834FRB Flame Retardant Epoxy (Part A)

MG Chemicals UK Limited

Version No: **A-1.01**
Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

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L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	834FRB-A
Synonyms	SDS Code: 834FRB-Part A; 834FRB-375ML, 834FRB-3L, 834FRB-60L
Other means of identification	Flame Retardant Epoxy (Part A)

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

Relevant identified uses	resin for use with epoxy hardener
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	www.mgchemicals.com
Email	sales@mgchemicals.com	Info@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	**Verisk 3E (Access code: 335388)**	**Not Available**
Emergency telephone numbers	+(44) 20 35147487	Not Available
Other emergency telephone numbers	+(0) 800 680 0425	Not Available

SECTION 2 HAZARDS IDENTIFICATION

2.1.

Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	H315 - Skin Corrosion/Irritation Category 2, H319 - Eye Irritation Category 2, H317 - Skin Sensitizer Category 1, H351 - Carcinogenicity Category 2, H411 - Chronic Aquatic Hazard Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

2.2. Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.
H411	Toxic to aquatic life with long lasting effects.

Supplementary statement(s)

Continued...

Not Applicable

Precautionary statement(s) Prevention

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

Precautionary statement(s) Response

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

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

2.3. Other hazards

antimony trioxide	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)

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	Classification according to regulation (EC) No 1272/2008 [CLP]
1.28064-14-4 2.Not Available 3.Not Available 4.Not Available	60	bisphenol F glycidyl ether/ formaldehyde copolymer	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Chronic Aquatic Hazard Category 2, Skin Sensitizer Category 1; H315, H319, H411, H317, EUH205, EUH019 [1]
1.1309-64-4 2.215-175-0 3.051-005-00-X 4.01-2119475613-35-XXXX|01-2120763584-46-XXXX	8	antimony trioxide	Carcinogenicity Category 2; H351 [2]
1.68609-97-2 2.271-846-8 3.603-103-00-4 4.01-2119485289-22-XXXX	6	(C12-14)alkylglycidyl ether	Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H317, H315 [2]
1.25068-38-6 2.500-033-5 3.603-074-00-8 4.01-2119456619-26-XXXX	6	bisphenol A/ diglycidyl ether resin, liquid	Eye Irritation Category 2, Chronic Aquatic Hazard Category 2, Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H319, H411, H317, H315 [2]
1.64741-65-7. 2.265-067-2 3.649-275-00-4 4.01-2120009436-62-XXXX	1	naphtha petroleum, heavy alkylate	Flammable Liquid Category 3, Aspiration Hazard Category 1, Specific target organ toxicity - single exposure Category 3 (narcotic effects); H226, H304, H336 [1]
1.1333-86-4 2.215-609-9 3.Not Available 4.01-2119384822-32-XXXX|01-2120767622-50-XXXX|01-0000016864-62-XXXX	0.6	carbon black	Carcinogenicity Category 2; H351 [1]
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available*		

SECTION 4 FIRST AID MEASURES

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4.1. Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

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.

- Chelation with British Anti-Lewisite (BAL) for serious antimony exposures should be employed.
- Dialyse as needed. The role of exchange diffusion is not clear.
- Be sure to monitor for dysrhythmias.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

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

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

5.3. Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) aldehydes other pyrolysis products typical of burning organic material.

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

Minor Spills	Environmental hazard - contain spillage. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.

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Major Spills

Environmental hazard - contain spillage.
Chemical Class: phenols and cresols
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
wood fiber - pillow	1	throw	pitchfork	R, P, DGC, RT
foamed glass - pillow	2	shovel	shovel	R, W, P, DGC
sorbent clay - particulate	2	shovel	shovel	R, I, P
wood fibre - particulate	3	shovel	shovel	R, W, P, DGC
LAND SPILL - MEDIUM				
cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	R, SS, DGC
wood fiber - particulate	4	blower	skiploader	R, W, P, DGC
expanded moneral - particulate	4	blower	skiploader	R, I, W, P, DGC

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988
Moderate hazard.

- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- No smoking, naked lights or ignition sources.
- Increase ventilation.
- Stop leak if safe to do so.
- Contain spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

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

- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- **DO NOT** enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights or ignition sources.
- Avoid contact with incompatible materials.
- When handling, **DO NOT** eat, drink or smoke.
- Keep containers securely sealed when not in use.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- Use good occupational work practice.
- Observe manufacturer's storage and handling recommendations contained within this SDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
- **DO NOT** allow clothing wet with material to stay in contact with skin

Fire and explosion protection

See section 5

Other information

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

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7.2. Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ 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 ▸ Phenols are incompatible with strong reducing substances such as hydrides, nitrides, alkali metals, and sulfides. ▸ Avoid use of aluminium, copper and brass alloys in storage and process equipment. ▸ Heat is generated by the acid-base reaction between phenols and bases. ▸ Phenols are sulfonated very readily (for example, by concentrated sulfuric acid at room temperature), these reactions generate heat. ▸ Phenols are nitrated very rapidly, even by dilute nitric acid. ▸ Nitrated phenols often explode when heated. Many of them form metal salts that tend toward detonation by rather mild shock. 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

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	antimony trioxide	Antimony and compounds except stibine (as Sb)	0.5 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m3	7 mg/m3	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
bisphenol F glycidyl ether/ formaldehyde copolymer	Phenol, polymer with formaldehyde, oxiranylmethyl ether	30 mg/m3	330 mg/m3	2,000 mg/m3
antimony trioxide	Antimony oxide	1.8 mg/m3	16 mg/m3	96 mg/m3
bisphenol A/ diglycidyl ether resin, liquid	Epoxy resin includes EPON 1001, 1007, 820, ERL-2795	90 mg/m3	990 mg/m3	5,900 mg/m3
carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
bisphenol F glycidyl ether/ formaldehyde copolymer	Not Available	Not Available
antimony trioxide	50 mg/m3	Not Available
(C12-14)alkylglycidyl ether	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Not Available	Not Available
naphtha petroleum, heavy alkylate	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available

MATERIAL DATA

The wide-ranging effects of antimony compounds have made it difficult to recommend an exposure standard which characterises the toxicology of these substances. One criteria, reflecting the irritant properties of antimony pentachloride, produced a calculated value of 5.0 mg/m3 (as antimony), which on the basis of experience was felt to be too high but did act as an 'out-rider'. The present value reflects this thinking.

8.2. Exposure controls

8.2.1. Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: 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

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'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.

8.2.2. Personal protection

Eye and face protection

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

Skin protection

See Hand protection below

Hands/feet protection

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and.has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

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

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

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data

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	should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. When handling liquid-grade epoxy resins wear chemically protective gloves , boots and aprons. The performance, based on breakthrough times ,of: · Ethyl Vinyl Alcohol (EVAL laminate) is generally excellent · Butyl Rubber ranges from excellent to good · Nitrile Butyl Rubber (NBR) from excellent to fair. · Neoprene from excellent to fair · Polyvinyl (PVC) from excellent to poor As defined in ASTM F-739-96 · Excellent breakthrough time > 480 min · Good breakthrough time > 20 min · Fair breakthrough time < 20 min · Poor glove material degradation Gloves should be tested against each resin system prior to making a selection of the most suitable type. Systems include both the resin and any hardener, individually and collectively) · **DO NOT** use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin). · **DO NOT** use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use. Replacement time should be considered when selecting the most appropriate glove. It may be more effective to select a glove with lower chemical resistance but which is replaced frequently than to select a more resistant glove which is reused many times
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C. apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class 1	-
up to 50	1000	-	A-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	A-2
up to 100	10000	-	A-3
100+		-	Airline**

* - Continuous Flow
** - Continuous-flow or positive pressure demand.
A(All classes) = Organic vapours, B AUS or B1 = Acid gases, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 deg C)

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)**	1.39
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	>465
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	>150	**Viscosity (cSt)**	1150
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available

