



# 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

## MG Chemicals UK Limited

Version No: A-2.00

Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

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L.REACH.GB.EN

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

#### 1.1. Product Identifier

Product name	832HD-A
Synonyms	SDS Code: 832HD-Part A; 832HD-25ML, 832HD-50ML, 832HD-400ML, 832HD-1.7L, 832HD-7.4L, 832HD-40L   UFI:S1G0-F0EM-U00P-5VEQ
Other means of identification	Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

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

Relevant identified uses	Epoxy resin for use with hardeners
Uses advised against	Not Applicable

#### 1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	+(44) 1663 362888	+(1) 800-201-8822
Fax	Not Available	+(1) 800-708-9888
Website	Not Available	<a href="http://www.mgchemicals.com">www.mgchemicals.com</a>
Email	sales@mgchemicals.com	Info@mgchemicals.com

#### 1.4. Emergency telephone number

Association / Organisation	Verisk 3E (Access code: 335388)
Emergency telephone numbers	+(44) 20 35147487
Other emergency telephone numbers	+(0) 800 680 0425

### SECTION 2 Hazards identification

#### 2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H411 - Hazardous to the Aquatic Environment Long-Term Hazard Category 2, H315 - Skin Corrosion/Irritation Category 2, H319 - Serious Eye Damage/Eye Irritation Category 2, H317 - Sensitisation (Skin) Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567

#### 2.2. Label elements

Hazard pictogram(s)	
Signal word	Warning

#### Hazard statement(s)

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

#### Supplementary statement(s)

EUH205	Contains epoxy constituents. May produce an allergic reaction.
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## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

## Precautionary statement(s) Prevention

<b>P280</b>	Wear protective gloves, protective clothing, eye protection and face protection.
<b>P261</b>	Avoid breathing mist/vapours/spray.
<b>P273</b>	Avoid release to the environment.
<b>P264</b>	Wash all exposed external body areas thoroughly after handling.
<b>P272</b>	Contaminated work clothing should not be allowed out of the workplace.

## Precautionary statement(s) Response

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

## Precautionary statement(s) Storage

Not Applicable

## Precautionary statement(s) Disposal

<b>P501</b>	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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## 2.3. Other hazards

Cumulative effects may result following exposure\*.

Limited evidence of a carcinogenic effect\*.

Possible respiratory sensitizer\*.

May possibly affect fertility\*.

<b>naphtha petroleum, heavy alkylate</b>	Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors
<b>bisphenol A/ diglycidyl ether resin, liquid</b>	Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors

## SECTION 3 Composition / information on ingredients

## 3.1.Substances

See 'Composition on ingredients' in Section 3.2

## 3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
1.1675-54-3 2.216-823-5 3.603-073-00-2 603-074-00-8 4.Not Available	89	<u>bisphenol A diglycidyl ether</u>	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Sensitisation (Skin) Category 1; H315, H319, H317 [2]	Not Available
1.17557-23-2 2.241-536-7 3.603-094-00-7 4.Not Available	6	<u>neopentyl glycol diglycidyl ether</u>	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1; H315, H317 [2]	Not Available
1.64741-65-7. 2.265-067-2 3.649-275-00-4 4.Not Available	2	<u>naphtha petroleum, heavy alkylate</u> [e]	Flammable Liquids Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Aspiration Hazard Category 1; H226, H336, H304 [1]	Not Available
1.25068-38-6 2.500-033-5 3.603-074-00-8 4.Not Available	1	<u>bisphenol A/ diglycidyl ether resin, liquid</u> [e]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H315, H319, H317, H411 [2]	Not Available
1.1333-86-4 2.215-609-9 435-640-3 422-130-0 3.Not Available 4.Not Available	0.4	<u>carbon black</u>	Carcinogenicity Category 2; H351 [1]	Not Available
1.68609-97-2 2.271-846-8 3.603-103-00-4 4.Not Available	0.3	<u>(C12-14)alkylglycidyl ether</u>	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1; H315, H317 [2]	Not Available

**Legend:**

1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&amp;L; \* EU IOELVs available; [e] Substance identified as having endocrine disrupting properties

## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

**SECTION 4 First aid measures****4.1. Description of first aid measures**

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

**4.2 Most important symptoms and effects, both acute and delayed**

See Section 11

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

Treat symptomatically.

**SECTION 5 Firefighting measures****5.1. Extinguishing media**

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

**5.2. Special hazards arising from the substrate or mixture**

<b>Fire Incompatibility</b>	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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**5.3. Advice for firefighters**

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear full body protective clothing with breathing apparatus.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Combustible.</li> <li>▶ Slight fire hazard when exposed to heat or flame.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> <li>▶ May emit acrid smoke.</li> <li>▶ Mists containing combustible materials may be explosive.</li> </ul> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) aldehydes other pyrolysis products typical of burning organic material.</p>

