SV50 Aqueous Epoxy Part A

On-Crete Australia Pty Ltd

Chemwatch Hazard Alert Code: **3**

Version No: **2.5**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **06/06/2019**
Print Date: **06/06/2019**
L.GHS.AUS.EN

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

Product Identifier

Product name	SV50 Aqueous Epoxy Part A
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid)
Other means of identification	Not Available

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

Relevant identified uses	Part A of an aqueous epoxy system for flooring

Details of the supplier of the safety data sheet

Registered company name	**On-Crete Australia Pty Ltd**
Address	4/489 Scottsdale Drive Varsity Lakes Queensland Australia
Telephone	+61 7 5593 6884
Fax	+61 7 5593 6885
Website	www.on-crete.com.au
Email	info@on-crete.com.au

Emergency telephone number

Association / Organisation	**On-Crete Australia Pty Ltd**
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. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

CHEMWATCH HAZARD RATINGS

		Min	Max
Flammability	0		
Toxicity	0		
Body Contact	3		
Reactivity	0		
Chronic	3		

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable

Continued...

Classification [1]	Skin Corrosion/Irritation Category 1B, Chronic Aquatic Hazard Category 2, Acute Aquatic Hazard Category 3, Serious Eye Damage Category 1, Reproductive Toxicity Category 1A, Skin Sensitizer Category 1, Germ cell mutagenicity Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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

Hazard statement(s)

H314	Causes severe skin burns and eye damage.
H411	Toxic to aquatic life with long lasting effects.
H402	Harmful to aquatic life.
H360FD	May damage fertility. May damage the unborn child.
H317	May cause an allergic skin reaction.
H341	Suspected of causing genetic defects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

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

Precautionary statement(s) Storage

P405	Store locked up.

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

Continued...

CAS No	%[weight]	Name
25068-38-6	10-30	bisphenol A/ diglycidyl ether resin, liquid
26447-14-3	<10	cresyl glycidyl ether
9038-95-3	<1	monobutyl ether ethoxylated, propoxylated
872-50-4	<1	N-methyl-2-pyrrolidone
55965-84-9	<1	isothiazolinones, mixed
7631-99-4	<1	sodium nitrate
330-54-1	<1	diuron
10605-21-7	<1	carbendazim
556-67-2	<1	octamethylcyclotetrasiloxane

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor.
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.
for diuron:
- Symptomatic and supportive action is indicated.
- Methaemoglobinaemia is possible
- if compound is hydrolysed in vivo to aniline.
- Methaemoglobinaemia causes cyanosis. Reversion of methaemoglobin to haemoglobin is spontaneous after removal from exposure, so moderate degrees of cyanosis need be treated only by supportive measures such as bed rest and oxygen inhalation.
- Thorough cleansing of the entire contaminated area of the body, including the scalp and nails is of the utmost importance.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

Continued...

- Water spray or fog - Large fires only.

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 breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use water delivered as a fine spray to control fire and cool adjacent 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	Combustible. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO_2) metal oxides other pyrolysis products typical of burning organic material.
HAZCHEM	•3Z

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ Clean up all spills immediately. ▸ Avoid contact with skin and eyes. ▸ Wear impervious gloves and safety goggles. ▸ Trowel up/scrape up. ▸ Place spilled material in clean, dry, sealed container. ▸ Flush spill area with water.
Major Spills	Environmental hazard - contain spillage. Minor hazard. ▸ Clear area of personnel. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Control personal contact with the substance, by using protective equipment as required. ▸ Prevent spillage from entering drains or water ways. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▸ Wash area and prevent runoff into drains or waterways. ▸ 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	▸ 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. ▸ Keep containers securely sealed when not in use.

Continued...

	▸ 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 maintained.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Titanium dioxide ▸ reacts with strong acids, strong oxidisers ▸ reacts violently with aluminium, calcium, hydrazine, lithium (at around 200 deg C.), magnesium, potassium, sodium, zinc, especially at elevated temperatures - these reactions involves reduction of the oxide and are accompanied by incandescence ▸ dust or powders can ignite and then explode in a carbon dioxide atmosphere ▸ WARNING: Avoid or control reaction with peroxides. All *transition metal* peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. ▸ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive. ▸ Avoid reaction with borohydrides or cyanoborohydrides ▸ Avoid reaction with amines, mercaptans, strong acids and oxidising agents Cellulose and its derivatives may react vigorously with calcium oxide, bleaching powder, perchlorates, perchloric acid, sodium chlorate, fluorine, nitric acid, sodium nitrate and sodium nitrite. May be incompatible with aminacrine hydrochloride, chlorocresol, mercuric chloride, phenol, resorcinol, tannic acid and silver nitrate. Glycidyl ethers: ▸ may form unstable peroxides on storage in air ,light, sunlight, UV light or other ionising radiation, trace metals - inhibitor should be maintained at adequate levels ▸ may polymerise in contact with heat, organic and inorganic free radical producing initiators ▸ may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines ▸ react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide ▸ attack some forms of plastics, coatings, and rubber

\+ X + O + + +

X — Must not be stored together
0 — 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

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	N-methyl-2-pyrrolidone	1-Methyl-2-pyrrolidone	25 ppm / 103 mg/m3	309 mg/m3 / 75 ppm	Not Available	Not Available
Australia Exposure Standards	diuron	Diuron	10 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3

Continued...

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
N-methyl-2-pyrrolidone	Methyl 2-pyrrolidinone, 1-; (N-Methylpyrrolidone)	30 ppm	32 ppm	190 ppm
sodium nitrate	Sodium nitrate	4.1 mg/m3	45 mg/m3	270 mg/m3
octamethylcyclotetrasiloxane	Octamethylcyclotetrasiloxane	30 ppm	68 ppm	130 ppm

Ingredient	Original IDLH	Revised IDLH
bisphenol A/ diglycidyl ether resin, liquid	Not Available	Not Available
cresyl glycidyl ether	Not Available	Not Available
monobutyl ether ethoxylated, propoxylated	Not Available	Not Available
N-methyl-2-pyrrolidone	Not Available	Not Available
isothiazolinones, mixed	Not Available	Not Available
sodium nitrate	Not Available	Not Available
diuron	Not Available	Not Available
carbendazim	Not Available	Not Available
octamethylcyclotetrasiloxane	Not Available	Not Available

MATERIAL DATA

for N-methyl-2-pyrrolidone (NMP):
Reports of skin and eye irritation and chronic headaches have been reported in workers exposed to 1-methyl-2-pyrrolidone. The Australian ES is based on a 10-fold uncertainty factor of the no-observable-adverse-effect level (NOAEL) of 24 ppm where adverse respiratory effects were observed in a 4-week inhalation study in rats.
for diuron:
Exposures at or below the recommended TLV-TWA is thought to protect the worker from the significant risk of anaemia and methaemoglobinaemia associated with use of the product.

