



Kit Revision Date: 09 March 2020

832HD Black 1:1 Epoxy Potting Encapsulating Compound KIT

MG Chemicals Multipart Product Kit

This product is a kit made up of multiple parts. Each part is an independently packaged chemical component and has independent hazard assessments.

Kit Content

<i>Part</i>	<i>Product Name</i>	<i>Product Use</i>
A	832HD-A	Epoxy resin for use with hardeners
B	832HD-B	Epoxy hardener for use with resins

Safety Data Sheets for each part listed above follow this cover sheet.

Transportation Instruction

Before offering this product kit for transport, read Section 14 for all parts listed above.



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

MG Chemicals UK Limited

Version No: A-1.02

Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date: 10/05/2019

Revision Date: 17/03/2020

L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)
Synonyms	SDS Code: 832HD-Part A; 832HD-25ML, 832HD-50ML, 832HD-400ML, 832HD-1.7L, 832HD-7.4L, 832HD-40L
Other means of identification	Not Applicable

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

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

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	+(44) 1663 362888	+(1) 800-201-8822
Fax	Not Available	+(1) 800-708-9888
Website	Not Available	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]	H411 - Chronic Aquatic Hazard Category 2, H315 - Skin Corrosion/Irritation Category 2, H319 - Eye Irritation Category 2, H317 - Skin Sensitizer Category 1
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)

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

Supplementary statement(s)

EUH205	Contains epoxy constituents. May produce an allergic reaction.
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Precautionary statement(s) Prevention

Continued...

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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

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

Not Applicable

Precautionary statement(s) Disposal

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

REACH - Art.57-59: The mixture does not contain Substances of Very High Concern (SVHC) at the SDS print date.

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]
1.1675-54-3 2.216-823-5 3.603-073-00-2 603-074-00-8 4.01-2119456619-26-XXXX	89	<u>bisphenol A diglycidyl ether</u>	Eye Irritation Category 2, Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H319, H317, H315 ^[2]
1.17557-23-2 2.241-536-7 3.603-094-00-7 4.01-2120759332-55-XXXX	6	<u>neopentyl glycol diglycidyl ether</u>	Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H317, H315 ^[2]
1.64741-65-7 2.265-067-2 3.649-275-00-4 4.01-2120009436-62-XXXX	2	<u>naphtha petroleum, heavy alkylate</u>	Flammable Liquid Category 3, Aspiration Hazard Category 1, Specific target organ toxicity - single exposure Category 3 (narcotic effects); H226, H304, H336 ^[1]
1.25068-38-6 2.500-033-5 3.603-074-00-8 4.01-2119456619-26-XXXX	1	<u>bisphenol A/ diglycidyl ether resin, liquid</u>	Eye Irritation Category 2, Chronic Aquatic Hazard Category 2, Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H319, H411, H317, H315 ^[2]
1.1333-86-4 2.215-609-9 3.Not Available 4.01-2119384822-32-XXXX 01-2120767622-50-XXXX 01-0000016864-62-XXXX	0.4	<u>carbon black</u>	Carcinogenicity Category 2; H351 ^[1]
1.68609-97-2 2.271-846-8 3.603-103-00-4 4.01-2119485289-22-XXXX	0.3	<u>(C12-14)alkylglycidyl ether</u>	Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 2; H317, H315 ^[2]
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

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.
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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.

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

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

5.2. Special hazards arising from the substrate or mixture

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

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 acid smoke. ▶ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO₂) 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	· In the event of a spill of a reactive diluent, the focus is on containing the spill to prevent contamination of soil and surface or ground water. · If irritating vapors are present, an approved air-purifying respirator with organic vapor canister is recommended for cleaning up spills and leaks. · For small spills, reactive diluents should be absorbed with sand. Environmental hazard - contain spillage. ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Industrial spills or releases of reactive diluents are infrequent and generally contained. If a large spill does occur, the material should be captured, collected, and reprocessed or disposed of according to applicable governmental requirements. An approved air-purifying respirator with organic-vapor canister is recommended for emergency work. Moderate hazard. ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course.

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- ▶ 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.

