time Weighted Average PC-STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index

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