Animals exposed by inhalation to 10 mg/m3 titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

Cellulose is considered a nuisance dust which has little adverse effect on lung and does not produce significant organic disease or toxic effects when appropriate controls are applied.
for propylene glycol monomethyl ether (PGME)
Odour Threshold: 10 ppm.
The TLV-TWA is protective against discomfort caused by odour, against eye and skin irritation, and chronic effects (including possible liver and kidney damage).
Individuals exposed to 100 ppm reported a transient unpleasant odour with slight eye irritation after about 1 or 2 hours. At 300 ppm, mild irritation of the eyes and nose developed within 5 minutes; some individuals found the irritation hardly bearable after about an hour. A concentration of 750 ppm was highly irritating. Signs of central nervous system depression developed at 1000 ppm. Neurological, clinical chemical and general medical examinations showed no other conspicuous toxicity.
Concentrations of the beta-isomer, 2-methoxy-1-propyl acetate are low in commercial grades of PGME and teratogenic effects associated with this isomer are expected to be absent.
Odour Safety Factor(OSF)
OSF=10 (propylene glycol monomethyl ether)
For epichlorohydrin
Odour Threshold Value: 0.08 ppm
NOTE: Detector tubes for epichlorohydrin, measuring in excess of 5 ppm, are commercially available.
Exposure at or below the recommended TLV-TWA is thought to minimise the potential for adverse respiratory, liver, kidney effects. Epichlorohydrin has been implicated as a human skin sensitiser, hence individuals who are hypersusceptible or otherwise unusually responsive to certain chemicals may NOT be adequately protected from adverse health effects.
Odour Safety Factor (OSF)
OSF=0.54 (EPICHLOROHYDRIN)

For ethylene glycol monobutyl ether (2-butoxyethanol)
Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)
Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.
Odour Safety Factor (OSF)
OSF=2E2 (2-BUTOXYETHANOL)

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions

Continued...

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

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

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

Within each range the appropriate value depends on:

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

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

Personal protection

Eye and face protection

- Chemical goggles.
- Full face shield may be required for supplementary but never for primary protection of eyes.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

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

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

Continued...

	· Polyvinyl (PVC) from excellent to poor As defined in ASTM F-739-96 · Excellent breakthrough time > 480 min · Good breakthrough time > 20 min · Fair breakthrough time < 20 min · Poor glove material degradation Gloves should be tested against each resin system prior to making a selection of the most suitable type. Systems include both the resin and any hardener, individually and collectively) · **DO NOT** use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin). · **DO NOT** use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use. Replacement time should be considered when selecting the most appropriate glove. It may be more effective to select a glove with lower chemical resistance but which is replaced frequently than to select a more resistant glove which is reused many times
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ 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:
SV50 Aqueous Epoxy Part A

Material	CPI
BUTYL	A
PE/EVAL/PE	A
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NEOPRENE	C
NITRILE	C
PVA	C
PVC	C
SARANEX-23	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

*** - Negative pressure demand ** - Continuous flow**
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(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)

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Reactive diluents are generally colourless to yellow/ amber, low viscosity liquids with mild ether-like odour; solubility in water varies across the family. May contain trace residuals of epichlorohydrin a known skin irritant. |white paste

Physical state	Free-flowing Paste	**Relative density (Water = 1)**	1.19
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available

Continued...

Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	7-8.5	**Decomposition temperature**	>200
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	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	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	▸ 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

Inhaled	
Ingestion	The material can produce chemical burns within the oral cavity and gastrointestinal tract following 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	The material can produce chemical burns following direct contact with the skin. 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. 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 harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

Continued...

	The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: ▸ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but mild, 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 (non allergic). 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.
Eye	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. 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. There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility 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. 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 Chronic effects of exposure to diuron may initially include skin irritation, or blurring of vision, liver enlargement; spleen and thyroid effects; red blood cell destruction; or reduction of the blood's oxygen carrying capacity with cyanosis (bluish discolourisation), weakness or shortness of breath by formation of methemoglobin. Significant skin permeation after contact appears unlikely. There are no reports of human sensitisation to diuron. At 2500 ppm diuron in the diet for 2 years, rats and dogs showed growth retardation, slight anaemia, presence of abnormal pigmentation, increased erythropoiesis and splenic haemosiderosis. Long term exposure to the dusts of titanium and several of its compounds produces chronic lung disease (fibrosis) in animals. Radiological evidence exists amongst titanium dioxide workers suggesting chronic lung changes which resemble a slight form of silicosis. Workers chronically exposed to titanium or titanium dioxide dusts show a high incidence of chronic bronchitis (endobronchitis and peribronchitis). Early stages of this disease are characterised by impaired pulmonary respiration and ventilatory capacity and by reduced blood alkalinity. Cardiac changes characteristic of pulmonary disease (with hypertrophy of the right auricle) have also been observed amongst workers. Titanium employed in implants has provoked immune responses which occur locally as metallosis and systemically as raised serum levels of activated T-lymphocytes. Some concern has been expressed about the potential for generating bone-resorbing mediators associated with titanium wear-debris.

Continued...

The largest of the cohort studies was among white male production workers in the titanium dioxide industry in six European countries. The study indicated a slightly increased risk for lung cancer compared with the general population. However, there was no evidence of an exposure-response relationship within the cohort. No increase in the mortality rates for kidney cancer was found when the cohort was compared with the general population, but there was a suggestion of an exposure-response relationship in internal analyses. The other cohort studies, both of which were conducted in the USA, did not report an increased risk for lung cancer or cancer at any other site; no results for kidney cancer were reported, presumably because there were few cases.
One population-based case-control study conducted in Montreal did not indicate an increased risk for lung or kidney cancer.
In summary, the studies do not suggest an association between occupational exposure to titanium dioxide as it occurred in recent decades in western Europe and North America and risk for cancer.
All the studies had methodological limitations; misclassification of exposure could not be ruled out. None of the studies was designed to assess the impact of particle size (fine or ultrafine) or the potential effect of the coating compounds on the risk for lung cancer.
An increased incidence of lung adenomas in rats of both sexes and of cystic keratinising lesions, diagnosed as squamous cell carcinomas in female rats, was seen in animals subject to high doses of inhaled titanium dioxide. Intratracheal delivery of titanium dioxide in combination with benz[a]pyrene produced an increase in benign and malignant tumours of the larynx, trachea and lungs in hamsters.
Squamous cell carcinomas developed after exposure to 250 mg/m3 for 6 hours/day, 5 days/week for 2 years in rats; the type of carcinoma that developed was considered to be a unique experimentally induced tumour and to be of questionable relevance for extrapolation of the results to humans. Given the extremely high level of dust in the lungs, the carcinomas were postulated to be the result of saturation of the normal pulmonary clearance mechanisms. At 50 mg/m3, massive accumulations of dust-laden macrophages, foamy dust cells and free particles were considered indicative of such overload.
On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