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	In general, uncured epoxy resins have only poor mechanical, chemical and heat resistance properties. However, good properties are obtained by reacting the linear epoxy resin with suitable curatives to form three-dimensional cross-linked thermoset structures. This process is commonly referred to as curing or gelation process. Curing of epoxy resins is an exothermic reaction and in some cases produces sufficient heat to cause thermal degradation if not controlled. Curing may be achieved by reacting an epoxy with itself (homopolymerisation) or by forming a copolymer with polyfunctional curatives or hardeners. In principle, any molecule containing a reactive hydrogen may react with the epoxide groups of the epoxy resin. Common classes of hardeners for epoxy resins include amines, acids, acid anhydrides, phenols, alcohols and thiols. Relative reactivity (lowest first) is approximately in the order: phenol < anhydride < aromatic amine < cycloaliphatic amine < aliphatic amine < thiol. The epoxy curing reaction may be accelerated by addition of small quantities of accelerators. Tertiary amines, carboxylic acids and alcohols (especially phenols) are effective accelerators. Bisphenol A is a highly effective and widely used accelerator, but is now increasingly replaced due to health concerns with this substance. Epoxy resin may be reacted with itself in the presence of an anionic catalyst (a Lewis base such as tertiary amines or imidazoles) or a cationic catalyst (a Lewis acid such as a boron trifluoride complex) to form a cured network. This process is known as catalytic homopolymerisation. The resulting network contains only ether bridges, and exhibits high thermal and chemical resistance, but is brittle and often requires elevated temperature to effect curing, so finds only niche applications industrially. Epoxy homopolymerisation is often used when there is a requirement for UV curing, since cationic UV catalysts may be employed (e.g. for UV coatings). Epoxydes: ▶ are highly reactive with acids, bases, and oxidising and reducing agents. ▶ react, possibly violently, with anhydrous metal chlorides, ammonia, amines and group 1 metals. ▶ may polymerise in the presence of peroxides or heat - polymerisation may be violent ▶ may react, possibly violently, with water in the presence of acids and other catalysts. Glycidyl ethers: ▶ may form unstable peroxides on storage in air, light, sunlight, UV light or other ionising radiation, trace metals - inhibitor should be maintained at adequate levels ▶ may polymerise in contact with heat, organic and inorganic free radical producing initiators ▶ may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines ▶ react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide ▶ attack some forms of plastics, coatings, and rubber Reactive diluents are stable under recommended storage conditions, but can decompose at elevated temperatures. In some cases, decomposition can cause pressure build-up in closed systems. ▶ Avoid cross contamination between the two liquid parts of product (kit). ▶ If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur. ▶ This excess heat may generate toxic vapour ▶ Avoid reaction with amines, mercaptans, strong acids and oxidising agents

7.3. Specific end use(s)

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

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)	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 A diglycidyl ether	Bisphenol A diglycidyl ether	39 mg/m3	430 mg/m3	2,600 mg/m3
bisphenol A diglycidyl ether	Epoxy resin includes EPON 1001, 1007, 820, ERL-2795	90 mg/m3	990 mg/m3	5,900 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 A diglycidyl ether	Not Available	Not Available
neopentyl glycol diglycidyl ether	Not Available	Not Available
naphtha petroleum, heavy alkylate	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
(C12-14)alkylglycidyl ether	Not Available	Not Available

MATERIAL DATA

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

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

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

For epichlorohydrin

Odour Threshold Value: 0.08 ppm

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

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

Odour Safety Factor (OSF)

OSF=0.54 (EPICHLOROHYDRIN)

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 '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:

Continued...

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	<table><tr><td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td><td>0.25-0.5 m/s (50-100 f/min)</td></tr><tr><td>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)</td><td>0.5-1 m/s (100-200 f/min.)</td></tr><tr><td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 f/min.)</td></tr><tr><td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td><td>2.5-10 m/s (500-2000 f/min.)</td></tr></table> Within each range the appropriate value depends on: <table><tr><td>Lower end of the range</td><td>Upper end of the range</td></tr><tr><td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr><tr><td>2: Contaminants of low toxicity or of nuisance value only.</td><td>2: Contaminants of high toxicity</td></tr><tr><td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr><tr><td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr></table> 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.	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.)	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
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4: Large hood or large air mass in motion	4: Small hood-local control only																		
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 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.																		

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	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 ▶ DO NOT use solvent to clean the skin
Body protection	See Other protection below
Other protection	- Overalls. - P.V.C. apron. - Barrier cream. - Skin cleansing cream. - Eye wash unit.

Respiratory protection

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

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

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

^ - Full-face

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

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

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

9.1. Information on basic physical and chemical properties

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

Continued...

