Environmental Envirotex Lite Resin Damar Industries Limited

Chemwatch: 7087953 Version No: 7.1.7.10

Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Chemwatch Hazard Alert Code: 2

Issue Date: 23/03/2020 Print Date: 31/08/2021 L.GHS.NZL.EN

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

Product	Identifier
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Product name	Environmental Envirotex Lite Resin
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid and (C12-14)alkylglycidyl ether)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	High gloss coating.
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Details of the supplier of the safety data sheet

Registered company name	Damar Industries Limited
Address	800 Te Ngae Road, Eastgate Park, Rotorua 3042 New Zealand
Telephone	+64 7 345 6007
Fax	+64 7 345 6019
Website	www.damarindustries.com
Email	info@damarindustries.co.nz

Emergency telephone number

Association / Organisation	CHEMCALL
Emergency telephone numbers	0800 243 622
Other emergency telephone numbers	1800 127 406 (outside New Zealand)

SECTION 2 Hazards identification

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Classified as Dangerous Goods for transport purposes.

Classification ^[1]	Serious Eye Damage/Eye Irritation Category 2, Sensitisation (Skin) Category 1, Specific Target Organ Toxicity - Single Exposure Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2, Skin Corrosion/Irritation Category 2
Legend:	1. Classified by Chernwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	6.3A, 6.4A, 6.5B (contact), 6.9B, 9.1B

Label elements

pictogram(s)			×
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Signal word

Warning

Hazard statement(s)

Hazard

H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H371	May cause damage to organs.
H373	May cause damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.

H315 Causes skin irritation.

Precautionary statement(s) Prevention

roodationally of the officer	
P260	Do not breathe mist/vapours/spray.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of 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.
P308+P311	IF exposed or concerned: Call a POISON CENTER/doctor/physician/first aider.
P314	Get medical advice/attention if you feel unwell.
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.

Precautionary statement(s) Disposal

P501 Disp

Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
25068-38-6	4-90	bisphenol A/ diglycidyl ether resin, liquid
68609-97-2	1-40	(C12-14)alkylglycidyl ether
122-60-1	NotSpec	phenyl glycidyl ether
80-05-7	NotSpec	bisphenol A
Legend: 1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 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: 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	 If fumes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
Ingestion	 For advice, contact a Poisons Information Centre or a doctor at once. Urgent hospital treatment is likely to be needed. If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

Extinguishing media

- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog Large fires only.
- Do not use water jets.

Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
Advice for firefighters	
Fire Fighting	 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	 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). May emit acrid smoke. Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO2) other pyrolysis products typical of burning organic material.

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	 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. 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	 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. 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. Collect recoverable product into labelled containers for recycling. Absorb remaining product with sand, earth or vermiculite. Collect solid residues and sea 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	 DO NOT allow clothing wet with material to stay in contact with skin 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. Work clothes should be laundered separately.
	 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.
	Store in original containers.
	 Keep containers securely sealed. No smoking, naked lights or ignition sources.
Other information	 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	 Metal can or drum Packaging as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.
Storage incompatibility	In general, uncured epoxy resins have only poor mechanical, chemical and heat resistance properties. However, good properties are obtained by reacting the linear epoxy resin with suitable curatives to form three-dimensional cross-linked thermoset structures. This process is commonly referred to as curing or gelation process. Curing of epoxy resins is an exothermic reaction and in some cases produces sufficient heat to cause thermal degradation if not controlled. Curing may be achieved by reacting an epoxy with itself (homopolymerisation) or by forming a copolymer with polyfunctional curatives or hardeners. In principle, any molecule containing a reactive hydrogen may react with the epoxide groups of the epoxy resin. Common classes of hardeners for epoxy resins include amines, acids, acid anhydrides, phenols, alcohols and hols. Relative reactivity (lowest first) is approximately in the order: phenol < anhydride < aromatic amine < cycloaliphatic amine < aliohatic amine < thiol. The epoxy curing reaction may be accelerated by addition of small quantities of accelerators. Tertiary amines, carboxylic acids and alcohols (especially phenols) are effective accelerators. Bisphenol A is a highly effective and widely used accelerator, but is now increasingly replaced due to health concerns with this substance. Epoxy resin may be reacted with liself in the presence of an anionic catalyst (a Lewis base such as tertiary amines or imidazoles) or a cationic catalyst (a Lewis acid such as a boron trifluoride complex) to form a cured network. This process is known as catalytic homopolymerisation. The resulting network contains only ether bridges, and exhibits high thermal and chemical resistance, but is britle and often requires elevated temperature to effect curing, so finds only niche applications industrially. Epoxy homopolymerisation is often used when there is a requirement for UV curing, since cationic UV catalysts may be employed (e.g. for UV coatings). Reactive diluents are stable under recommended storage conditions, bu