SV50 Aqueous Epoxy Part A

TOXICITY	IRRITATION
Not Available	Not Available

bisphenol A/ diglycidyl ether resin, liquid

TOXICITY	IRRITATION
dermal (rat) LD50: >1200 mg/kg[2]	Eye (rabbit): 100mg - Mild
Oral (rat) LD50: >1000 mg/kg[2]	

cresyl glycidyl ether

TOXICITY	IRRITATION
dermal (rat) LD50: >2150 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Oral (rat) LD50: 4300 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

monobutyl ether ethoxylated, propoxylated

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 14946 mg/kg[2]	Skin (rabbit): 500 mg open mild
Inhalation (rat) LC50: 0.147 mg/l/4h**[2]	
Oral (rat) LD50: 4240 mg/kg[2]	

N-methyl-2-pyrrolidone

TOXICITY	IRRITATION
dermal (rat) LD50: 2500-5000 mg/kg[2]	Eye (rabbit): 100 mg - moderate
Inhalation (rat) LC50: 8290.5297 mg/l/4H[2]	
Oral (rat) LD50: 3914 mg/kg[2]	

isothiazolinones, mixed

TOXICITY	IRRITATION
dermal (rat) LD50: >1008 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
Oral (rat) LD50: 53 mg/kg[2]	Skin: adverse effect observed (corrosive)[1]
	Skin: adverse effect observed (irritating)[1]

sodium nitrate

TOXICITY	IRRITATION
dermal (rat) LD50: >5000 mg/kg[1]	Not Available

Continued...

	Oral (rat) LD50: 1267 mg/kg[2]	

	TOXICITY	IRRITATION
diuron	dermal (rat) LD50: >1000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (rat) LD50: 1000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

	TOXICITY	IRRITATION
carbendazim	dermal (rat) LD50: 2000 mg/kg[2]	Eye (rabbit): non-irritating *
	Oral (rat) LD50: >5050 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): non-irritating *
		Skin: no adverse effect observed (not irritating)[1]

	TOXICITY	IRRITATION
octamethylcyclotetrasiloxane	dermal (rat) LD50: 1770 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
	Inhalation (rat) LC50: 36 mg/l/4Hd[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (rat) LD50: 1540 mg/kg[2]	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 Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

SV50 Aqueous Epoxy Part A	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. 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. 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"

Continued...

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).

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
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

Continued...

	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 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 body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. 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. 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 mg/kg
CRESYL GLYCIDYL ETHER	Mutagenic to bacteria

Continued...

MONOBUTYL ETHER ETHOXYLATED, PROPOXYLATED	Polyalkylene glycol monobutyl ether (PGME) polymers have a low degree of toxicity. Toxicity by ingestion is low, but highest for lower molecular weight products *(molecular weight <1500). Thus, the FDA has restricted the use of PGME in sanitizing solutions that may contact surfaces that contact foods to a 0.05% aqueous solution of polymers that have an average molecular weight of 2,400-3,300 and a cloud-point of 90-100 degrees C. In this application, large PGME polymers engulf (micellize) oils and smaller particulates that are subsequently precipitated at temperatures of 90-100 deg C. Toxicity by skin contact is low for all PGME species. Higher molecular weight PGME polymers have been found to be toxic in animals upon inhalation of mechanically generated mists. The products of thermal degradation are also toxic. Eye injury is possible only for the lowest molecular weight species. There have been no reported effects of PGME on human health. However, data from toxicity studies performed in appropriate animal species provides information on potential human toxicity. PGME polymers with molecular weight greater than 1500, have limited acute toxicity by the oral (LD50, rat, 8630 mg/kg) and dermal (LD50, rabbit, >8,000 mg/kg) routes, while toxicity of the high molecular weight products through inhalation is increased (LC50, 4 hours, rat, > 5 mg/kg). Oral and dermal toxicity in general increases with lower molecular weight products. This product has not been evaluated for genetic, developmental, or reproductive toxicity. PGME is not known to be an irritant or an allergen. Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products . Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity . Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates. Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficultto diagnose ACD to these compounds by patch testing. For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers): **Skin absorption:** Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm2/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/ cm2/hr . Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series , the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl; ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight. **Metabolism:** The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo.* The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid . Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers. The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur **Acute toxicity:** Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Continued...

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.
Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol
ethers in this category is required to produce systemic toxicity
In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation . Due to a high incidence of similar spontaneous changes
in normal New Zealand White rabbits , the testicular effects were considered not to be related to treatment . Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered
unremarkable.
A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day . In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or
haemolysed blood in the stomach These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats
In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity
Mutagenicity: Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.
Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater that the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).
Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with . 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.
Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:
EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)
EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41
EO > 15-20 gives Harmful (Xn) with R22-41
>20 EO is not classified (CESIO 2000)
Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin) .
AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO_2).Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO_2)). The metabolism of C12 AE yields PEG, carboxylic acids, and CO_2 as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been

Continued...