**SECTION 6 Accidental release measures****6.1. Personal precautions, protective equipment and emergency procedures**

See section 8

**6.2. Environmental precautions**

See section 12

**6.3. Methods and material for containment and cleaning up**

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>· In the event of a spill of a reactive diluent, the focus is on containing the spill to prevent contamination of soil and surface or ground water.</li> <li>· If irritating vapors are present, an approved air-purifying respirator with organic vapor canister is recommended for cleaning up spills and leaks.</li> <li>· For small spills, reactive diluents should be absorbed with sand.</li> </ul> <p>Environmental hazard - contain spillage.</p>
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## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

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

## 6.4. Reference to other sections

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

## SECTION 7 Handling and storage

## 7.1. Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ When handling, <b>DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> </ul>
Fire and explosion protection	See section 5
Other information	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ Store in a cool, dry, well-ventilated area.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>

## 7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> <li>▶ Metal can or drum</li> <li>▶ Packaging as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
Storage incompatibility	<p>In general, uncured epoxy resins have only poor mechanical, chemical and heat resistance properties. However, good properties are obtained by reacting the linear epoxy resin with suitable curatives to form three-dimensional cross-linked thermoset structures. This process is commonly referred to as curing or gelation process. Curing of epoxy resins is an exothermic reaction and in some cases produces sufficient heat to cause thermal degradation if not controlled.</p> <p>Curing may be achieved by reacting an epoxy with itself (homopolymerisation) or by forming a copolymer with polyfunctional curatives or hardeners. In principle, any molecule containing a reactive hydrogen may react with the epoxide groups of the epoxy resin. Common classes of hardeners for epoxy resins include amines, acids, acid anhydrides, phenols, alcohols and thiols. Relative reactivity (lowest first) is approximately in the order: phenol &lt; anhydride &lt; aromatic amine &lt; cycloaliphatic amine &lt; aliphatic amine &lt; thiol.</p> <p>The epoxy curing reaction may be accelerated by addition of small quantities of accelerators. Tertiary amines, carboxylic acids and alcohols (especially phenols) are effective accelerators. Bisphenol A is a highly effective and widely used accelerator, but is now increasingly replaced due to health concerns with this substance.</p> <p>Epoxy resin may be reacted with itself in the presence of an anionic catalyst (a Lewis base such as tertiary amines or imidazoles) or a cationic catalyst (a Lewis acid such as a boron trifluoride complex) to form a cured network. This process is known as catalytic homopolymerisation. The resulting network contains only ether bridges, and exhibits high thermal and chemical resistance, but is brittle and often requires elevated temperature to effect curing, so finds only niche applications industrially. Epoxy homopolymerisation is often used when there is a requirement for UV curing, since cationic UV catalysts may be employed (e.g. for UV coatings).</p> <p>Epoxides:</p> <ul style="list-style-type: none"> <li>▶ are highly reactive with acids, bases, and oxidising and reducing agents.</li> <li>▶ react, possibly violently, with anhydrous metal chlorides, ammonia, amines and group 1 metals.</li> </ul>

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- ▶ may polymerise in the presence of peroxides or heat - polymerisation may be violent
  - ▶ may react, possibly violently, with water in the presence of acids and other catalysts.
- 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
- Reactive diluents are stable under recommended storage conditions, but can decompose at elevated temperatures. In some cases, decomposition can cause pressure build-up in closed systems.
- ▶ Avoid cross contamination between the two liquid parts of product (kit).
  - ▶ If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur.
  - ▶ This excess heat may generate toxic vapour
  - ▶ Avoid reaction with amines, mercaptans, strong acids and oxidising agents

## 7.3. Specific end use(s)

See section 1.2

## SECTION 8 Exposure controls / personal protection

## 8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
bisphenol A diglycidyl ether	Dermal 0.75 mg/kg bw/day (Systemic, Chronic) Inhalation 4.93 mg/m <sup>3</sup> (Systemic, Chronic) Dermal 89.3 µg/kg bw/day (Systemic, Chronic) * Inhalation 0.87 mg/m <sup>3</sup> (Systemic, Chronic) * Oral 0.5 mg/kg bw/day (Systemic, Chronic) *	0.006 mg/L (Water (Fresh)) 0.001 mg/L (Water - Intermittent release) 0.018 mg/L (Water (Marine)) 0.341 mg/kg sediment dw (Sediment (Fresh Water)) 0.034 mg/kg sediment dw (Sediment (Marine)) 0.065 mg/kg soil dw (Soil) 10 mg/L (STP) 11 mg/kg food (Oral)
carbon black	Inhalation 1 mg/m <sup>3</sup> (Systemic, Chronic) Inhalation 0.5 mg/m <sup>3</sup> (Local, Chronic) Inhalation 0.06 mg/m <sup>3</sup> (Systemic, Chronic) *	1 mg/L (Water (Fresh)) 0.1 mg/L (Water - Intermittent release) 10 mg/L (Water (Marine))
(C12-14)alkylglycidyl ether	Dermal 1 mg/kg bw/day (Systemic, Chronic) Inhalation 3.6 mg/m <sup>3</sup> (Systemic, Chronic) Dermal 0.5 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.87 mg/m <sup>3</sup> (Systemic, Chronic) * Oral 0.5 mg/kg bw/day (Systemic, Chronic) *	0.106 mg/L (Water (Fresh)) 0.011 mg/L (Water - Intermittent release) 0.072 mg/L (Water (Marine)) 307.16 mg/kg sediment dw (Sediment (Fresh Water)) 30.72 mg/kg sediment dw (Sediment (Marine)) 1.234 mg/kg soil dw (Soil) 10 mg/L (STP)