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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. In animal testing, exposure to aerosols of some reactive diluents (notably o-cresol glycidyl ether, CAS RN: 2210-79-9) has been reported to affect the adrenal gland, central nervous system, kidney, liver, ovaries, spleen, testes, thymus, and respiratory tract. Inhalation hazard is increased at higher temperatures. Not normally a hazard due to non-volatile nature of product
Ingestion	Reactive diluents exhibit a range of ingestion hazards. Small amounts swallowed incidental to normal handling operations are not likely to cause injury. However, swallowing larger amounts may cause injury. Male rats exposed to a single oral dose of bisphenol A diglycidyl ether (BADGE) at 750, 1000, and 2000 mg/kg/day showed a significantly increase in the number of immature and maturing sperm on the testis. There were no significant differences with respect to sperm head count, sperm motility, and sperm abnormality in the BADGE treatment groups The material has NOT been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Bisphenol A diglycidyl ether (BADGE) may produce contact dermatitis characterised by erythema and oedema, with weeping followed by crusting and scaling. A liquid resin with a molecular weight of 350 produced severe skin irritation in rabbits when applied daily for 4 hours over 20 days. Following the initial contact there may be a discrete erythematous lesion, confined to the point of contact, which may persist for 48 hours to 10 days; the erythema may give way to a papular, vesicular rash with scaling. In animals uncured resin produces moderate ante-mortem depression, loss of body weight and diarrhoea. Local irritation, inflammation and death resulting from respiratory system depression are recorded. Higher molecular weight resins generally produce lower toxicity. Skin contact with reactive diluents may cause slight to moderate irritation with local redness. Repeated or prolonged skin contact may cause burns. Open cuts, 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. The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (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.
Eye	Eye contact with reactive diluents may cause slight to severe irritation with the possibility of chemical burns or moderate to severe corneal injury. Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Chemicals containing epoxy groups are of concern for cancer effects, though the concern is lower for epoxy groups with di-substituted carbons (US EPA 1994) The epoxide group is an alkylating agent and thus may produce damage to nucleotides found within the cell; such damage is potentially tumourigenic. 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) needs months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. Chemicals containing epoxy functional groups are of concern for reproductive effects, though the concern for epoxy groups with di-substituted carbons is lower than that for singly substituted epoxy groups (US EPA, 1994). Bisphenol A diglycidyl ethers (BADGEs) produce sensitisation dermatitis characterised by a papular, vesicular eczema with considerable itching of the back of the hand, the forearm and face and neck. This lesion may persist for 10-14 days after withdrawal from exposure and recur immediately on re-exposure. This dermatitis may persist for longer periods following each exposure but is unlikely to become more intense. Lesions may develop a brownish colour and scaling occurs frequently. Lower molecular weight species produce sensitisation more readily. In mice technical grades of bisphenol A diglycidyl ether produced epidermal tumours and a small increase in the incidence kidney tumours in males and of lymphoreticular/ haematopoietic tumours in females. Subcutaneous injection produced a small number of fibrosarcomas in rats. BADGE is listed as an IARC Group 3 carcinogen, meaning it is 'not classifiable as to its carcinogenicity to humans'. Concern has been raised over this

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possible carcinogenicity because BADGE is used in epoxy resins in the lining of some tin cans for foodstuffs, and unreacted BADGE may end up in the contents of those cans.

For some reactive diluents, prolonged or repeated skin contact may result in absorption of potentially harmful amounts or allergic skin reactions. Exposure to some reactive diluents (notably neopentylglycol diglycidyl ether, CAS RN:17557-23-2) has caused cancer in some animal testing. All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.

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

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

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

832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)	TOXICITY	IRRITATION
	Not Available	Not Available
bisphenol A diglycidyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 2 mg/24h - SEVERE
	Oral (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg - mild
		Skin: adverse effect observed (irritating) ^[1]
neopentyl glycol diglycidyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (rat) LD50: 4500 mg/kg ^[2]	Skin (human): Sensitiser [Shell]
		Skin: adverse effect observed (irritating) ^[1]
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]	
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]	
carbon black	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: >15400 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
(C12-14)alkylglycidyl ether	TOXICITY	IRRITATION
	Oral (rat) LD50: >10000 mg/kg ^[2]	Eye (rabbit): mild [Ciba]
		Eye: adverse effect observed (irritating) ^[1]
		Skin (guinea pig): sensitiser
		Skin (human): Irritant
		Skin (human): non- sensitiser
		Skin (rabbit): moderate
		Skin : Moderate
		Skin: adverse effect observed (irritating) ^[1]