	established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations. AEs are not contact sensitisers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust. In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.
N-METHYL-2-PYRROLIDONE	for N-methyl-2-pyrrolidone (NMP): **Acute toxicity:** In rats, NMP is absorbed rapidly after inhalation, oral, and dermal administration, distributed throughout the organism, and eliminated mainly by hydroxylation to polar compounds, which are excreted via urine. About 80% of the administered dose is excreted as NMP and NMP metabolites within 24 h. A probably dose-dependent yellow coloration of the urine in rodents is observed. The major metabolite is 5-hydroxy-*N*-methyl-2-pyrrolidone. Studies in humans show comparable results. Dermal penetration through human skin has been shown to be very rapid. NMP is rapidly biotransformed by hydroxylation to 5-hydroxy-*N*-methyl-2-pyrrolidone, which is further oxidized to *N*-methylsuccinimide; this intermediate is further hydroxylated to 2-hydroxy-*N*-methylsuccinimide. These metabolites are all colourless. The excreted amounts of NMP metabolites in the urine after inhalation or oral intake represented about 100% and 65% of the administered doses, respectively. NMP has a low potential for skin irritation and a moderate potential for eye irritation in rabbits. Repeated daily doses of 450 mg/kg body weight administered to the skin caused painful and severe haemorrhage and eschar formation in rabbits. These adverse effects have not been seen in workers occupationally exposed to pure NMP, but they have been observed after dermal exposure to NMP used in cleaning processes. No sensitisation potential has been observed. In acute toxicity studies in rodents, NMP showed low toxicity. Uptake of oral, dermal, or inhaled acutely toxic doses causes functional disturbances and depressions in the central nervous system. Local irritation effects were observed in the respiratory tract when NMP was inhaled and in the pyloric and gastrointestinal tracts after oral administration. In humans, there was no irritative effect in the respiratory system after an 8-h exposure to 50 mg/m3. **Repeat dose toxicity:** There is no clear toxicity profile of NMP after multiple administration. In a 28-day dietary study in rats, a compound-related decrease in body weight gain was observed in males at 1234 mg/kg body weight and in females at 2268 mg/kg body weight. Testicular degeneration and atrophy in males and thymic atrophy in females were observed at these dose levels. The no-observed-adverse-effect level (NOAEL) was 429 mg/kg body weight in males and 1548 mg/kg body weight in females. In a 28-day intubation study in rats, a dose-dependent increase in relative liver and kidney weights and a decrease in lymphocyte count in both sexes were observed at 1028 mg/kg body weight. The NOAEL in this study was 514 mg/kg body weight. In another rat study, daily dietary intake for 90 days caused decreased body weights at doses of 433 and 565 mg/kg body weight in males and females, respectively. There were also neurobehavioural effects at these dose levels. The NOAELs in males and females were 169 and 217 mg/kg body weight, respectively. The toxicity profile after exposure to airborne NMP depends strongly on the ratio of vapour to aerosol and on the area of exposure (i.e., head-only or whole-body exposure). Because of higher skin absorption for the aerosol, uptake is higher in animals exposed to aerosol than in those exposed to vapour at similar concentrations. Studies in female rats exposed head only to 1000 mg/m3 showed only minor nasal irritation, but massive mortality and severe effects on major organs were observed when the females were whole-body exposed to the same concentration of coarse droplets at high relative humidity. Several studies in rats following repeated exposure to NMP at concentrations between 100 and 1000 mg/m3 have shown systemic toxicity effects at the lower dose levels. In most of the studies, the effects were not observed after a 4-week observation period. In rats, exposure to 3000 mg NMP/m3 (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight gain, an increase in erythrocytes, haemoglobin, haematocrit, and mean corpuscular volume, decreased absolute testis weight, and cell loss in the germinal epithelium of the testes. The NOAEL was 500 mg/m3. There are no data in humans after repeated-dose exposure.

Continued...

	Carcinogenicity: NMP did not show any clear evidence for carcinogenicity in rats exposed to concentrations up to 400 mg/m3 in a long-term inhalation study. **Genotoxicity:** The mutagenic potential of NMP is weak. Only a slight increase in the number of revertants was observed when tested in a *Salmonella* assay with base-pair substitution strains. NMP has been shown to induce aneuploidy in yeast *Saccharomyces cerevisiae* cells. No investigations regarding mutagenicity in humans were available. **Reproductive toxicity:** In a two-generation reproduction study in rats, whole-body exposure of both males and females to 478 mg/m3 of NMP vapour for 6 h/day, 7 days/week, for a minimum of 100 days (pre-mating, mating, gestation, and lactation periods) resulted in a 7% decrease in fetal weight in the F1 offspring. A 4-11% transient, non-dose-dependent decrease was observed in the average pup weight at all exposure levels tested (41, 206, and 478 mg/m3). **Developmental toxicity:** When NMP was administered dermally, developmental toxicity was registered in rats at 750 mg/kg body weight. The observed effects were increased preimplantation losses, decreased fetal weights, and delayed ossification. The NOAEL for both developmental effects and maternal toxicity (decreased body weight gain) was 237 mg/kg body weight. Inhalation studies in rats (whole-body exposure) demonstrated developmental toxicity as increased preimplantation loss without significant effect on implantation rate or number of live fetuses at 680 mg/m3 and behavioural developmental toxicity at 622 mg/m3. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m3, and the NOAEL for developmental effects was 360 mg/m3. A tolerable inhalation concentration, 0.3 mg/m3, based on mortality and organ damage, is expected to be protective against any possible reproductive toxicity. Similarly, an oral tolerable intake of 0.6 mg/kg body weight per day, based on a 90-day study, is expected to provide adequate protection against possible reproductive effects. Because of non-existent data on the exposure of the general population and very limited information on occupational exposure, no meaningful risk characterisation can be performed
DIURON	Note: Equivocal animal tumorigenic agent by RTECS criteria. NOTE: This substance may contain impurities (tetrachlorazobenzene and tetrachloroazoxybenzene). Maximum impurity levels are proscribed under various jurisdictions ADI: 0.006 mg/kg/day NOEL: 0.625 mg/kg/day
CARBENDAZIM	for carbendazim: Benomyl (a precursor to carbendazim) causes dermal sensitization in humans. Benomyl and carbendazim represent a very low risk for acute poisoning in humans. In animal systems, carbendazim is metabolized to (5-hydroxy- 1H-benzimidazol-2-yl)-carbamate (5-HBC) and other polar metabolites, which are rapidly excreted. Carbendazim has not been observed to accumulate in any biological system. Carbendazim has low acute toxicity. The LD50 values range from > 2000 to 15 000 mg/kg in a wide variety of test animals and routes of administration. However, significant adverse reproductive effects have been noted following a single exposure Carbendazim is well absorbed (80-85%) after oral exposure but much less so by dermal exposure. Absorbed carbendazim is metabolised into many compounds within the organism. The main metabolites are 5-HBC and 5,6-HOBC-N-oxides. The tissue distribution of carbendazim showed no bioconcentration. In the rat, the highest concentration after oral carbendazim administration (< 1% of the dose) occurred in the liver. It was distributed as carbendazim in the mitochondria, 5-HBC in the cytostol, and 2-aminobenzimidazole (2-AB) in the microsomes. Carbendazim is excreted in the urine and faeces within 72 h after oral dosing in rats. In rats and mice, high doses of carbendazim, both in the diet and by gavage, affect certain liver microsomal enzymes. **Short-term exposure** Dietary administration of carbendazim for up to 90 days produced slight effects on liver weight in female rats exposed to 360 mg/kg body weight per day. In a 90-day gavage study in the rat, the NOEL was 16 mg/kg per day based on hepatotoxicity. Short-term feeding studies on dogs were not adequate for establishing a NOEL. A 10-day dermal study in the rabbit revealed no systemic toxicity at the only dose tested (200 mg/kg). **Long-term exposure** Male and female rats fed 2500 mg/kg diet showed reduced erythrocyte count and haemoglobin and haematocrit values. No liver-related toxicity was noted. Male rats fed 2500 mg/kg diet or more presented a marginal increase in diffuse testicular atrophy and prostatitis. The NOEL in the rat is 500 mg/kg diet. Male and female mice fed 5000 mg/kg diet showed increased absolute liver weight. There was also significant centrilobular hypertrophy, necrosis and swelling of the liver in male mice fed 1500 mg/kg diet. **Reproduction, embryotoxicity and teratogenicity** Carbendazim was without adverse effects on reproduction when it was fed to rats in a three-generation reproduction study at levels up to and including 500 mg/kg diet. Male fertility was depressed in rats when carbendazim (200 mg/kg per day) was administered by gavage for 85 days. A dose of 50 mg/kg body weight per day in this study caused a significant decrease in epididymal sperm count. Following a single oral dose to rats, histological examination revealed early (0-2 days) disruption of spermatogenesis with occlusion of efferent ducts and increased testicular weights at 100 mg/kg body weight. No effect was observed at 50 mg/kg in this single dose study. These effects persisted until day 70 in rats treated with 400 mg/kg. Carbendazim caused an increase in malformations and anomalies in rats when administered at daily dose levels greater than 10 mg/kg on days 7-16 of gestation. There was a slightly decreased rate of implantation in rabbits administered 20 and 125 mg/kg per day on days 7-19 of gestation and an increased incidence of resorption at 125 mg/kg per day. Maternal toxicity was observed at 20 mg/kg per day and 125 mg/kg per day in the rat and rabbit, respectively. In rats there was a significant increase in foetal malformations at 90 mg/kg per day. These consisted primarily of hydrocephaly, microphthalmia, anophthalmia, malformed scapulea and axial skeletal malformations