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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	Partly miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2. Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
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	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. Inhalation of antimony and its compounds may produce respiratory and gastrointestinal tract discomfort with sore throat, shallow respiration, coughing, headaches, breathing difficulties, dizziness, weight loss, gingivitis, anaemia, eosinophilia and enzyme inhibition. Inflammation of the upper and lower respiratory tract may occur. Pulmonary congestion and oedema may also occur. Other symptoms include rhinitis, eye irritation, vomiting and diarrhoea, weight loss, dysomnia, hair loss and haematological disorders. Death due to circulatory failure has been described, with pathology showing acute congestion of the heart (myocardial failure), liver and kidneys.
Ingestion	The material has **NOT** been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. Ingestion of antimony compounds may produce violent irritation of the nose, throat, stomach and gastrointestinal tract, vomiting, purging with bloody stools, slow shallow respiration, pulmonary oedema, convulsions, loss of consciousness, coma, and death due to circulatory or respiratory failure. Early signs of antimony intoxication include: fatigue, muscle weakness, myopathy, nausea, low back pain, headache, and metallic taste. Later symptoms include blood disorders (haemolytic anemia, myoglobinuria, haematuria) and renal failure. The substance may cause cough, salivation, nausea, and diarrhoea. It may also cause dizziness, laryngitis, anaemia, muscular and neuralgic pains. Other symptoms of overexposure may include tightness of the chest, pain, swelling of the cervical glands, pustular eruptions ('antimony spots'), particularly on the scrotum, difficult urination, sexual disorders, nervousness, loss of sleep, leukopenia, unconsciousness and death. [NIOSHTIC] The minimum lethal dose in man, of antimony, is 130 mg (although 15000 mg has been survived). Antimony is a strong irritant and emetic but the emetic dose (30 mg by mouth) is dangerously high if vomiting fails to occur. Trivalent compounds are generally more lethal than pentavalent derivatives. The trivalent antimony compounds are cardiotoxic. The insoluble salts however are less likely to produce significant toxic effects. Antimony salts are poorly absorbed from the gastrointestinal tract. Most trivalent compounds of antimony are slowly excreted in the urine and bile. Antimony compounds are used medicinally, but therapeutic dose is close to the toxic dose. Antimony poisoning closely parallels arsenic poisoning although vomiting is probably more prominent as absorption from the gastrointestinal tract is generally lower. Like arsenic, antimony has a high affinity for sulfhydryl groups on many enzymes. Antimony is conjugated with glutathione and excreted in urine and feces. Therefore, excessive exposure to antimony has the potential to deplete intracellular glutathione pools. Temporary changes in heart rhythm occurs amongst humans while poisoned animals exhibit severe heart damage. Periodic medical examinations covering lungs, skin, nervous system, heart and gastro-intestinal tract are recommended for occupationally exposed workers. [ILO Encyclopedia]
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition 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. Skin contact with antimony compounds may result in redness and severe irritation with the formation of itchy papules, pustules, skin lesions/ small septic blisters (antimony spots) within a few hours. Rhinitis may also result from dermal contact. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

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Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. 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. All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether. A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not *n*-butyl glycidyl ether, induced morphological transformation in mammalian cells *in vitro*. *n*-Butyl glycidyl ether induced micronuclei in mice *in vivo* following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations *in vivo* or chromosomal aberrations in animal cells *in vitro*. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in *Drosophila*. The glycidyl ethers were generally mutagenic to bacteria Repeated or prolonged exposure to antimony and its compounds may produce stomatitis, dry throat, metallic taste, gingivitis, septal and laryngeal perforation, laryngitis, headache, dyspnea, indigestion, nausea, vomiting, diarrhoea, anorexia, anaemia, weight loss, pain and chest tightness, sleeplessness, muscular pain and weakness, dizziness, pharyngitis,tracheitis, bronchitis, pneumonitis, benign pneumoconiosis (with obstructive lung disease and emphysema) and haematological disorders. Degenerative changes of the liver and kidney may occur. Symptoms can be variable, and may including fatigue, myopathy (muscle aches and inflammation), hypotension, angina and immune dysregulation and hypertrophy of splenic follicles. Antimony's deposition in body tissues and its detrimental effects depend upon the oxidation state of the element. The trivalent antimony compounds are cardiotoxic. Trivalent antimony affects liver functions, impairs enzymes, and may interfere with sulfur chemistry. If antimony impairs phosphofructokinase (PFK), then purine metabolism may be disrupted, resulting in elevated blood and/or urine levels of hypoxanthine, uric acid and possibly ammonia. Pentavalent antimony deposits in bone, kidney, and in organs of the endocrine system. Chronic exposure to antimony compounds may result in itchiness, papules and pustules around sweat and sebaceous glands ('antimony spots'), but rarely around the face, and dermatitis. Collapse and sudden death due to anaphylactic-type reactions have occurred. Therapeutic doses given intravenously cause nausea, vomiting, cough and abdominal pain and diarrhoea. Other side-effects include anorexia, chest, muscle and joint pains, pruritus, skin rashes, dizziness and oedema. Renal and hepatic damage occur rarely and haemolytic anaemia has been reported. Continuous treatment with small doses of antimony may give rise to subacute poisoning similar to chronic arsenic poisoning. Smelter workers often show skin rashes on the forearms and thighs resembling chicken pox pustules. Workers exposed to inorganic antimony compounds show a benign pneumoconiosis and obstructive lung disease - these are probably non-specific. Woman appear to more susceptible to systemic effects following exposure. Antimony crosses the placenta, is present in amniotic fluids, and is excreted in breast milk. There are suggestions that exposure may produce an increased incidence of spontaneous late abortions, premature births, and gynecological problems among female antimony smelter workers. An excess of deaths from lung cancer has been reported in smelter workers with more than 7 years exposure to relatively high levels of dust and fume. Animal studies demonstrate that the dust may produce pathological changes in cardiac muscle and may produce an interstitial pneumonitis and endogenous pneumonia. One animal study has also suggested that inhalation of the dust by rats induced a significantly increased incidence of carcinogenic tumours of the lungs and thorax. Increased chromosome defects occur when human lymphocytes are incubated with a soluble antimony salt. The inhalation data suggests that the myocardium is a target of antimony toxicity. It is possible that antimony affects circulating glucose by interfering with enzymes of the glycogenolysis and gluconeogenesis pathways. The mechanism of action of antimony remains unclear. However, some studies suggest that antimony combines with sulfhydryl groups including those in several enzymes important for tissue respiration Bisphenol F, bisphenol A, fluorine-containing bisphenol A (bisphenol AF), and other diphenylalkanes were found to be oestrogenic in a bioassay with MCF7 human breast cancer cells in culture Bisphenol F (4,4'-dihydroxydiphenylmethane) has been reported to exhibit oestrogen agonistic properties in the uterotrophic assay. Bisphenol F (BPF) is present in the environment and as a contaminant of food. Humans may, therefore, be exposed to BP. BPF has been shown to have genotoxic and endocrine-disruptor properties in a human hepatoma cell line (HepG2), which is a model system for studies of xenobiotic toxicity. BPF was largely metabolised into the corresponding sulfate by the HepG2 cell line. BPF was metabolised into both sulfate and glucuronide by human hepatocytes, but with differences between individuals. The metabolism of BPF in both HepG2 cells and human hepatocytes suggests the existence of a detoxification pathway Bisphenol F was orally administered at doses 0, 20, 100 and 500 mg/kg per day for at least 28 days, but no clear endocrine-mediated changes were detected, and it was concluded to have no endocrine-mediated effects in young adult rats. On the other hand, the main effect of bisphenol F was concluded to be liver toxicity based on clinical biochemical parameters and liver weight, but without histopathological changes. The no-observed-effect level for bisphenol F is concluded to be under 20 mg/kg per day since decreased body weight accompanied by decreased serum total cholesterol, glucose, and albumin values were observed in the female rats given 20 mg/kg per day or higher doses of bisphenol F. Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone.. Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned or are under review. A 2009 study on Chinese workers in bisphenol A factories found that workers were four times more likely to report erectile dysfunction, reduced sexual desire and overall dissatisfaction with their sex life than workers with no heightened bisphenol A exposure. Bisphenol A workers were also seven times more likely to have ejaculation difficulties. They were also more likely to report reduced sexual function within one year of beginning employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties. Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/ litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades' One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials' One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood. A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells.[whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes. Bisphenol A is the isopropyl adduct of 4,4'-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called 'cytostatic hormones'. Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown

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	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).

834FRB Flame Retardant Epoxy (Part A)

TOXICITY	IRRITATION
Not Available	Not Available

bisphenol F glycidyl ether/ formaldehyde copolymer

TOXICITY	IRRITATION
dermal (rat) LD50: 4000 mg/kg[2]	Eyes * (-) (-) Slight irritant
Oral (rat) LD50: 4000 mg/kg[2]	Skin * (-) (-) Slight irritant

antimony trioxide

TOXICITY	IRRITATION
Oral (rat) LD50: >34000 mg/kg[2]	Not Available

(C12-14)alkylglycidyl ether

TOXICITY	IRRITATION
Oral (rat) LD50: >10000 mg/kg[2]	Eye (rabbit): mild [Ciba]
	Skin (guinea pig): sensitiser
	Skin (human): Irritant
	Skin (human): non- sensitiser
	Skin (rabbit): moderate
	Skin : Moderate

bisphenol A/ diglycidyl ether resin, liquid

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

naphtha petroleum, heavy alkylate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
Inhalation (rat) LC50: >3.83 mg/l/4H[2]	
Oral (rat) LD50: >7000 mg/kg[2]	

carbon black

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Not Available
Oral (rat) LD50: >15400 mg/kg[2]	

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

834FRB Flame Retardant Epoxy (Part A)

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

ANTIMONY TRIOXIDE

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

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	dyspnea, cough and mucus production. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. (intermittent) [CCINFO] Reproductive effector
BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg). **Reproductive and Developmental Toxicity**: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg. **Carcinogenicity:** IARC concluded that 'there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals.' Its overall evaluation was 'Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3). In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997). **Genotoxicity**: In S. typhimurium strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg). **Immunotoxicity:** Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs - **Consumer exposure** to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/ kg/body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weigh/day (male rats) from the 2-year carcinogenicity study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs. Foetoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity; NOEL (maternal 60 mg/kg
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 identified for other LBPNs. An LOAEC of 200 mg/m3 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/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3 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 LBPN 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.

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While the majority of in vivo genotoxicity results for LBPN 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 effect

s of human exposure to LBPN 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/m3 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 LBPN group.

Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPN 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 LBPN, 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 was 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 LBPN 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/m3 (CAS RN 8052-41-3) to 27 687 mg/m3 (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m3 to 27 059 mg/m3 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/m3, 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/m3 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 that 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:

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.

This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.

This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents

Carcinogenicity: 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.

Mutagenicity: 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.

Reproductive Toxicity: 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.

Human Effects: 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.

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.

Continued...

CARBON BLACK	No significant acute toxicological data identified in literature search. Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported
834FRB Flame Retardant Epoxy (Part A) & BISPHENOL F GLYCIDYL ETHER/ FORMALDEHYDE COPOLYMER & (C12-14)ALKYLGLYCIDYL ETHER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
834FRB Flame Retardant Epoxy (Part A) & BISPHENOL F GLYCIDYL ETHER/ FORMALDEHYDE COPOLYMER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities. Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.
ANTIMONY TRIOXIDE & CARBON BLACK	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

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

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

SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

834FRB Flame Retardant Epoxy (Part A)

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

bisphenol F glycidyl ether/ formaldehyde copolymer

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

antimony trioxide

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	0.93mg/L	2
EC50	48	Crustacea	1mg/L	2
EC50	96	Algae or other aquatic plants	0.61mg/L	2
NOEC	720	Fish	>0.0075mg/L	2

(C12-14)alkylglycidyl ether

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>5-mg/L	2
EC50	48	Crustacea	6.07mg/L	2
NOEC	48	Crustacea	<10mg/L	2

bisphenol A/ diglycidyl ether resin, liquid

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

naphtha petroleum, heavy alkylate

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
EC50	72	Algae or other aquatic plants	=13mg/L	1
NOEC	72	Algae or other aquatic plants	=0.1mg/L	1

carbon black

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	>100mg/L	2

Continued...

EC50	72	Algae or other aquatic plants	>10-mg/L	2
EC10	72	Algae or other aquatic plants	>10-mg/L	2
NOEC	96	Fish	>=1-mg/L	2

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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
For bisphenol A and related bisphenols:
Environmental fate:
Biodegradability (28 d) 89% - Easily biodegradable
Bioconcentration factor (BCF) 7.8 mg/l
Bisphenol A, its derivatives and analogues, can be released from polymers, resins and certain substances by metabolic products
Substance does not meet the criteria for PBT or vPvB according to Regulation (EC) No 1907/2006, Annex XIII
As an environmental contaminant, bisphenol A interferes with nitrogen fixation at the roots of leguminous plants associated with the bacterial symbiont Sinorhizobium meliloti. Despite a half-life in the soil of only 1-10 days, its ubiquity makes it an important pollutant. According to Environment Canada, 'initial assessment shows that at low levels, bisphenol A can harm fish and organisms over time. Studies also indicate that it can currently be found in municipal wastewater.' However, a study conducted in the United States found that 91-98% of bisphenol A may be removed from water during treatment at municipal water treatment plants.
Ecotoxicity:
Fish LC50 (96 h): 4.6 mg/l (freshwater fish); 11 mg/l (saltwater fish): NOEC 0.016 mg/l (freshwater fish- 144 d); 0.064 mg/l (saltwater fish 164 d)
Fresh water invertebrates EC50 (48 h): 10.2 mg/l: NOEC 0.025 mg/l - 328 d)
Marine water invertebrate EC50 (96 h): 1.1 mg/l; NOEC 0.17 mg/l (28 d)
Freshwater algae (96 h): 2.73 mg/l
Marine water algae (96 h): 1.1 mg/l
Fresh water plant EC50 (7 d): 20 mg/l: NOEC 7.8 mg/l
In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms.
Among freshwater organisms, fish appear to be the most sensitive species. Evidence of endocrine-related effects in fish, aquatic invertebrates, amphibians and reptiles has been reported at environmentally relevant exposure levels lower than those required for acute toxicity. There is a widespread variation in reported values for endocrine-related effects, but many fall in the range of 1 ug/L to 1 mg/L
A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations.
A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.
Although abundant data are available on the toxicity of bisphenol-A (2,2-bis (4-hydroxydiphenyl)propane;(BPA) A variety of BPs were examined for their acute toxicity against Daphnia magna, mutagenicity, and oestrogenic activity using the Daphtoxkit (Creasel Ltd.), the umu test system, and the yeast two-hybrid system, respectively, in comparison with BPA. BPA was moderately toxic to D. magna (48-h EC50 was 10 mg/l) according to the current U.S. EPA acute toxicity evaluation standard, and it was weakly oestrogenic with 5 orders of magnitude lower activity than that of the natural estrogen 17 beta-oestradiol in the yeast screen, while no mutagenicity was observed. All seven BPs tested here showed moderate to slight acute toxicity, no mutagenicity, and weak oestrogenic activity as well as BPA. Some of the BPs showed considerably higher oestrogenic activity than BPA, and others exhibited much lower activity. Bisphenol S (bis(4-hydroxydiphenyl)sulfone) and bis(4-hydroxyphenyl)sulfide) showed oestrogenic activity.
Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F ([bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem,
Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe3+ ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.