\* Values for General Population

## Occupational Exposure Limits (OEL)

## INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m <sup>3</sup>	7 mg/m <sup>3</sup>	Not Available	Not Available

## Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
bisphenol A diglycidyl ether	39 mg/m <sup>3</sup>	430 mg/m <sup>3</sup>	2,600 mg/m <sup>3</sup>
bisphenol A diglycidyl ether	90 mg/m <sup>3</sup>	990 mg/m <sup>3</sup>	5,900 mg/m <sup>3</sup>
bisphenol A/ diglycidyl ether resin, liquid	90 mg/m <sup>3</sup>	990 mg/m <sup>3</sup>	5,900 mg/m <sup>3</sup>
carbon black	9 mg/m <sup>3</sup>	99 mg/m <sup>3</sup>	590 mg/m <sup>3</sup>

Ingredient	Original IDLH	Revised IDLH
bisphenol A diglycidyl ether	Not Available	Not Available
neopentyl glycol diglycidyl ether	Not Available	Not Available
naphtha petroleum, heavy alkylate	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Not Available	Not Available
carbon black	1,750 mg/m <sup>3</sup>	Not Available
(C12-14)alkylglycidyl ether	Not Available	Not Available

## Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
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**Notes:** Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

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## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
bisphenol A diglycidyl ether	E	≤ 0.1 ppm
neopentyl glycol diglycidyl ether	E	≤ 0.1 ppm
bisphenol A/ diglycidyl ether resin, liquid	E	≤ 0.1 ppm
(C12-14)alkylglycidyl ether	E	≤ 0.1 ppm

**Notes:** Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

**MATERIAL DATA**

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

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

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

For epichlorohydrin

Odour Threshold Value: 0.08 ppm

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

Exposure at or below the recommended TLV-TWA is thought to minimise the potential for adverse respiratory, liver, kidney effects. Epichlorohydrin has been implicated as a human skin 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)


NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

**8.2. Exposure controls**

<b>8.2.1. Appropriate engineering controls</b>	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.	
	General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.	
	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	
3: Intermittent, low production.	3: High production, heavy use	
4: Large hood or large air mass in motion	4: Small hood-local control only	
Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.		

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8.2.2. Personal protection	
Eye and face protection	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in 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]</li> </ul>
Skin protection	See Hand protection below
Hands/feet protection	<p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>· frequency and duration of contact,</li> <li>· chemical resistance of glove material,</li> <li>· glove thickness and</li> <li>· dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>· Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>· Excellent when breakthrough time &gt; 480 min</li> <li>· Good when breakthrough time &gt; 20 min</li> <li>· Fair when breakthrough time &lt; 20 min</li> <li>· Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.</li> <li>· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential</li> </ul> <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>When handling liquid-grade epoxy resins wear chemically protective gloves, boots and aprons.</p> <p>The performance, based on breakthrough times, of:</p> <ul style="list-style-type: none"> <li>· Ethyl Vinyl Alcohol (EVAL laminate) is generally excellent</li> <li>· Butyl Rubber ranges from excellent to good</li> <li>· Nitrile Butyl Rubber (NBR) from excellent to fair.</li> <li>· Neoprene from excellent to fair</li> <li>· Polyvinyl (PVC) from excellent to poor</li> </ul> <p>As defined in ASTM F-739-96</p> <ul style="list-style-type: none"> <li>· Excellent breakthrough time &gt; 480 min</li> <li>· Good breakthrough time &gt; 20 min</li> <li>· Fair breakthrough time &lt; 20 min</li> <li>· Poor glove material degradation</li> </ul> <p>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)</p> <ul style="list-style-type: none"> <li>· <b>DO NOT use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin).</b></li> <li>· <b>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.</b></li> </ul> <p>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</p> <ul style="list-style-type: none"> <li>▶ <b>DO NOT use solvent to clean the skin</b></li> </ul>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> </ul>

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▸ Eye wash unit.

### Respiratory protection

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

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

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

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

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

### 8.2.3. Environmental exposure controls

See section 12

## SECTION 9 Physical and chemical properties

### 9.1. Information on basic physical and chemical properties

Appearance	Black		
Physical state	Liquid	Relative density (Water = 1)	1.15
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>235
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	5086.96
Initial boiling point and boiling range (°C)	>150	Molecular weight (g/mol)	Not Available
Flash point (°C)	142	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

### 9.2. Other information

Not Available

## SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> <li>▸ Unstable in the presence of incompatible materials.</li> <li>▸ Product is considered stable.</li> <li>▸ Hazardous polymerisation will not occur.</li> </ul>

Continued...