Continued...

	(vertebral, rib and sternebral fusions, exencephaly, hemivertebrae and rib hyperplasia). However, in the rabbit there were no significant malformations. **Mutagenicity and related end-points** Assays in mammalian and non-mammalian systems *in vitro* and *in vivo* and in somatic cells as well as in germ cells show that carbendazim does not interact with DNA, induce point mutation or cause germ cell mutation. Carbendazim does, however, cause numerical chromosome aberrations (aneuploidy and/or polyploidy) in experimental systems, both *in vitro* and *in vivo.* **Carcinogenicity:** Benomyl and its decomposition product carbendazim feeding resulted in an increase in the incidence of hepatocellular tumours in CD-1 and SPF Swiss mice. A carcinogenicity study of carbendazim using CD-1 mice showed a statistically significant dose-related increase in the incidence of hepatocellular neoplasia in females. There was also a statistically significant increase in the mid-dose (1500 mg/kg diet) males, but not in the high-dose males because of a high mortality rate. A carcinogenicity study of carbendazim in a genetically related mouse strain, SPF mice (Swiss random strain) at doses of 0, 150, 300 and 1000 mg/kg diet (increased to 5000 mg/kg during the study) showed an increase in the incidence of combined hepatocellular adenomas and carcinomas. Carcinogenicity studies of both benomyl and carbendazim in rats were negative. **Mechanism of toxicity - mode of action** The biological effects of benomyl and carbendazim result from their interaction with cell microtubules. These structures are involved in vital functions such as cell division, which is inhibited by benomyl and carbendazim. Benomyl and carbendazim toxicities in mammals are linked to microtubular dysfunction. Benomyl and carbendazim, as well as other benzimidazole compounds, display species-selective toxicity. This selectivity is, at least in part, explained by the different binding of benomyl and carbendazim to tubulins of target and non-target species ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]*** Intraperitoneal (Rat, adult male) LD50: 7320 mg/kg * Intraperitoneal (Rat, adult female) LD50: 15000 mg/kg * Inhalation LC50 (4 h) for rats, rabbits, guinea pigs or cats no effect with suspension (10 g/l water). * NOEL (2 y) for dogs 300 mg/kg diet, corresponding to 6-7 mg/kg b.w. ADI 0.01 mg/kg b.w. * Toxicity Class WHO III;EPA IV
OCTAMETHYLCYCLOTETRASILOXANE	Does not cause skin sensitization Genotoxicity in vitro : Test Type: Bacterial reverse mutation assay (AMES) Result: negative Remarks: Based on test data Test Type: Mutagenicity (in vitro mammalian cytogenetic test) Result: negative Remarks: Based on test data Test Type: Chromosome aberration test in vitro Result: negative Remarks: Based on test data Test Type: In vitro sister chromatid exchange assay in mammalian cells Result: negative Remarks: Based on test data Test Type: DNA damage and repair, unscheduled DNA synthesis in mammalian cells (in vitro) Result: negative Remarks: Based on test data Genotoxicity in vivo : Test Type: Mammalian erythrocyte micronucleus test (in vivo cytogenetic assay) Species: Rat Application Route: inhalation (vapor) Result: negative Remarks: Based on test data Test Type: Rodent dominant lethal test (germ cell) (in vivo) Species: Rat Application Route: Ingestion Result: negative Remarks: Based on test data Germ cell mutagenicity - Assessment : Animal testing did not show any mutagenic effects Effects on fertility : Test Type: Two-generation reproduction toxicity study Species: Rat, male and female Application Route: inhalation (vapor) Symptoms: Effects on fertility. Remarks: Based on test data Effects on fetal development : Test Type: Prenatal development toxicity study (teratogenicity) Species: Rabbit Application Route: inhalation (vapor) Symptoms: No effects on fetal development. Remarks: Based on test data Reproductive toxicity - Assessment : Some evidence of adverse effects on sexual function and fertility, based on animal experiments. STOT-single exposure May cause damage to organs (Eyes, Central nervous system Routes of exposure: Ingestion Assessment: No significant health effects observed in animals at concentrations of 100 mg/kg bw or less. Routes of exposure: inhalation (vapor) Assessment: No significant health effects observed in animals at concentrations of 1 mg/l/6h/d or less. Routes of exposure: Skin contact Assessment: No significant health effects observed in animals at concentrations of 200 mg/kg bw or less. Results from a 2 year repeated vapor inhalation exposure study to rats of octamethylcyclotetrasiloxane (D4) indicate effects (benign uterine adenomas) in the uterus of female animals. This finding occurred at the highest exposure dose (700 ppm) only. Studies to date have not demonstrated if these effects occur through pathways that are relevant to humans. Repeated exposure in rats to D4 resulted in protoporphyrin accumulation in the liver. Without knowledge of the specific mechanism leading to the protoporphyrin accumulation the relevance of this finding to humans is unknown
SV50 Aqueous Epoxy Part A & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & CRESYL GLYCIDYL ETHER & ISOTHIAZOLINONES, MIXED	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.
SV50 Aqueous Epoxy Part A & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	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

Continued...