SECTION 8 Exposure controls / personal protection

Control parameters

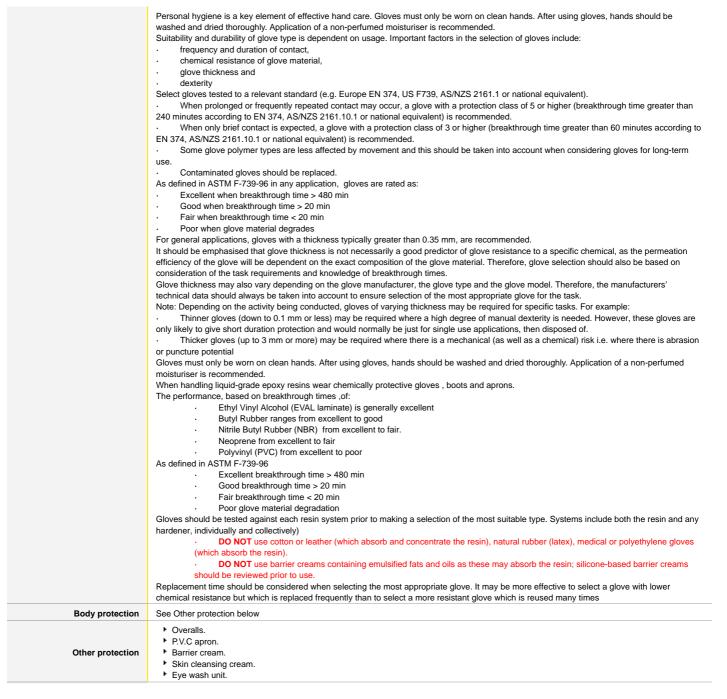
Occupational Exposure Limits (OEL)

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	bisphenol A/ diglycidyl ether resin, liquid	Particulates not otherwise classified	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	bisphenol A/ diglycidyl ether resin, liquid	Particulates not otherwise classified respirable dust	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	phenyl glycidyl ether	Phenyl glycidyl ether (PGE)	0.1 ppm / 0.6 mg/m3	Not Available	Not Available	skin-Skin absorption (dsen)-Dermal sensitiser 6.7B-Suspected carcinogen
New Zealand Workplace Exposure Standards (WES)	bisphenol A	Particulates not otherwise classified respirable dust	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	bisphenol A	Particulates not otherwise classified	10 mg/m3	Not Available	Not Available	Not Available

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Ingredient	TEEL-1	TEEL-2	TEEL-3
bisphenol A/ diglycidyl ether resin, liquid	90 mg/m3	990 mg/m3	5,900 mg/m3
bisphenol A	15 mg/m3	110 mg/m3	650 mg/m3