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10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

## SECTION 11 Toxicological information

## 11.1. Information on toxicological effects

Inhaled	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>In animal testing, exposure to aerosols of some reactive diluents (notably o-cresol glycidyl ether, CAS RN: 2210-79-9) has been reported to affect the adrenal gland, central nervous system, kidney, liver, ovaries, spleen, testes, thymus, and respiratory tract.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>Not normally a hazard due to non-volatile nature of product</p>
Ingestion	<p>Reactive diluents exhibit a range of ingestion hazards. Small amounts swallowed incidental to normal handling operations are not likely to cause injury. However, swallowing larger amounts may cause injury.</p> <p>Male rats exposed to a single oral dose of bisphenol A diglycidyl ether (BADGE) at 750, 1000, and 2000 mg/kg/day showed a significantly increase in the number of immature and maturing sperm on the testis. There were no significant differences with respect to sperm head count, sperm motility, and sperm abnormality in the BADGE treatment groups</p> <p>The material has <b>NOT</b> been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p>
Skin Contact	<p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Bisphenol A diglycidyl ether (BADGE) may produce contact dermatitis characterised by erythema and oedema, with weeping followed by crusting and scaling. A liquid resin with a molecular weight of 350 produced severe skin irritation in rabbits when applied daily for 4 hours over 20 days. Following the initial contact there may be a discrete erythematous lesion, confined to the point of contact, which may persist for 48 hours to 10 days; the erythema may give way to a papular, vesicular rash with scaling.</p> <p>In animals uncured resin produces moderate ante-mortem depression, loss of body weight and diarrhoea. Local irritation, inflammation and death resulting from respiratory system depression are recorded. Higher molecular weight resins generally produce lower toxicity.</p> <p>Skin contact with reactive diluents may cause slight to moderate irritation with local redness. Repeated or prolonged skin contact may cause burns.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> <li>▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▶ 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.</li> </ul> <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
Eye	<p>Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury. Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>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.</p> <p>Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity..</p> <p>Testicular abnormalities (including testicular atrophy with decreased spermatogenic activity) following exposure to glycidyl ethers have been</p>

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

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

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

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

Lesions may develop a brownish colour and scaling occurs frequently. Lower molecular weight species produce sensitisation more readily.

In mice technical grades of bisphenol A diglycidyl ether produced epidermal tumours and a small increase in the incidence kidney tumours in males and of lymphoreticular/ haematopoietic tumours in females. Subcutaneous injection produced a small number of fibrosarcomas in rats. BADGE is listed as an IARC Group 3 carcinogen, meaning it is 'not classifiable as to its carcinogenicity to humans'. Concern has been raised over this possible carcinogenicity because BADGE is used in epoxy resins in the lining of some tin cans for foodstuffs, and unreacted BADGE may end up in the contents of those cans.

For some reactive diluents, prolonged or repeated skin contact may result in absorption of potentially harmful amounts or allergic skin reactions. Exposure to some reactive diluents (notably neopentylglycol diglycidyl ether, CAS RN:17557-23-2) has caused cancer in some animal testing.

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

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

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

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

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

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

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

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

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

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

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

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

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
bisphenol A diglycidyl ether	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 2 mg/24h - SEVERE
	Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>

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		Skin (rabbit): 500 mg - mild
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
neopentyl glycol diglycidyl ether	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 2150 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Oral(Rat) LD50; 4500 mg/kg <sup>[2]</sup>	Skin (human): Sensitiser [Shell]
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
naphtha petroleum, heavy alkylate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[2]</sup>	Not Available
	Inhalation(Rat) LC50; >5.04 mg/l4h <sup>[2]</sup>	
	Oral(Rat) LD50; >7000 mg/kg <sup>[2]</sup>	
bisphenol A/ diglycidyl ether resin, liquid	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >1200 mg/kg <sup>[2]</sup>	Eye (rabbit): 100mg - Mild
	Oral(Mouse) LD50; >500 mg/kg <sup>[2]</sup>	
carbon black	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral(Rat) LD50; >8000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
(C12-14)alkylglycidyl ether	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral(Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): mild [Ciba]
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (guinea pig): sensitiser
		Skin (human): Irritant
		Skin (human): non- sensitiser
		Skin (rabbit): moderate
		Skin : Moderate
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
<b>Legend:</b>	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)	<p>The various members of the bisphenol family produce hormone like effects, seemingly as a result of binding to estrogen receptor-related receptors (ERRs; not to be confused with estrogen receptors)</p> <p>A suspected estrogen-related receptors (ERR) binding agent:</p> <p>Estrogen-related receptors (ERR, oestrogen-related receptors) are so named because of sequence homology with estrogen receptors but do not appear to bind estrogens or other tested steroid hormones. The ERR family have been demonstrated to control energy homeostasis, oxidative metabolism and mitochondrial biogenesis, while effecting mammalian physiology in the heart, brown adipose tissue, white adipose tissue, placenta, macrophages, and demonstrated additional roles in diabetes and cancer.</p> <p>ERRs bind enhancers throughout the genome where they exert effects on gene regulation</p> <p>Although their overall functions remain uncertain, they also share DNA-binding sites, co-regulators, and target genes with the conventional estrogen receptors ERalpha and ERbeta and may function to modulate estrogen signaling pathways.</p> <ul style="list-style-type: none"> <li>ERR-alpha has wide tissue distribution but it is most highly expressed in tissues that preferentially use fatty acids as energy sources such as kidney, heart, brown adipose tissue, cerebellum, intestine, and skeletal muscle. ERRalpha has been detected in normal adrenal cortex tissues, in which its expression is possibly related to adrenal development, with a possible role in fetal adrenal function, in dehydroepiandrosterone (DHEAS) production in adrenarache, and also in steroid production of post-adrenarache/adult life. DHEA and other adrenal androgens such as androstenedione, although relatively weak androgens, are responsible for the androgenic effects of adrenarache, such as early pubic and axillary hair growth, adult-type body odor, increased oiliness of hair and skin, and mild acne.</li> <li>ERR-beta is a nuclear receptor. Its function is unknown; however, a similar protein in mouse plays an essential role in placental development</li> <li>ERR-gamma is a nuclear receptor that behaves as a constitutive activator of transcription. There is evidence that bisphenol A functions as an endocrine disruptor by binding strongly to ERRgamma. BPA as well as its nitrated and chlorinated metabolites seems to binds strongly to ERR-gamma (dissociation constant = 5.5 nM), but not to the estrogen receptor (ER). BPA binding to ERR-gamma preserves its basal constitutive activity. Different expression of ERR-gamma in different parts of the body may account for variations in bisphenol A effects. For instance, ERR-gamma has been found in high concentration in the placenta, explaining reports of high bisphenol A accumulation there</li> </ul>
	<p>Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. The presence of the p-hydroxy group on the benzene rings is thought to be responsible for the oestradiol mimicry.</p> <p>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.</p> <p>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</p>