Legend:

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

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832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)	Bisphenol A diglycidyl ethers (BADGEs) produce sensitisation dermatitis characterised by a papular, vesicular eczema with considerable itching of the back of the hand, the forearm and face and neck. This lesion may persist for 10-14 days after withdrawal from exposure and recur immediately on re-exposure. This dermatitis may persist for longer periods following each exposure but is unlikely to become more intense. Lesions may develop a brownish colour and scaling occurs frequently. Lower molecular weight species produce sensitisation more readily. In mice technical grades of bisphenol A diglycidyl ether produced epidermal tumours and a small increase in the incidence kidney tumours in males and of lymphoreticular/ haematopoietic tumours in females. Subcutaneous injection produced a small number of fibrosarcomas in rats. BADGE is listed as an IARC Group 3 carcinogen, meaning it is 'not classifiable as to its carcinogenicity to humans'. Concern has been raised over this possible carcinogenicity because BADGE is used in epoxy resins in the lining of some tin cans for foodstuffs, and unreacted BADGE may end up in the contents of those cans.
BISPHENOL A DIGLYCIDYL ETHER	55badger
NEOPENTYL GLYCOL DIGLYCIDYL ETHER	* Anchor SDS]
NAPHTHA PETROLEUM, HEAVY ALKYLATE	For Low Boiling Point Naphthas (LBPNS): Acute toxicity: LBPNS generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure Most LBPNS are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices. Sensitisation: LBPNS do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies Repeat dose toxicity: The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPNS 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 LBPNS substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results. For in vivo genotoxicity tests, LBPNS exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells. For in vitro genotoxicity studies, LBPNS were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNS exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay. Mixed results were observed for UDS and the mouse lymphoma assay. While the majority of in vivo genotoxicity results for LBPNS substances are negative, the potential for genotoxicity of LBPNS as a group cannot be discounted based on the mixed in vitro genotoxicity results. Carcinogenicity: Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect of human exposure to LBPNS substances. No inhalation studies assessing the carcinogenicity of the site-restricted LBPNS were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/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 LBPNS group. Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPNS substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPNS, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans). Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNS. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals' lifetime or until a tumour persisted

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	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 LBP 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 LBP substances 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 LBP substances, 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 than iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the 'hydrocarbon continuum hypothesis', and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. for petroleum: 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.
BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	Foetotoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity); NOEL (maternal) 60 mg/kg
CARBON BLACK	No significant acute toxicological data identified in literature search. WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Inhalation (rat) TCLo: 50 mg/m³/6h/90D-I Nil reported
832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A) & BISPHENOL A DIGLYCIDYL ETHER & NEOPENTYL GLYCOL DIGLYCIDYL ETHER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & (C12-14)ALKYLGlycidyl ETHER	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.
832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A) & BISPHENOL A DIGLYCIDYL ETHER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg). Reproductive and Developmental Toxicity: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage 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

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	(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 <i>S. typhimurium</i> 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 <i>in vivo</i> and <i>in vitro</i> assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/kg body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weight/day (male rats) from the 2-year carcinogenicity study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELS from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs.
832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A) & 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.
832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A) & BISPHENOL A DIGLYCIDYL ETHER	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 hypospadias and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades' One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials' One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood. A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another <i>in vitro</i> 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. <i>In vitro</i> studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potentially promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes. Bisphenol A is the isopropyl adduct of 4,4'-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called 'cytostatic hormones'. Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown to be oestrogenic <i>in vitro</i>; 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, <i>in vitro</i>, significantly inhibit bisphenol A glucuronidation (detoxification). All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether. A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not <i>n</i>-butyl glycidyl ether, induced morphological transformation in mammalian cells <i>in vitro</i>. <i>n</i>-Butyl glycidyl ether induced micronuclei in mice <i>in vivo</i> following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations <i>in vivo</i> or chromosomal aberrations in animal cells <i>in vitro</i>. 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 <i>Drosophila</i>. The glycidyl ethers were generally mutagenic to bacteria 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.