Ingredient	Original IDLH	Revised IDLH	
bisphenol A/ diglycidyl ether	Not Available Not Available		
resin, liquid			
(C12-14)alkylglycidyl ether	Not Available	Not Available	
phenyl glycidyl ether	100 ppm	Not Available	
bisphenol A	Not Available	Not Available	
Occupational Exposure Banding	3		
Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit	
(C12-14)alkylglycidyl ether	E	≤ 0.1 ppm	
Notes:	Occupational exposure banding is a process of assigning ch adverse health outcomes associated with exposure. The out range of exposure concentrations that are expected to prote	put of this process is an occupational exposure band (OEB)	
MATERIAL DATA			
Exposure controls	Engineering controls are used to remove a hazard or place a be highly effective in protecting workers and will typically be The basic types of engineering controls are: Process controls which involve changing the way a job activi Enclosure and/or isolation of emission source which keeps a "adds" and "removes" air in the work environment. Ventilatio ventilation system must match the particular process and che Employers may need to use multiple types of controls to pre- General exhaust is adequate under normal operating conditi overexposure exists, wear approved respirator. Correct fit is or closed storage areas. Air contaminants generated in the v velocities" of fresh circulating air required to effectively remo- Type of Contaminant: solvent, vapours, degreasing etc., evaporating from tank (i aerosols, fumes from pouring operations, intermittent conta drift, plating acid fumes, pickling (released at low velocity i direct spray, spray painting in shallow booths, drum filling, generation into zone of rapid air motion) grinding, abrasive blasting, tumbling, high speed wheel ge very high rapid air motion). Within each range the appropriate value depends on: Lower end of the range 1: Room air currents minimal or favourable to capture 2: Contaminants of low toxicity or of nuisance value only. 3: Intermittent, low production. 4: Large hood or large air mass in motion Simple theory shows that air velocity falls rapidly with distance with the square of distance from the extraction point (in simp accordingly, after reference to distance from the contaminatii 1-2 m/s (200-400 f/min) for extraction of solvents generated producing performance deficits within the extraction apparati	independent of worker interactions to provide this high level ty or process is done to reduce the risk. selected hazard "physically" away from the worker and ven in can remove or dilute an air contaminant if designed proper emical or contaminant in use. vent employee overexposure. ons. Local exhaust ventilation may be required in specific cit essential to obtain adequate protection. Provide adequate v vorkplace possess varying "escape" velocities which, in turn ve the contaminant. In still air). ainer filling, low speed conveyer transfers, welding, spray nto zone of active generation) conveyer loading, crusher dusts, gas discharge (active merated dusts (released at high initial velocity into zone of Upper end of the range 1: Disturbing room air currents 2: Contaminants of high toxicity 3: High production, heavy use 4: Small hood-local control only the cases). Therefore the air speed at the extraction pipe. Veloci in a tank 2 meters distant from the extraction point. Other more in a tank 2 meters distant from the extraction point. Other more the set of the range to the air speed at the extraction point. Other more in a tank 2 meters distant from the extraction point. Other more in a tank 2 meters distant from the extraction point. Other more the set of the trange to the set of the transpecies at the extraction point. Other more in a tank 2 meters distant from the extraction point. Other more the set of the transpecies at the extraction point. Other more the transfer the set of the transpecies at the extraction point. Other more the transfer the set of the transpecies at the extraction point. Other more in a tank 2 meters distant from the extraction point. Other more in the extraction point. Other more	of protection. tilation that strategically ty. The design of a cumstances. If risk of entilation in warehouse determine the "capture Air Speed: 0.25-0.5 m/s (50-100 f/min) 0.5-1 m/s (100-200 f/min.) 1-2.5 m/s (200-500 f/min.) 2.5-10 m/s (500-2000 f/min.) ty generally decreases build be adjusted, should be a minimum of echanical considerations
Personal protection	more when extraction systems are installed or used.		
Eye and face protection	the wearing of lenses or restrictions on use, should be c and adsorption for the class of chemicals in use and an their removal and suitable equipment should be readily a remove contact lens as soon as practicable. Lens should	lenses may absorb and concentrate irritants. A written policy reated for each workplace or task. This should include a revi account of injury experience. Medical and first-aid personnel available. In the event of chemical exposure, begin eye irriga d be removed at the first signs of eye redness or irritation - le nds thoroughly. [CDC NIOSH Current Intelligence Bulletin 55	ew of lens absorption should be trained in tion immediately and ens should be removed i
Skin protection	See Hand protection below		
Hands/feet 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.		



Respiratory protection

Type A-P 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 P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.

- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

Information on basic physical and chemical properties

Appearance Clear viscous liquid with mild characteristic odour; partly mixes with water.			
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Characteristic, Slight	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)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>200 (SCC)	Taste	Not Available
Evaporation rate	Not Available	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)	Negligible
Vapour pressure (kPa)	<0.133	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (%)	Not Available
Vapour density (Air = 1)	>1	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

inormation on toxicological ci	
Inhaled	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. 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.
Ingestion	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. Male rats exposed to a single oral dose of bisphenol A diglycidyl ether (BADGE) at 750, 1000, and 2000 mg/kg/day showed a significantly increase in the number of immature and maturing sperm on the testis. There were no significant differences with respect to sperm head count, sperm motility, and sperm abnormality in the BADGE treatment groups The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
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 level 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 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, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmfu

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 Eve (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. 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. 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. Substances than can cuase 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 Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. 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. Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests 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 Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence 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. 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 though 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 Chronic A may be one of the causes of congenital masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that "it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades" One review has concluded that obesity may be increased as a function of bisphenol A exposure, which "...merits concern among scientists and public health officials' One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood. A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, "these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls". Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells. [whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A. during lactation. increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes. Bisphenol A is the isopropyl adduct of 4,4-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called "cytostatic hormones". Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown to be oestrogenic in vitro; such sealants may represent an additional source of xenoestrogens in humans and may be the cause of additional concerns in children. Concerns have been raised about the possible developmental effects on the foetus/embryo or neonate resulting from the leaching of bisphenol A from epoxy linings in metal cans which come in contact with food-stuffs. Many drugs, including naproxen, salicylic acid, carbamazepine and mefenamic acid can, in vitro, significantly inhibit bisphenol A glucuronidation (detoxification). BPA belongs to the list of compounds having this property as the rodent models have shown that BPA exposure is linked with increased body weigh (obesogens)t. Several mechanisms can help explain the effect of BPA on body weight increase. A possible mechanism leading to triglyceride accumulation is the decreased production of the hormone adiponectin from all human adipose tissue tested when exposed to very low levels (below nanomolar range) of BPA in cell or explant culture settings . The expression of leptin as well as several enzymes and transcription factors is also affected by BPA exposure in vivo as well as in vitro. Together, the altered expression and activity of these important mediators of fat metabolism could explain the increase in weight following BPA exposure in rodent models. These results also suggest that, together with other obesogens, low, environmentally relevant levels of BPA may contribute to the human obesity phenomenon. All glycidyl ethers show genotoxic potential due 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Environmental Envirotex Lite	ΤΟΧΙΟΙΤΥ	IRRITATION		
Resin	Not Available	Not Available		
	ΤΟΧΙΟΙΤΥ	IRRITATION		
bisphenol A/ diglycidyl ether resin, liquid	dermal (rat) LD50: >1200 mg/kg ^[2]	Eye (rabbit): 100mg - Mild		
resin, iiquiu	Oral(Mouse) LD50; >500 mg/kg ^[2]			
	тохісіту	IRRITATION		
	Oral(Rat) LD50; >2000 mg/kg ^[1]	Eye (rabbit): mild [Ciba]		
		Eye: adverse effect observed (irritating) ^[1]		
		Skin (guinea pig): sensitiser		
(C12-14)alkylglycidyl ether		Skin (human): Irritant		
		Skin (human): non- sensitiser		
		Skin (rabbit): moderate		
		Skin : Moderate		
		Skin: adverse effect observed (irritating) ^[1]		
	ΤΟΧΙΟΙΤΥ	IRRITATION		
	dermal (mouse) LD50: 760 mg/kg ^[1]	Eye (rabbit): 0.25 mg/24h-SEVERE		
phenyl glycidyl ether	Inhalation(Rat) LC50; 1.984 mg/L4h ^[1]	Eye (rabbit): 111 mg - mild		
	Oral(Mouse) LD50; 1400 mg/kg ^[2]	Skin (rabbit): 10 mg/24h-SEVERE		
		Skin (rabbit): 24 mg/24h-moderate		
	ΤΟΧΙΟΙΤΥ	IRRITATION		
	Dermal (rabbit) LD50: ~3000 mg/kg ^[1]	Eye (rabbit): 0.25 mg/24h-SEVERE		
	Oral(Rat) LD50; 1200 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]		
bisphenol A		Skin (rabbit): 250 mg open - mild		
		Skin (rabbit): 500 mg/24h - mild		
		Skin: adverse effect observed (irritating) ^[1]		
		Skin: no adverse effect observed (not irritating) ^[1]		
Legend:	1. Value obtained from Europe ECHA Registered Substar specified data extracted from RTECS - Register of Toxic E	nces - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise Effect of chemical Substances		
	Foetoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity; NOEL (maternal 60 mg/kg The substance is classified by IARC as Group 3:			
	NOT classifiable as to its carcinogenicity to humans.			
BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	Evidence of carcinogenicity may be inadequate or limited in animal testing. 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			