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employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties.

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

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

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

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

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

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

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

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

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

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

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

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

55badger

**NEOPENTYL GLYCOL  
DIGLYCIDYL ETHER**

\* Anchor SDS]

**NAPHTHA PETROLEUM,  
HEAVY ALKYLATE**

For Low Boiling Point Naphthas (LBPNs):

**Acute toxicity:**

LBPNs generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure

Most LBPNs are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices.

**Sensitisation:**

LBPNs do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies

**Repeat dose toxicity:**

The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPN substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNs in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNs, were considered species- and sex-specific. These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.

Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNs. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3

No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats

No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNs and very few non-cancer chronic toxicity studies were

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identified for other LBPNS. An LOAEC of 200 mg/m<sup>3</sup> was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m<sup>3</sup>, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m<sup>3</sup>). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m<sup>3</sup>.

A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.

**Genotoxicity:**

Although few genotoxicity studies were identified for the site-restricted LBPNS, the genotoxicity of several other LBPNS substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results.

For in vivo genotoxicity tests, LBPNS exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells.

For in vitro genotoxicity studies, LBPNS were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations. LBPNS exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays. Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay. Mixed results were observed for UDS and the mouse lymphoma assay.

While the majority of in vivo genotoxicity results for LBPNS substances are negative, the potential for genotoxicity of LBPNS as a group cannot be discounted based on the mixed in vitro genotoxicity results.

**Carcinogenicity:**

Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effects of human exposure to LBPNS substances.

No inhalation studies assessing the carcinogenicity of the site-restricted LBPNS were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m<sup>3</sup> of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously. Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol. However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPNS group.

Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPNS substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPNS, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans).

Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNS. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals' lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light

straight-run naphtha and naphtha. Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha

or unleaded gasoline were dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.

**Reproductive/ Developmental toxicity:**

No reproductive or developmental toxicity was observed for the majority of LBPNS substances evaluated. Most of these studies were carried out by inhalation exposure in rodents.

NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m<sup>3</sup> (CAS RN 8052-41-3) to 27 687 mg/m<sup>3</sup> (CAS RN 64741-63-5) for the LBPNS group evaluated, and from 7690 mg/m<sup>3</sup> to 27 059 mg/m<sup>3</sup> for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m<sup>3</sup>, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted. For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13.

For most LBPNS, no treatment-related developmental effects were observed by the different routes of exposure. However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m<sup>3</sup> delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

**Low Boiling Point Naphthas [Site-Restricted]**

Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.

The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption is known as the 'hydrocarbon continuum hypothesis', and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.

for petroleum:

## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

	<p>Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline. This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.</p> <p>This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.</p> <p>This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents</p> <p><b>Carcinogenicity:</b> Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.</p> <p><b>Mutagenicity:</b> There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.</p> <p><b>Reproductive Toxicity:</b> Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.</p> <p><b>Human Effects:</b> Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.</p> <p>Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.</p>
BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	Foetotoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity); NOEL (maternal 60 mg/kg)
CARBON BLACK	<p>Inhalation (rat) TCLo: 50 mg/m<sup>3</sup>/6h/90D-1 Nil reported No significant acute toxicological data identified in literature search.</p> <p><b>WARNING:</b> This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A) & BISPHENOL A DIGLYCIDYL ETHER & NEOPENTYL GLYCOL DIGLYCIDYL ETHER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & (C12-14)ALKYLGLYCIDYL ETHER	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A) & BISPHENOL A DIGLYCIDYL ETHER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	<p>In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at &gt;100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg).</p> <p><b>Reproductive and Developmental Toxicity:</b> BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg.</p> <p><b>Carcinogenicity:</b> IARC concluded that 'there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals.' Its overall evaluation was 'Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3).</p> <p>In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997).</p> <p><b>Genotoxicity:</b> 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).</p> <p><b>Immunotoxicity:</b> Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs</p> <p>-</p> <p><b>Consumer exposure</b> to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/kg body weight/day from the 90-day study, and a NOAEL of 15 mg/kg body weight/day (male rats) from the 2-year carcinogenicity study. Both NOAELs are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELs of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs.</p>
832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A) & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	<p>The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics.</p> <p>Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities.</p> <p>Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.</p>

## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

	In vitro cell models were used to evaluate the ability of 22 bisphenols (BPs) to induce or inhibit estrogenic and androgenic activity. BPA, Bisphenol AF (BPAF), bisphenol Z (BPZ), bisphenol C (BPC), tetramethyl bisphenol A (TMBPA), bisphenol S (BPS), bisphenol E (BPE), 4,4-bisphenol F (4,4-BPF), bisphenol AP (BPAP), bisphenol B (BPB), tetrachlorobisphenol A (TCBPA), and benzylparaben (PHBB) induced estrogen receptor (ER)alpha and/or ERbeta-mediated activity. With the exception of BPS, TCBPA, and PHBB, these same BPs were also androgen receptor (AR) antagonists. Only 3 BPs were found to be ER antagonists. Bisphenol P (BPP) selectively inhibited ERbeta-mediated activity and 4-(4-phenylmethoxyphenyl)sulfonylphenol (BPS-MPE) and 2,4-bisphenol S (2,4-BPS) selectively inhibited ERalpha-mediated activity. None of the BPs induced AR-mediated activity.
<b>832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A) &amp; BISPHENOL A DIGLYCIDYL ETHER &amp; NEOPENTYL GLYCOL DIGLYCIDYL ETHER &amp; (C12-14)ALKYLGLYCIDYL ETHER</b>	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.
<b>BISPHENOL A DIGLYCIDYL ETHER &amp; NEOPENTYL GLYCOL DIGLYCIDYL ETHER &amp; (C12-14)ALKYLGLYCIDYL ETHER</b>	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.
<b>BISPHENOL A DIGLYCIDYL ETHER &amp; BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID</b>	The substance is classified by IARC as Group 3: <b>NOT</b> classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.

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

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

## 11.2.1. Endocrine Disruption Properties

Many chemicals may mimic or interfere with the body's hormones, known as the endocrine system. Endocrine disruptors are chemicals that can interfere with endocrine (or hormonal) systems. Endocrine disruptors interfere with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body. Any system in the body controlled by hormones can be derailed by hormone disruptors. Specifically, endocrine disruptors may be associated with the development of learning disabilities, deformations of the body various cancers and sexual development problems. Endocrine disrupting chemicals cause adverse effects in animals. But limited scientific information exists on potential health problems in humans. Because people are typically exposed to multiple endocrine disruptors at the same time, assessing public health effects is difficult.

## SECTION 12 Ecological information

## 12.1. Toxicity

<b>832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	Not Available	Not Available	Not Available	Not Available	Not Available
<b>bisphenol A diglycidyl ether</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	9.4mg/l	2
	LC50	96h	Fish	1.2mg/l	2
	EC50	48h	Crustacea	1.1mg/l	2
	NOEC(ECx)	504h	Crustacea	0.3mg/l	2
<b>neopentyl glycol diglycidyl ether</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	Not Available	Not Available	Not Available	Not Available	Not Available
<b>naphtha petroleum, heavy alkylate</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	NOEC(ECx)	72h	Algae or other aquatic plants	0.1mg/l	1
	EC50	72h	Algae or other aquatic plants	13mg/l	1
<b>bisphenol A/ diglycidyl ether resin, liquid</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	~2mg/l	2
	EC50(ECx)	48h	Crustacea	~2mg/l	2

Continued...

## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

carbon black	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	48h	Crustacea	33.076-41.968mg/l	4
	NOEC(ECx)	24h	Crustacea	3200mg/l	1

(C12-14)alkylglycidyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	48h	Crustacea	6.07mg/l	2
	LC50	96h	Fish	>5000mg/l	2
	EC50	48h	Crustacea	6.07mg/l	2

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

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

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

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

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

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 µg/L to 1 mg/L

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

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

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

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

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

They would not be expected to persist in the environment.

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

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

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

for 1,2-butylene oxide (ethyloxirane):

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

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

Ethyloxirane is hydrolysable, with a half-life of 6.5 days, and biodegradable up to 100% degradation and is not expected to persist in water. A further model-predicted biodegradation



## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

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 (  $t_{1/2\text{water}} : t_{1/2\text{soil}} : t_{1/2\text{sediment}} = 1 : 1 : 4$  ) (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).