Significant environmental findings are limited. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit common characteristics with respect to environmental fate and ecotoxicology. One such oxirane is ethyloxirane and data presented here may be taken as representative.
for 1,2-butylene oxide (ethyloxirane):
Environmental fate: Ethyloxirane is highly soluble in water and has a very low soil-adsorption coefficient, which suggests that if released to water, adsorption of ethyloxirane to sediment and suspended solids is not expected. Volatilisation of ethyloxirane from water surfaces would be expected based on the moderate estimated Henry's Law constant. If ethyloxirane is released to soil, it is expected to have low adsorption and thus very high mobility. Volatilisation from moist soil and dry soil surfaces is expected, based on its vapour pressure. It is expected that ethyloxirane exists solely as a vapour in ambient atmosphere, based on its very high vapour pressure. Ethyloxirane may also be removed from the atmosphere by wet deposition processes, considering its relatively high water solubility.
Persistence: The half-life in air is about 5.6 days from the reaction of ethyloxirane with photochemically produced hydroxyl radicals which indicates that this chemical meets the persistence criterion in air (half-life of = 2 days)*.
Ethyloxirane is hydrolysable, with a half-life of 6.5 days, and biodegradable up to 100% degradation and is not expected to persist in water. A further model-predicted biodegradation half-life of 15 days in water was obtained and used to predict the half-life of this chemical in soil and sediment by applying Boethling's extrapolation factors (t1/2water : t1/2 soil : t1/2sediment = 1: 1: 4) (Boethling 1995). According to these values, it can be concluded that ethyloxirane does not meet the persistence criteria in water and soil (half-lives = 182 days) and sediments (half-life = 365 days).
Experimental and modelled log Kow values of 0.68 and 0.86, respectively, indicate that the potential for bioaccumulation of ethyloxirane in organisms is likely to be low. Modelled bioaccumulation -factor (BAF) and bioconcentration -factor (BCF) values of 1 to 17 L/kg indicate that ethyloxirane does not meet the bioaccumulation criteria (BCF/BAF = 5000)*
Ecotoxicity:
Experimental ecotoxicological data for ethyloxirane (OECD 2001) indicate low to moderate toxicity to aquatic organisms. For fish and water flea, acute LC50/EC50 values vary within a narrow range of 70-215 mg/L; for algae, toxicity values exceed 500 mg/L, while for bacteria they are close to 5000 mg/L

* *Persistence and Bioaccumulation Regulations* (Canada 2000).
Environmental toxicity is a function of the n-octanol/ water partition coefficient (log Pow, log Kow). Phenols with log Pow >7.4 are expected to exhibit low toxicity to aquatic organisms. However the toxicity of phenols with a lower log Pow is variable, ranging from low toxicity (LC50 values >100 mg/l) to highly toxic (LC50 values <1 mg/l) dependent on log Pow, molecular weight and substitutions on the aromatic ring. Dinitrophenols are more toxic than predicted from QSAR estimates. Hazard information for these groups is not generally available.
Drinking Water Standards: hydrocarbon total: 10 ug/l (UK max.).
Antimony exists in the atmosphere in low concentrations. Urban air contains 0.05 to 0.06 ppm of antimony. There are very low concentrations in water due to minimal solubility. Volatilisation from water is not likely. The soil usually contains 0.1 to 10 mg/kg dry weight. Antimony concentrations in freshwater fish are low, approximately 3 mg/kg wet weight.
Little is known of the adsorptive behavior of antimony, its compounds, and ions in soils and sediments. The binding of antimony to soil is determined by the nature of the soil and the form of antimony deposited on the soil. Some forms of antimony may bind to inorganic and organic ligands. On the other hand, a mineral form would be unavailable for binding. Some studies suggest that antimony is fairly mobile under diverse environmental conditions, while others suggest that it is strongly adsorbed to soil.Since antimony has an anionic character (e.g. Sb(OH)i), it is expected to have little affinity for organic carbon. It is not expected that cation exchange, which generally dominates adsorption to clay, would be important for anionic antimony. Antimony is known to form coprecipitates with hydrous iron, manganese, and aluminum oxides in soil and sediment. Antimony adsorbs strongly to colloidal material in soil. The partition coefficient of antimony to 0.05-0.003 um colloids was 1,300. Antimony adsorbed to such material can be transported with the colloids in groundwater.
Leaching experiments performed with river sediment samples from a mining district in Idaho indicated that Sb(V) was the major species released during leaching. The fraction of antimony leached