	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 C3T mice; it was, however, weakly carcinogenic to the skin of C3TBL/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 TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 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
	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.
(C12-14)ALKYLGLYCIDYL ETHER	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 enhalation one to be served in mice exposed chronically via deministered 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
PHENYL GLYCIDYL ETHER	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. For bisphenol A (BPA) Following oral administration absorption of BPA is rapid and extensive while dermal absorption is limited. Extensive first pass metabolism occurs
BISPHENOL A	Following brai administration absorption of tePA is rapid and extensive while dermal absorption is imited. Extensive first pass metabolism occurs following absorption from the gastrointestinal tract with glucuronide conjugation being the major metabolic pathway. Bisphenol A is of low acute toxicity (rodent oral LD50 values from 3300-4100 mg/kg, ar abbit oral LD50 value 2230 mg/kg and a rat acute inhalation 6-hour LC50 value s170 mg/m3). Bisphenol A is not a skin irritant, however, it is severely irritating to the eyes. BPA was negative in gene mutation and clastogenicity assays in cultured mammalian cells, as well as in a micronucleus test for clastogenicity <i>in vivo</i> ; therefore, BPA is considered not to present a genotoxic concern for human health. BPA results in minimal effects on the liver and kidney (LOAEL from chronic exposure in the diet was 50 mg/kg/day). For reproductive toxicity, data from a three-generation study in the rat, BPA was not a selective reproductive toxicant at doses ranging from 0.001 to 500 mg/kg/day. BPA is not a developmental toxicant in rats or mice. Inconsistent findings are reported in the "low dose" literature for bisphenol A. The inherent challenge of conducting these types of studies may be exacerbated with bisphenol A because the endpoints of concern are endocrine-mediated and potentially impacted by factors that include phytoestrogen content of the animal feed, extent of bisphenol A exposure from caging or water bottles, and the alleged sensitivity of the animal model to oestrogens. Further, none of these studies with multiple dose groups using several strains of rats and mice have been conducted and none of these detected any adverse reproductive effects at low to moderate dosage levels of BPA administered via the relevant route of human exposures. Further, none of these studies and tholecular alterations of endocrine function also produces a cascade of effects increasing in severity resulting in clearly adverse alterations at higher doses, albeit the effects can be different

though it is not clear the reported effects constitute an adverse toxicological response. In summary: For pregnant women and foetuses, the Expert Panel has different levels of concern for the different developmental endpoints that may be susceptible to bisphenol A disruption, as follows: For neural and behavioral effects, the Expert Panel has some concern; For prostate effects, the Expert Panel has minimal concern; For the potential effect of accelerated puberty, the Expert Panel has minimal concern; and For birth defects and malformations, the Expert Panel has negligible concern. For infants and children, the Expert Panel has the following levels of concern for biological processes that might be altered by Bisphenol A, as follows Some concern for neural and behavioral effects: and Minimal concern for the effect of accelerated puberty. For adults, the Expert Panel has negligible concern for adverse reproductive effects following exposures in the general population to Bisphenol A. For highly exposed subgroups, such as occupationally exposed populations, the level of concern is elevated to minimal NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Bisphenol A National Toxicology Program US Department of Health and Human Services September 2008 NTP Publication No 08-5994 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. The following information refers to contact allergens as a group and may not be specific to this product. **BISPHENOL A/ DIGLYCIDYL** Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact ETHER RESIN. LIQUID & eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria (C12-14)ALKYLGLYCIDYL involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the ETHER & PHENYL GLYCIDYL distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely ETHER & BISPHENOL A distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics. Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative **BISPHENOL A/ DIGLYCIDYL** potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active ETHER RESIN, LIQUID & compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular BISPHENOL A configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor. 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 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. 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 PHENYL GLYCIDYL ETHER & spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal **BISPHENOL A** 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 Ý ~ Serious Eye Damage/Irritation STOT - Single Exposure Respiratory or Skin -STOT - Repeated Exposure sensitisation × × Mutagenicity Aspiration Hazard