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

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

832HD-A Black 1:1 Epoxy Potting and Encapsulating Compound (Part A)	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
bisphenol A diglycidyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.2mg/L	2
	EC50	48	Crustacea	1.1mg/L	2
	EC50	72	Algae or other aquatic plants	9.4mg/L	2
	EC0	48	Crustacea	<1mg/L	2
	NOEC	504	Crustacea	0.3mg/L	2
neopentyl glycol diglycidyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	12.318mg/L	3
	EC50	96	Algae or other aquatic plants	ca.1-73.67mg/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
bisphenol A/ diglycidyl ether resin, liquid	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	48	Crustacea	ca.2mg/L	2
carbon black	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	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
(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

Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12

Continued...

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(QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

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

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

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

Liquid epoxy resins and some reactive diluents are not readily biodegradable, although its epoxy functional groups are hydrolysed in contact with water, they have the potential to bio-accumulate and are moderately toxic to aquatic organisms. They are generally classified as dangerous for the environment according to the European Union classification criteria.

Uncured solid resins on the other hand are not readily bio-available, not toxic to aquatic and terrestrial organisms, not readily biodegradable, but hydrolysable. They present no significant hazard for the environment.

For bisphenol A and related bisphenols:

Environmental fate:

Biodegradability (28 d) 89% - Easily biodegradable

Bioconcentration factor (BCF) 7.8 mg/l

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

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

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

Ecotoxicity:

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

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

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

Freshwater algae (96 h): 2.73 mg/l

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

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

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

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

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

They would not be expected to persist in the environment.

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

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

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

for 1,2-butylene oxide (ethyloxirane):

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

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

Ethyloxirane is hydrolysable, with a half-life of 6.5 days, and biodegradable up to 100% degradation and is not expected to persist in water. A further model-predicted biodegradation half-life of 15 days in water was obtained and used to predict the half-life of this chemical in soil and sediment by applying Boethling's extrapolation factors (t_{1/2}water : t_{1/2}soil : t_{1/2}sediment = 1: 1: 4) (Boethling 1995). According to these values, it can be concluded that ethyloxirane does not meet the persistence criteria in water and soil (half-lives = 182 days) and sediments (half-life = 365 days).

Experimental and modelled log Kow values of 0.68 and 0.86, respectively, indicate that the potential for bioaccumulation of ethyloxirane in organisms is likely to be low. Modelled bioaccumulation -factor (BAF) and bioconcentration -factor (BCF) values of 1 to 17 L/kg indicate that ethyloxirane does not meet the bioaccumulation criteria (BCF/BAF = 5000)*

Ecotoxicity:

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

* Persistence and Bioaccumulation Regulations (Canada 2000).

12.2. Persistence and degradability

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

Continued...

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bisphenol A/ diglycidyl ether resin, liquid	HIGH	HIGH
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12.3. Bioaccumulative potential

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

12.4. Mobility in soil

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

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not 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. Waste Management Production waste from epoxy resins and resin systems should be treated as hazardous waste in accordance with National regulations. Fire retarded resins containing halogenated compounds should also be treated as special waste. Accidental spillage of resins, curing agents and their formulations should be contained and absorbed by special mineral absorbents to prevent them from entering the environment. Contaminated or surplus product should not be washed down the sink, but preferably be fully reacted to form cross-linked solids which is non-hazardous and can be more easily disposed. Finished articles made from fully cured epoxy resins are hard, infusible solids presenting no hazard to the environment. However, finished articles from flame-retarded material containing halogenated resins should be considered hazardous waste, and disposed as required by National laws. Articles made from epoxy resins, like other thermosets, can be recycled by grinding and used as fillers in other products. Another way of disposal and recovery is combustion with energy recovery. 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. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ Material may be disposed of by controlled burning in an approved incinerator or buried in an approved landfill. ▶ Prior to disposal in a landfill the material should be mixed with the other component and reacted to render the material inert. ▶ Extreme caution should be taken when heating the resin/curing agent mix. ▶ Recycle containers where possible, or dispose of in an authorised landfill.	
	Waste treatment options	Not Available
	Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