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from sediment with deionized water after 10 days was highly correlated with the free iron and manganese oxide content of the sediment. The release of antimony from the sediment increased at low pH and increased sharply at high pH. The form of released antimony was also sensitive to pH. At pH 2.7, the bulk of antimony released was as Sb(II1); at pH 4.3, the concentrations of tri- and pentavalent antimony were comparable; and at pH 6.3 and above, Sb(V) was the predominant species.
Antimony does not appear to bioconcentrate appreciably in fish and aquatic organisms. No detectable bioconcentration occurred during a 28-day test in bluegills (EPA 1980). Only low levels of antimony have been reported in fish and aquatic organisms collected off the coast of Africa, Australia, and the Danube River in Austria. Bioconcentration factors for antimony ranged from 0.15 to 390. A study of the distribution of antimony around a smelter site indicated that antimony occurring in plants results from surface deposition. Uptake from soil is minor and appears to be correlated with the amount of available antimony (that which is soluble or easily exchangeable).Antimony bioconcentration was measured in voles, shrews, rabbits, and invertebrates around a smelter. Analysis of antimony in organs of the small mammals, compared with estimates of their antimony intake from food, showed that, although the amount of antimony in the organs was elevated, it was low compared to the amount ingested. The results suggest that antimony does not biomagnify from lower to higher trophic levels in the food chain.
Thermodynamically, most dissolved antimony in natural waters under aerobic conditions should be present in the +5 oxidation state as antimonate species. At 0.001 M total antimony, the dominant species were Sb(OH)6? and Sb(OH)5 0. A small quantity of polymeric hydroxy species were found, but these will be less significant when the total antimony concentration is low, such as in natural water. While industrial inputs will commonly contain antimony in the +3 oxidation state (e.g., antimony trioxide), it is not known how fast antimonite would oxidize to antimonate under natural conditions. Under reducing conditions, trivalent species such as Sb(OH) 3 0, Sb(OH) 4? , and Sb2S4 4- may be significant.
Antimony compounds may undergo photochemical reactions, but these do not appear to be significant in determining their aquatic fate. Antimony trioxide suspensions strongly absorb ultraviolet radiation below 325 nm and darken. The process is reversible, and when the light is removed, the white color slowly returns. The effect is believed to be due to peroxide radical formation on the crystal surface. Both water and oxygen seem to be necessary for the reoxidation of the reduced antimony.
Antimony can be reduced and methylated by microorganisms in the aquatic environment, similar to arsenic, and become mobilized. This reaction is most likely to occur in reducing environments, such as in bed sediment. In the case of arsenic, this reaction may be mediated by fungi and bacteria, but it is not known whether this is the case with antimony. The resulting trimethylstibine is initially oxidized by atmospheric oxygen to a mixture of trimethylstilbine oxide ((CH3)3SbOH) and trimethylstibinic acid ((CH3)2SbO3H), and then to antimony oxides and insoluble polymers. The rate constant is estimated to be of the order of 0.1 to 0.2 L/molsec. Trimethylstibine has a high vapor pressure, 103 mmHg at 25 deg C, and might volatilize before it is completely oxidized. The oxidation product, (CH3)3SbO, is much more soluble than trimethylstibine; therefore, oxidation will reduce volatilization. Oxidation of trimethylstibine in the gas phase is very rapid; the rate is 0.11/mmHg-sec or 2000 L/mol-sec. Trimethylstibine has been shown to react with alkyl iodides and bromides; this results in the formation of quaternary salts. Should antimony occur in a landfill with alkyl halides, the formation of quaternary salts should greatly enhance antimony's mobility.
There is evidence that phytoplankton can reduce Sb(V) to the Sb(III). Sb(III) decreases to very low levels at the base of the seasonal thermocline and remains low down to the sediment where increasing levels are again observed. Sb(III) only accounts for 44% of the inorganic antimony in the anoxic zone, and speciation in this region is unclear. Thermodynamically, the antimony should be in the trivalent state. Thiocomplexes are thought to account for some of the antimony in this zone. Methylated antimony species existed throughout the water column and made up 10% of total antimony. Monomethyl antimony species were more abundant in surface waters and in the anoxic zone. There was no sharp increase in methyl antimony near the sediment, which would be expected if these species were formed biosynthetically. Since the highest antimony concentration is at the surface, it is unlikely that antimony is taken up by phytoplankton, as is the case with arsenic. A decrease in antimony concentration with depth suggests scavenging by particulate matter and, at lower depths, by iron hydroxyoxides.
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

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

12.3. Bioaccumulative potential

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

12.4. Mobility in soil

Ingredient	Mobility
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 Applicable	Not Applicable	Not Applicable
PBT Criteria fulfilled?	Not Applicable	Not Applicable	Not Applicable

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.