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

SECTION 12 Ecological information

	Endpoint	Test Duration (hr)	Species	Value	Source
Environmental Envirotex Lite Resin	Not Available	Not Available	Not Available	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	~2mg/l	2
	EC50(ECx)	48h	Crustacea	~2mg/l	2
	Endpoint	Test Duration (hr)	Species	Value	Source
(C12-14)alkylglycidyl ether	EC50(ECx)	48h	Crustacea	6.07mg/l	2
	LC50	96h	Fish	>5000mg/l	2

	EC50	48h	Crustacea		6.07mg/l	2
	Endpoint	Test Duration (hr)	Species		Value	Sourc
phenyl glycidyl ether	LC50	96h	Fish	Fish 43mg/		2
	EC50(ECx)	96h	Fish		43mg/L	5
	Endpoint	Test Duration (hr)	Species	Value		Sourc
	ErC50	72h	Algae or other aquatic plants	2.7-3.1mg/l		1
	NOEC(ECx)	96h	Crustacea	0.51mg/l		1
	BCF	1008h	Fish	5.1	13.3	7
bisphenol A	EC50	72h	Algae or other aquatic plants	1.2	5-1.89mg/l	4
	LC50	96h	Fish	3-5mg/l		2
	EC50	48h	Crustacea	10.2mg/l		1
	EC50	96h	Algae or other aquatic plants	1m	a/l	1

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.

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

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. 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 (46 h): 1.1 mg/l; NOEC 0.023 mg/l - 328 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)sulfore) 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.

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).

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.

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.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air	
bisphenol A/ diglycidyl ether resin, liquid	HIGH	HIGH	
phenyl glycidyl ether	LOW	LOW	
bisphenol A	HIGH (Half-life = 360 days)	LOW (Half-life = 0.31 days)	

Bioaccumulative potential

Ingredient	Bioaccumulation
bisphenol A/ diglycidyl ether resin, liquid	LOW (LogKOW = 2.6835)
phenyl glycidyl ether	LOW (LogKOW = 1.6136)
bisphenol A	LOW (BCF = 100)

Mobility in soil

Ingredient	Mobility
bisphenol A/ diglycidyl ether resin, liquid	LOW (KOC = 51.43)
phenyl glycidyl ether	LOW (KOC = 41.09)
bisphenol A	LOW (KOC = 75190)

SECTION 13 Disposal considerations

Waste treatment methods	
Product / Packaging disposal	 Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. Otherwise: If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product. Waste Management Production waste from epoxy resins and resin systems should be treated as hazardous waste in accordance with National regulations. Fire retarded resins containing halogenated compounds should also be treated as special waste. Accidental spillage of resins, curing agents and their formulations should be contained and absorbed by special mineral absorbents to prevent them from entering the environment. Contaminated or surplus product should not be washed down the sink, but preferably be fully reacted to form cross-linked solids which is non-hazardous and can be more easily disposed. Finished articles made from thuly cured epoxy resins are hard, infusible solids presenting no hazard to the environment. However, finished articles from flame-retarded material containing halogenated resins should be considered hazardous waste, and disposed as required by National laws. Atticles made from epoxy resins, like other thermosets, can be recycled by ginding and used as fillers in other products. Another way of disposal and recovery is combustion with energy recovery. 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 Reso Bisposal (if all les fails) This meterial may be recycled if unsed, or if it has not

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. M. Suzuki, and E. Musashi J Appl Polym Sci, 118(2):721 - 732; October 2010 F. 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.