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

**12.2. Persistence and degradability**

Ingredient	Persistence: Water/Soil	Persistence: Air
bisphenol A diglycidyl ether	HIGH	HIGH
neopentyl glycol diglycidyl ether	HIGH	HIGH
bisphenol A/ diglycidyl ether resin, liquid	HIGH	HIGH

**12.3. Bioaccumulative potential**

Ingredient	Bioaccumulation
bisphenol A diglycidyl ether	MEDIUM (LogKOW = 3.8446)
neopentyl glycol diglycidyl ether	LOW (LogKOW = 0.2342)
bisphenol A/ diglycidyl ether resin, liquid	LOW (LogKOW = 2.6835)

**12.4. Mobility in soil**

Ingredient	Mobility
bisphenol A diglycidyl ether	LOW (KOC = 1767)
neopentyl glycol diglycidyl ether	LOW (KOC = 10)
bisphenol A/ diglycidyl ether resin, liquid	LOW (KOC = 51.43)

**12.5. Results of PBT and vPvB assessment**

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗
PBT Criteria fulfilled?	No		
vPvB	No		

**12.6. Endocrine Disruption Properties**

The evidence linking adverse effects to endocrine disruptors is more compelling in the environment than it is in humans. Endocrine disruptors profoundly alter reproductive physiology of ecosystems and ultimately impact entire populations. Some endocrine-disrupting chemicals are slow to break-down in the environment. That characteristic makes them potentially hazardous over long periods of time. Some well established adverse effects of endocrine disruptors in various wildlife species include; eggshell-thinning, displayed of characteristics of the opposite sex and impaired reproductive development. Other adverse changes in wildlife species that have been suggested, but not proven include; reproductive abnormalities, immune dysfunction and skeletal deformities.

**12.7. Other adverse effects**

Not Available

**SECTION 13 Disposal considerations****13.1. Waste treatment methods**

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Containers may still present a chemical hazard/ danger when empty.</li> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> </ul> <p>Waste Management</p> <p>Production waste from epoxy resins and resin systems should be treated as hazardous waste in accordance with National regulations. Fire retarded resins containing halogenated compounds should also be treated as special waste. Accidental spillage of resins, curing agents and their formulations should be contained and absorbed by special mineral absorbents to prevent them from entering the environment. Contaminated or surplus product should not be washed down the sink, but preferably be fully reacted to form cross-linked solids which is non-hazardous and can be more easily disposed.</p> <p>Finished articles made from fully cured epoxy resins are hard, infusible solids presenting no hazard to the environment. However, finished articles from flame-retarded material containing halogenated resins should be considered hazardous waste, and disposed as required by National laws. Articles made from epoxy resins, like other thermosets, can be recycled by grinding and used as fillers in other products. Another way of disposal and recovery is combustion with energy recovery.</p>
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## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

	<p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>▶ Reduction</li> <li>▶ Reuse</li> <li>▶ Recycling</li> <li>▶ Disposal (if all else fails)</li> </ul> <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> </ul> <p>Removal of bisphenol A (BPA) from aqueous solutions was accomplished by adsorption of enzymatically generated quinone derivatives on chitosan beads. The use of chitosan in the form of beads was found to be more effective because heterogeneous removal of BPA with chitosan beads was much faster than homogeneous removal of BPA with chitosan solutions, and the removal efficiency was enhanced by increasing the amount of chitosan beads dispersed in the BPA solutions and BPA was completely removed by quinone adsorption in the presence of chitosan beads more than 0.10 cm<sup>3</sup>/cm<sup>3</sup>. In addition, a variety of bisphenol derivatives were completely or effectively removed by the procedure constructed in this study, although the enzyme dose or the amount of chitosan beads was further increased as necessary for some of the bisphenol derivatives used.</p> <p>M. Suzuki, and E Musashi J Appl Polym Sci, 118(2):721 - 732; October 2010</p> <ul style="list-style-type: none"> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Management Authority for disposal.</li> <li>▶ Material may be disposed of by controlled burning in an approved incinerator or buried in an approved landfill.</li> <li>▶ Prior to disposal in a landfill the material should be mixed with the other component and reacted to render the material inert.</li> <li>▶ Extreme caution should be taken when heating the resin/curing agent mix.</li> <li>▶ Recycle containers where possible, or dispose of in an authorised landfill.</li> </ul>
<b>Waste treatment options</b>	Not Available
<b>Sewage disposal options</b>	Not Available

## SECTION 14 Transport information

## Labels Required

	<p>NOT REGULATED by Ground ADR Special Provision 375</p> <p>NOT REGULATED by Air IATA Special Provision A197</p> <p>NOT REGULATED by Sea IMDG per 2.10.2.7</p> <p>NOT REGULATED by ADN Special Provision 274 (The provision of 3.1.2.8 apply)</p>
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## Land transport (ADR-RID)