	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 - **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.
SV50 Aqueous Epoxy Part A & DIURON	Diuron is absorbed readily through the gut and lungs while uptake through the skin is more limited. It is slightly toxic to mammals but juveniles are more susceptible than adults(18). The oral LD50 in rats is 3-4 g/kg and the dermal LD50 is > 2 g/kg(19). An early study indicated that animals fed protein-deficient diets were considerably more vulnerable to diuron toxicity; rats fed a diet of 3% protein were five times more sensitive to diuron. Exposure to sub-lethal doses of diuron causes formation of methaemoglobin, an abnormal form of the protein haemoglobin which carries oxygen in the blood. Diuron can decrease the number of red blood cells (RBCs), increase the number of abnormally shaped RBCs, and increase the number of white blood cells. Diuron may cause the spleen to become congested due to the increased demand to remove damaged RBCs. Increases in liver size are also observed and are indicative of the extra load placed on this organ, the body's major site of detoxification. Diuron can also cause eye and skin irritation. Diuron contains two significant impurities from the manufacturing process 3,3',4,4'-tetrachloroazobenzene (TCAB) and 3,3',4,4'-tetrachloroazoxybenzene (TCAOB), both potent 'dioxin-like' substances. TCAB levels between 0.15 and 28 ppm have been found in diuron samples tested. TCAOB is present at lower levels. Both TCAB and TCAOB cause chloracne a serious skin disease **Carcinogenicity:** The US Environmental Protection Agency (EPA) has classified diuron as a 'known/likely' carcinogen since 1997 based on the results of two studies. One study on rats indicated that both males and females fed diuron had a higher incidence of bladder cancer than control animals. The male rats in this study

Continued...

	also had a higher incidence of kidney cancer than the control animals. In a study of mice animals with higher exposures had more breast cancer **Mutagenicity:** There is conflicting evidence on whether diuron can cause mutations **Developmental Toxicity:** Rats fed relatively high levels (125 mg/kg/day) of diuron produced offspring with delayed bone formation(26) and other studies indicate that similar levels of diuron reduce birth weight. The US Toxics Release Inventory list diuron as a developmental toxin In mammals, metabolism principally occurs through hydroxylation and dealkylation. **Metabolites**. Breakdown of this compound is similar in animals, plants and soil. The first step is N-demethylation followed by ring cleavage. The main breakdown product of diuron is 3,4-dichloroaniline(3,4-DCA). The oral LD50 of 3,4-DCA in rats is around 60 mg/kg and by the inhalation route the LC50 ranges from 2.8 to 4.7 mg/l/4hrs indicating that 3,4-DCA is considerably more toxic than diuron itself. Dermal and inhalation absorption is rapid leading to formation of methaemoglobin. A marked species difference is dermal toxicity is noted with rabbit considerably more sensitive than rats. No human data is available but by extrapolating from other aromatic amines humans could be considerably more sensitive to methaemoglobin formation than rats. 3,4-DCA should be regarded as a potential respiratory sensitiser. The carcinogenic potential of 3,4-DCA remains uncertain.
SV50 Aqueous Epoxy Part A & N-METHYL-2-PYRROLIDONE & 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.
ISOTHIAZOLINONES, MIXED & DIURON	No significant acute toxicological data identified in literature search.
ISOTHIAZOLINONES, MIXED & OCTAMETHYLCYCLOTETRASILOXANE	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.

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

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

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

SV50 Aqueous Epoxy Part A

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available

bisphenol A/ diglycidyl ether resin, liquid

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
EC50	48	Crustacea	ca.2mg/L	2

cresyl glycidyl ether

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	12.597mg/L	3
LC50	96	Fish	ca.6.5mg/L	2
EC50	48	Crustacea	ca.3.3mg/L	2

Continued...

	EC50	72	Algae or other aquatic plants	ca.5.1mg/L	2

monobutyl ether ethoxylated, propoxylated

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available

N-methyl-2-pyrrolidone

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	464mg/L	1
EC50	48	Crustacea	ca.4897mg/L	1
EC50	72	Algae or other aquatic plants	>500mg/L	2
EC0	24	Crustacea	>1-mg/L	2
NOEC	504	Crustacea	12.5mg/L	2

isothiazolinones, mixed

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	0.129mg/L	2
EC50	48	Crustacea	0.007mg/L	2
EC50	72	Algae or other aquatic plants	0.0063mg/L	2
NOEC	48	Algae or other aquatic plants	0.00049mg/L	2

sodium nitrate

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	1-559mg/L	2
EC50	48	Crustacea	3-581mg/L	2
EC50	96	Algae or other aquatic plants	1181.887mg/L	3
NOEC	2880	Fish	1.6mg/L	4

diuron

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	0.5mg/L	4
EC50	48	Crustacea	1.4mg/L	2
EC50	72	Algae or other aquatic plants	0.00055mg/L	4
BCF	792	Algae or other aquatic plants	0.159mg/L	4
NOEC	336	Algae or other aquatic plants	0.0000005mg/L	4

carbendazim

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	0.007mg/L	4
EC50	48	Crustacea	0.02mg/L	4
EC50	96	Algae or other aquatic plants	3.945mg/L	3
NOEC	480	Crustacea	<0.0031mg/L	4

octamethylcyclotetrasiloxane

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>0.0063mg/L	2
EC50	48	Crustacea	>0.015mg/L	2
EC50	96	Algae or other aquatic plants	>0.022mg/L	2
BCF	120	Fish	0.00053mg/L	4
NOEC	336	Fish	<=0.0044mg/L	4

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.

Continued...