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous. Only dispose to the environment if a tolerable exposure limit has been set for the substance.

Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 Transport information

Labels Required	
Marine Pollutant	
HAZCHEM	•3Z
Land transport (UN)	

and transport (UN)

UN number	3082		
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid and (C12-14)alkylglycidyl ether)		
Transport hazard class(es)	Class 9 Subrisk Not Applicable		
Packing group	II		
Environmental hazard	Environmentally hazardous		
Special precautions for user	Special provisions274; 331; 335; 375Limited quantity5 L		

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 and (C12-14)alkylglycidyl ether)			
Transport hazard class(es)	ICAO/IATA Class	9		
	ICAO / IATA Subrisk ERG Code			
Packing group	111			
Environmental hazard	Environmentally hazardous			
	Special provisions		A97 A158 A197 A215	
	Cargo Only Packing Instructions		964	
	Cargo Only Maximum Qty / Pack		450 L	
Special precautions for user	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 and (C12-14)alkylglycidyl ether)

Transport hazard class(es)	IMDG Class	9	
	IMDG Subrisk	Not Applicable	
Packing group	11		
Environmental hazard	Marine Pollutant		
Special precautions for user	EMS Number Special provisions Limited Quantities		

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
(C12-14)alkylglycidyl ether	Not Available
phenyl glycidyl ether	Not Available
bisphenol A	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
bisphenol A/ diglycidyl ether resin, liquid	Not Available
(C12-14)alkylglycidyl ether	Not Available
phenyl glycidyl ether	Not Available
bisphenol A	Not Available

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard	
HSR002670	Surface Coatings and Colourants Subsidiary Hazard Group Standard 2020	

of Chemicals - Classification Data

of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

New Zealand Workplace Exposure Standards (WES)

New Zealand Workplace Exposure Standards (WES)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

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

Chemical Footprint Project - Chemicals of High Concern List

New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) \mbox{Act} - Classification of Chemicals

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

Chemical Footprint Project - Chemicals of High Concern List

New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

phenyl glycidyl ether is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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

New Zealand Approved Hazardous Substances with controls

bisphenol A is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) \mbox{Act} - Classification of Chemicals

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantities
Not Applicable	Not Applicable

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg) Maximum quantity per package for each classification	
6.5A or 6.5B	120	1	3	

Tracking Requirements

Not Applicable

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; (C12-14)alkylglycidyl ether; phenyl glycidyl ether; bisphenol A)	
China - IECSC	Yes	
Europe - EINEC / ELINCS / NLP	Yes	
Japan - ENCS	No ((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)	
Vietnam - NCI	Yes	
Russia - FBEPH	Yes	
Legend:	yend: Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.	

SECTION 16 Other information

Revision Date	23/03/2020
Initial Date	03/03/2015

SDS Version Summary

Version	Date of Update	Sections Updated
6.1.1.1	20/03/2020	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Appearance, Chronic Health, Classification, Disposal, Environmental, Fire Fighter (extinguishing media), Fire Fighter (fire/explosion hazard), First Aid (swallowed), Ingredients, Personal Protection (hands/feet), Physical Properties, Spills (major), Spills (minor), Storage (storage incompatibility), Synonyms, Transport, Transport Information
7.1.1.1	23/03/2020	Acute Health (skin), Appearance, Ingredients
7.1.2.1	29/04/2021	Regulation Change
7.1.2.2	30/05/2021	Template Change
7.1.2.3	04/06/2021	Template Change
7.1.2.4	05/06/2021	Template Change
7.1.2.5	09/06/2021	Template Change
7.1.2.6	11/06/2021	Template Change
7.1.3.6	14/06/2021	Regulation Change
7.1.3.7	15/06/2021	Template Change
7.1.3.8	05/07/2021	Template Change
7.1.4.8	14/07/2021	Regulation Change
7.1.4.9	01/08/2021	Template Change
7.1.5.9	02/08/2021	Regulation Change
7.1.6.9	05/08/2021	Regulation Change
7.1.7.9	09/08/2021	Regulation Change
7.1.7.10	29/08/2021	Template Change

Other information

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

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

Definitions and abbreviations

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