14.1. UN number	3082												
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A diglycidyl ether)												
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>Class</td> <td>9</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	9	Subrisk	Not Applicable								
Class	9												
Subrisk	Not Applicable												
14.4. Packing group	III												
14.5. Environmental hazard	Environmentally hazardous												
14.6. Special precautions for user	<table border="1"> <tr> <td>Hazard identification (Kemler)</td> <td>90</td> </tr> <tr> <td>Classification code</td> <td>M6</td> </tr> <tr> <td>Hazard Label</td> <td>9</td> </tr> <tr> <td>Special provisions</td> <td>274 335 375 601</td> </tr> <tr> <td>Limited quantity</td> <td>5 L</td> </tr> <tr> <td>Tunnel Restriction Code</td> <td>3 (-)</td> </tr> </table>	Hazard identification (Kemler)	90	Classification code	M6	Hazard Label	9	Special provisions	274 335 375 601	Limited quantity	5 L	Tunnel Restriction Code	3 (-)
Hazard identification (Kemler)	90												
Classification code	M6												
Hazard Label	9												
Special provisions	274 335 375 601												
Limited quantity	5 L												
Tunnel Restriction Code	3 (-)												

## Air transport (ICAO-IATA / DGR)

14.1. UN number	3082						
14.2. UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains bisphenol A diglycidyl ether)						
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>ICAO/IATA Class</td> <td>9</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>9L</td> </tr> </table>	ICAO/IATA Class	9	ICAO / IATA Subrisk	Not Applicable	ERG Code	9L
ICAO/IATA Class	9						
ICAO / IATA Subrisk	Not Applicable						
ERG Code	9L						
14.4. Packing group	III						
14.5. Environmental hazard	Environmentally hazardous						
14.6. Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>A97 A158 A197 A215</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>964</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>450 L</td> </tr> </table>	Special provisions	A97 A158 A197 A215	Cargo Only Packing Instructions	964	Cargo Only Maximum Qty / Pack	450 L
Special provisions	A97 A158 A197 A215						
Cargo Only Packing Instructions	964						
Cargo Only Maximum Qty / Pack	450 L						

## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

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)

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

## Inland waterways transport (ADN)

14.1. UN number	3082	
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A diglycidyl ether)	
14.3. Transport hazard class(es)	9	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Classification code	M6
	Special provisions	274; 335; 375; 601
	Limited quantity	5 L
	Equipment required	PP
	Fire cones number	0

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

Not Applicable

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

Product name	Group
bisphenol A diglycidyl ether	Not Available
neopentyl glycol diglycidyl ether	Not Available
naphtha petroleum, heavy alkylate	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Not Available
carbon black	Not Available
(C12-14)alkylglycidyl ether	Not Available

## 14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
bisphenol A diglycidyl ether	Not Available
neopentyl glycol diglycidyl ether	Not Available
naphtha petroleum, heavy alkylate	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Not Available
carbon black	Not Available
(C12-14)alkylglycidyl ether	Not Available

## SECTION 15 Regulatory information

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

bisphenol A diglycidyl ether is found on the following regulatory lists

Continued...

## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

Chemical Footprint Project - Chemicals of High Concern List  
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances  
Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)  
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI  
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**neopentyl glycol diglycidyl ether is found on the following regulatory lists**

Chemical Footprint Project - Chemicals of High Concern List  
Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)  
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

**naphtha petroleum, heavy alkylate is found on the following regulatory lists**

Chemical Footprint Project - Chemicals of High Concern List  
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles  
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 2) Carcinogens: category 1B (Table 3.1)/category 2 (Table 3.2)  
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 4) Mutagens: category 1B (Table 3.1)/category 2 (Table 3.2)

Europe EC Inventory  
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)  
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

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

Chemical Footprint Project - Chemicals of High Concern List  
Europe EC Inventory

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

**carbon black is found on the following regulatory lists**

Chemical Footprint Project - Chemicals of High Concern List  
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances  
Europe EC Inventory  
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs  
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans  
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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

Chemical Footprint Project - Chemicals of High Concern List  
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances  
Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)  
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

**15.2. Chemical safety assessment**

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (bisphenol A diglycidyl ether; neopentyl glycol diglycidyl ether; naphtha petroleum, heavy alkylate; bisphenol A/ diglycidyl ether resin, liquid; carbon black; (C12-14)alkylglycidyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (naphtha petroleum, heavy alkylate; (C12-14)alkylglycidyl ether)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (bisphenol A diglycidyl ether; neopentyl glycol diglycidyl ether; (C12-14)alkylglycidyl ether)
Vietnam - NCI	Yes
Russia - FBEPH	No (neopentyl glycol diglycidyl ether; naphtha petroleum, heavy alkylate)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

**SECTION 16 Other information**

Revision Date	04/10/2021
Initial Date	09/05/2019

**Full text Risk and Hazard codes**

## 832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)

<b>H226</b>	Flammable liquid and vapour.
<b>H304</b>	May be fatal if swallowed and enters airways.
<b>H336</b>	May cause drowsiness or dizziness.
<b>H351</b>	Suspected of causing cancer.

**Other information**

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

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered. For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

**Definitions and abbreviations**

PC—TWA: Permissible Concentration-Time Weighted Average

PC—STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit.

IDLH: Immediately Dangerous to Life or Health Concentrations

ES: Exposure Standard

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

AIIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory

NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act

TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

**Reason For Change**

A-2.00 - Update to ingredients and added UFI number