For bisphenol A and related bisphenols:
Environmental fate:
Biodegradability (28 d) 89% - Easily biodegradable
Bioconcentration factor (BCF) 7.8 mg/l
Bisphenol A, its derivatives and analogues, can be released from polymers, resins and certain substances by metabolic products
Substance does not meet the criteria for PBT or vPvB according to Regulation (EC) No 1907/2006, Annex XIII
As an environmental contaminant, bisphenol A interferes with nitrogen fixation at the roots of leguminous plants associated with the bacterial symbiont Sinorhizobium meliloti. Despite a half-life in the soil of only 1-10 days, its ubiquity makes it an important pollutant. According to Environment Canada, "initial assessment shows that at low levels, bisphenol A can harm fish and organisms over time. Studies also indicate that it can currently be found in municipal wastewater." However, a study conducted in the United States found that 91-98% of bisphenol A may be removed from water during treatment at municipal water treatment plants.
Ecotoxicity:
Fish LC50 (96 h): 4.6 mg/l (freshwater fish); 11 mg/l (saltwater fish): NOEC 0.016 mg/l (freshwater fish- 144 d); 0.064 mg/l (saltwater fish 164 d)
Fresh water invertebrates EC50 (48 h): 10.2 mg/l: NOEC 0.025 mg/l - 328 d)
Marine water invertebrate EC50 (96 h): 1.1 mg/l; NOEC 0.17 mg/l (28 d)
Freshwater algae (96 h): 2.73 mg/l
Marine water algae (96 h): 1.1 mg/l
Fresh water plant EC50 (7 d): 20 mg/l: NOEC 7.8 mg/l
In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms.
Among freshwater organisms, fish appear to be the most sensitive species. Evidence of endocrine-related effects in fish, aquatic invertebrates, amphibians and reptiles has been reported at environmentally relevant exposure levels lower than those required for acute toxicity. There is a widespread variation in reported values for endocrine-related effects, but many fall in the range of 1 ug/L to 1 mg/L
A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations.
A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.
Although abundant data are available on the toxicity of bisphenol-A (2,2-bis (4-hydroxydiphenyl)propane;(BPA) A variety of BPs were examined for their acute toxicity against Daphnia magna, mutagenicity, and oestrogenic activity using the Daphtoxkit (Creasel Ltd.), the umu test system, and the yeast two-hybrid system, respectively, in comparison with BPA. BPA was moderately toxic to D. magna (48-h EC50 was 10 mg/l) according to the current U.S. EPA acute toxicity evaluation standard, and it was weakly oestrogenic with 5 orders of magnitude lower activity than that of the natural estrogen 17 beta-oestradiol in the yeast screen, while no mutagenicity was observed. All seven BPs tested here showed moderate to slight acute toxicity, no mutagenicity, and weak oestrogenic activity as well as BPA. Some of the BPs showed considerably higher oestrogenic activity than BPA, and others exhibited much lower activity. Bisphenol S (bis(4-hydroxydiphenyl)sulfone) and bis(4-hydroxyphenyl)sulfide) showed oestrogenic activity.
Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F ([bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem,
Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and 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).
Diuron is a systemic substituted phenylurea herbicide. Diuron is easily taken up from soil solution by the root system of plants and rapidly translocated into stems and leaves by the transpiration system, moving primarily via the xylem. Diuron primarily functions by inhibiting the Hill reaction in photosynthesis, limiting the production of high-energy compounds such as adenosine triphosphate (ATP) used for various metabolic processes.
Air: Diuron is non-volatile, as indicated by its low vapor pressure of 6.90 x10-8 mm Hg (25 C), and a low Henry's law constant of 5.10 x 10-10 atm m3 mol-1. Its low vapor pressure and low Henry's law constant indicate that diuron is unlikely to be dispersed in air over a large area and has a low tendency

Continued...

to volatilise from water or moist soils. Volatilisation is insignificant except when diuron is exposed on the soil surface for several days or weeks under hot, dry conditions.
Water: Diuron's relatively low KOC indicates a relatively low tendency to sorb to soils and sediments, while its hydrolysis and aqueous photolysis half-lives are relatively long. Consequently diuron is both mobile and relatively persistent, and is therefore prone to off-site movement in surface runoff, and migration to ground water.
Microbes are the primary agents in the degradation of diuron in aquatic environments. The aerobic biodegradation pathway for diuron is well established (Figure 1), proceeding by successive demethylation steps to form DCPMU, DCPU [1-(3,4-dichlorophenyl)urea] and DCA (3,4-dichloroaniline). Reductive dechlorination has been observed in anaerobic pond sediments and leads to the formation of the dechlorinated product, 3-(3-chlorophenyl)-1,1-dimethylurea.
3,3',4,4'-Tetrachloroazobenzene (TCAB) and 3,3',4,4'-tetrachloroazoxybenzene (TCAOB) are contaminants derived during the synthesis of 3,4-dichloroaniline and dichloroaniline derivatives pesticides. TCAB and TCAOB are formed by photolysis of 3,4-DCA which is followed by dimerisation of DCA to TCAB. TCAB and TCAOB sorb very strongly to soils and are not likely to leach .
TCAB and TCAOB are isosteric to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), a potent mutagen, teratogen, hepatotoxin, and chloracnegen. TCAB and TCAOB have been shown to bind to TCDD receptors with high affinity and to have similar, but less potent, toxic potential.
The major photoproducts observed in the photolysis of diuron [3-(3,4-dichlorophenyl)-1,1-dimethylurea] in aqueous solution resulted from a heterolytic substitution of chlorine by OH. A wavelength effect was observed: at 254 nm the formation of 3-(4-chloro-3-hydroxyphenyl)-1,1-dimethylurea accounted for more than 90% of the conversion, whereas when the solution was irradiated in 'black light' (85% of photons emitted at 365 nm, about 7% at 344 nm), the major photoproduct was 3-(3-chloro-4-hydroxyphenyl)-1,1-dimethylurea. However photolysis is not generally a principal route of diuron degradation in aqueous systems.
Soil: Diuron is moderately to highly persistent in soils. The commonly reported average field dissipation half-life is 90 days, although such half-lives are typically highly variable. Phytotoxic residues generally dissipate within a season when applied at low selective rates. At higher application rates, residues may persist for more than one year. Microbial degradation is the primary means of diuron dissipation from soil. Photodegradation is not considered a primary dissipation route, but losses can be significant if diuron remains on the soil surface for several days or weeks.
Diuron is mobile in soil. Similar to many other pesticides, diuron sorption is highly correlated with organic matter . Consequently leaching is greatest in low organic matter soils. Other soil conditions that favor diuron leaching include high soil permeability to water, such as in coarse soils.
Biota:
Ecotoxicity:
Mallard Duck LD50 >2000mg/kg
Mallard Duck 8 day LC50 >5000 ppm
Bobwhite Quail 8 day LC50 1730 ppm
Bluegill Sunfish LC50 (96 hrs) 5.9 ppm
Rainbow Trout LC50 (96 hrs) 190 ppm
Sheepshead Minnow LC50 (96 hrs) 6.7 ppm
Daphnia Magna LC50 (48 hrs) 8 ppm
Honeybee LD50 (48 hrs) 145 ug/bee
Oyster Shell EC50 (96 hrs) 4.8 ppm
Plants: Diuron is readily absorbed through the root system of plants and less readily through the leaves and stems. Diuron is translocated rapidly from roots to shoots via the xylem. Little to no diuron moves from the apex downward toward the base of a treated leaf via the phloem. Diuron symptoms of disease are foliar chlorosis concentrated around veins or sometimes interveinal followed by necrosis.
In plants, diuron is metabolized via N-demethylation of the nitrogen atom and hydroxylation at position 2 of the benzene ring. Differential metabolism via N-demethylation may be the basis for diuron selectivity. For example, demethylation is catalysed in cotton by an enzyme called N-demethylase. Diuron was metabolized to conjugates of monomethyldiuron in *Torilis arvenis* and to N-dealkylated derivatives in *Lolium rigidum.*
Mammals. Once diuron is ingested, it is excreted through feces and urine of test animals. In a study, cows fed very low doses of diuron in their diets had small amounts of residues in whole milk. Cattle fed small amounts, accumulated low levels of diuron in fat, muscle, liver, and kidney. Little tissue storage under field conditions is anticipated
Fish and Aquatic Invertebrates. Diuron is toxic to fish and aquatic invertebrates. The LC50 (48 hr) values for diuron range from 4.3 to 42 mg/L in fish, and range from 1 to 2.5 mg/L for aquatic invertebrates. The LC50 (96hr) is 3.5 mg/L for rainbow trout. Therefore diuron is moderately toxic to fish and to aquatic invertebrates
Cellulosic products, including cellulose ethers, generally have a low biodegradation rate and are generally of low toxicity to fish.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
bisphenol A/ diglycidyl ether resin, liquid	HIGH	HIGH
cresyl glycidyl ether	LOW (Half-life = 49 days)	LOW (Half-life = 0.67 days)
N-methyl-2-pyrrolidone	LOW	LOW
sodium nitrate	LOW	LOW
diuron	HIGH	HIGH
carbendazim	HIGH	HIGH
octamethylcyclotetrasiloxane	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
bisphenol A/ diglycidyl ether resin, liquid	LOW (LogKOW = 2.6835)
cresyl glycidyl ether	LOW (LogKOW = 2.1609)