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	▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

	For 834FRB-375ML, 834FRB-3L NOT REGULATED by Ground ADR Special Provision 375 NOT REGULATED by Air IATA Special Provision A197 NOT REGULATED by Sea IMDG per 2.10.2.7 NOT REGULATED by ADN Special Provision 274 (The provision of 3.1.2.8 apply)

Land transport (ADR)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol a/ diglycidyl ether resin, liquid)
14.3. **Transport hazard class(es)**	Class: 9 Subrisk: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Hazard identification (Kemler): 90 Classification code: M6 Hazard Label: 9 Special provisions: 274 335 375 601 Limited quantity: 5 L

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 resin, liquid)
14.3. **Transport hazard class(es)**	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**	Special provisions: A97 A158 A197 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 Passenger and Cargo Maximum Qty / Pack: 450 L Passenger and Cargo Limited Quantity Packing Instructions: Y964 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol a/ diglycidyl ether resin, liquid)
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

Continued...

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 resin, liquid)
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

SECTION 15 REGULATORY INFORMATION

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

BISPHENOL F GLYCIDYL ETHER/ FORMALDEHYDE COPOLYMER(28064-14-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

ANTIMONY TRIOXIDE(1309-64-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
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
Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)
Europe European Customs Inventory of Chemical Substances - ECICS (Slovak)
Europe European Customs Inventory of Chemical Substances ECICS (Bulgarian)
Europe European Customs Inventory of Chemical Substances ECICS (Czech)
Europe European Customs Inventory of Chemical Substances ECICS (Romanian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
UK Workplace Exposure Limits (WELs)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

(C12-14)ALKYLGLYCIDYL ETHER(68609-97-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

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

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways
Europe EC Inventory
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification
European Customs Inventory of Chemical Substances ECICS (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
International Air Transport Association (IATA) Dangerous Goods Regulations
International FOSFA List of Banned Immediate Previous Cargoes
International Maritime Dangerous Goods Requirements (IMDG Code)
Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

NAPHTHA PETROLEUM, HEAVY ALKYLATE(64741-65-7.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
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	European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 2) Carcinogens: category 1B (Table 3.1)/category 2 (Table 3.2)	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 4) Mutagens: category 1B (Table 3.1)/category 2 (Table 3.2)	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
Europe EC Inventory	European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
Europe European Agreement concerning the International Carriage of Dangerous Goods by Road - ADR 2017 (Russian)	European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (French)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Norwegian)	European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (German)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Portuguese)	GESAMP/EHS Composite List - GESAMP Hazard Profiles
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)	IMO IBC Code Chapter 17: Summary of minimum requirements
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2015, German)	International Air Transport Association (IATA) Dangerous Goods Regulations
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)	International Maritime Dangerous Goods Requirements (IMDG Code)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2019, French)	Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR-S 2019, Swedish)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
European Customs Inventory of Chemical Substances ECICS (English)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)

CARBON BLACK(1333-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances	European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
European Customs Inventory of Chemical Substances ECICS (English)	UK Workplace Exposure Limits (WELs)
European List of Notified Chemical Substances (ELINCS)	

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) 2015/830; 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 - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (antimony trioxide; (C12-14)alkylglycidyl ether; bisphenol A/ diglycidyl ether resin, liquid; naphtha petroleum, heavy alkylate; bisphenol F glycidyl ether/ formaldehyde copolymer; carbon black)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (bisphenol F glycidyl ether/ formaldehyde copolymer)
Japan - ENCS	No ((C12-14)alkylglycidyl ether; naphtha petroleum, heavy alkylate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Legend:	*Yes = All ingredients are on the inventory* *No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Revision Date	17/03/2020
Initial Date	23/06/2016

Full text Risk and Hazard codes

H226	Flammable liquid and vapour.
H304	May be fatal if swallowed and enters airways.
H336	May cause drowsiness or dizziness.

Other information

Continued...

Ingredients with multiple cas numbers

Name	CAS No
bisphenol F glycidyl ether/ formaldehyde copolymer	28064-14-4, 42616-71-7, 59029-73-1, 94422-39-6
bisphenol A/ diglycidyl ether resin, liquid	25068-38-6, 25085-99-8

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
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

Reason For Change

A-1.01 - Update to the emergency phone number information.

end of SDS