Continued...

N-methyl-2-pyrrolidone	LOW (BCF = 0.16)
sodium nitrate	LOW (LogKOW = 0.209)
diuron	LOW (BCF = 14)
carbendazim	LOW (BCF = 3.5)
octamethylcyclotetrasiloxane	HIGH (BCF = 12400)

Mobility in soil

Ingredient	Mobility
bisphenol A/ diglycidyl ether resin, liquid	LOW (KOC = 51.43)
cresyl glycidyl ether	LOW (KOC = 66.54)
N-methyl-2-pyrrolidone	LOW (KOC = 20.94)
sodium nitrate	LOW (KOC = 14.3)
diuron	LOW (KOC = 136)
carbendazim	LOW (KOC = 175.8)
octamethylcyclotetrasiloxane	LOW (KOC = 17960)

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. ▸ **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 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.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

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

Continued...

Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

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

Sea transport (IMDG-Code / GGVSee)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-A , S-F Special provisions: 274 335 969 Limited Quantities: 5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID(25068-38-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
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) - Index
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
International Air Transport Association (IATA) Dangerous Goods Regulations
International FOSFA List of Banned Immediate Previous Cargoes
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

CRESYL GLYCIDYL ETHER(26447-14-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
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) - Index
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

MONOBUTYL ETHER ETHOXYLATED, PROPOXYLATED(9038-95-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

N-METHYL-2-PYRROLIDONE(872-50-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
GESAMP/EHS Composite List - GESAMP Hazard Profiles
IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk

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

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

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

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
GESAMP/EHS Composite List - GESAMP Hazard Profiles
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

DIURON(330-54-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

CARBENDAZIM(10605-21-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes	International Air Transport Association (IATA) Dangerous Goods Regulations
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International Maritime Dangerous Goods Requirements (IMDG Code)
Australia Inventory of Chemical Substances (AICS)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index	

OCTAMETHYLCYCLOTETRASILOXANE(556-67-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List	IMO IBC Code Chapter 17: Summary of minimum requirements
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes	IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International Air Transport Association (IATA) Dangerous Goods Regulations
Australia Inventory of Chemical Substances (AICS)	International Maritime Dangerous Goods Requirements (IMDG Code)
GESAMP/EHS Composite List - GESAMP Hazard Profiles	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

National Inventory Status

National Inventory	Status
Australia - AICS	No (isothiazolinones, mixed)
Canada - DSL	Yes
Canada - NDSL	No (octamethylcyclotetrasiloxane; bisphenol A/ diglycidyl ether resin, liquid; isothiazolinones, mixed; cresyl glycidyl ether; diuron; carbendazim; sodium nitrate; monobutyl ether ethoxylated, propoxylated; N-methyl-2-pyrrolidone)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (isothiazolinones, mixed; monobutyl ether ethoxylated, propoxylated)
Japan - ENCS	No (isothiazolinones, mixed; monobutyl ether ethoxylated, propoxylated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (isothiazolinones, mixed)
Taiwan - TCSI	Yes
Mexico - INSQ	No (isothiazolinones, mixed; cresyl glycidyl ether)
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Thailand - TECI	No (bisphenol A/ diglycidyl ether resin, liquid; isothiazolinones, mixed; cresyl glycidyl ether; monobutyl ether ethoxylated, propoxylated)
Legend:	*Yes = All declared 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

Revision Date	06/06/2019
Initial Date	12/08/2015

SDS Version Summary

Version	Issue Date	Sections Updated
1.5.1.1.1	06/06/2019	Physical Properties

Other information

Ingredients with multiple cas numbers

Name	CAS No
bisphenol A/ diglycidyl ether resin, liquid	25068-38-6, 25085-99-8
cresyl glycidyl ether	26447-14-3, 2210-79-9, 2180-25-8, 2186-24-5

Continued...

N-methyl-2-pyrrolidone	872-50-4, 26138-58-9
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

Powered by AuthorITe, from Chemwatch.