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For 832HD-25ML, 832HD-50ML, 832HD-400ML, 832HD-1.7L, 832HD-7.4L
 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)										
14.3. Transport hazard class(es)	<table> <tr> <td>Class</td><td>9</td></tr> <tr> <td>Subrisk</td><td>Not Applicable</td></tr> </table>	Class	9	Subrisk	Not Applicable						
Class	9										
Subrisk	Not Applicable										
14.4. Packing group	III										
14.5. Environmental hazard	Environmentally hazardous										
14.6. Special precautions for user	<table> <tr> <td>Hazard identification (Kemler)</td><td>90</td></tr> <tr> <td>Classification code</td><td>M6</td></tr> <tr> <td>Hazard Label</td><td>9</td></tr> <tr> <td>Special provisions</td><td>274 335 375 601</td></tr> <tr> <td>Limited quantity</td><td>5 L</td></tr> </table>	Hazard identification (Kemler)	90	Classification code	M6	Hazard Label	9	Special provisions	274 335 375 601	Limited quantity	5 L
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)														
14.3. Transport hazard class(es)	<table> <tr> <td>ICAO/IATA Class</td><td>9</td></tr> <tr> <td>ICAO / IATA Subrisk</td><td>Not Applicable</td></tr> <tr> <td>ERG Code</td><td>9L</td></tr> </table>	ICAO/IATA Class	9	ICAO / IATA Subrisk	Not Applicable	ERG Code	9L								
ICAO/IATA Class	9														
ICAO / IATA Subrisk	Not Applicable														
ERG Code	9L														
14.4. Packing group	III														
14.5. Environmental hazard	Environmentally hazardous														
14.6. Special precautions for user	<table> <tr> <td>Special provisions</td><td>A97 A158 A197</td></tr> <tr> <td>Cargo Only Packing Instructions</td><td>964</td></tr> <tr> <td>Cargo Only Maximum Qty / Pack</td><td>450 L</td></tr> <tr> <td>Passenger and Cargo Packing Instructions</td><td>964</td></tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td><td>450 L</td></tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td><td>Y964</td></tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td><td>30 kg G</td></tr> </table>	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
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)						
14.3. Transport hazard class(es)	<table> <tr> <td>IMDG Class</td><td>9</td></tr> <tr> <td>IMDG Subrisk</td><td>Not Applicable</td></tr> </table>	IMDG Class	9	IMDG Subrisk	Not Applicable		
IMDG Class	9						
IMDG Subrisk	Not Applicable						
14.4. Packing group	III						
14.5. Environmental hazard	Marine Pollutant						
14.6. Special precautions for user	<table> <tr> <td>EMS Number</td><td>F-A , S-F</td></tr> <tr> <td>Special provisions</td><td>274 335 969</td></tr> <tr> <td>Limited Quantities</td><td>5 L</td></tr> </table>	EMS Number	F-A , S-F	Special provisions	274 335 969	Limited Quantities	5 L
EMS Number	F-A , S-F						
Special provisions	274 335 969						
Limited Quantities	5 L						

Inland waterways transport (ADN)

14.1. UN number	3082				
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A diglycidyl ether)				
14.3. Transport hazard class(es)	<table> <tr> <td>9</td><td>Not Applicable</td></tr> </table>	9	Not Applicable		
9	Not Applicable				
14.4. Packing group	III				
14.5. Environmental hazard	Environmentally hazardous				
14.6. Special precautions for user	<table> <tr> <td>Classification code</td><td>M6</td></tr> <tr> <td>Special provisions</td><td>274; 335; 375; 601</td></tr> </table>	Classification code	M6	Special provisions	274; 335; 375; 601
Classification code	M6				
Special provisions	274; 335; 375; 601				

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

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 A DIGLYCIDYL ETHER(1675-54-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances	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 ECHA Registered Substances - Classification and Labelling - DSD-DPD	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 2017, English)	IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
European Customs Inventory of Chemical Substances ECICS (English)	International Air Transport Association (IATA) Dangerous Goods Regulations
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation	International FOSFA List of Banned Immediate Previous Cargoes
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	International Maritime Dangerous Goods Requirements (IMDG Code)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31	Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)
European Union (EU) No-Longer Polymers List (NLP) (67/548/EEC)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)

NEOPENTYL GLYCOL DIGLYCIDYL ETHER(17557-23-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Europe EC Inventory	European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	International FOSFA List of Banned Immediate Previous Cargoes

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

ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways	European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
Europe EC Inventory	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2011, Spanish)	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format
European Agreement concerning the International Carriage of Dangerous Goods by Road (ADR 2017, English)	European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List (English)
European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification	GESAMP/EHS Composite List - GESAMP Hazard Profiles
European Customs Inventory of Chemical Substances ECICS (English)	IMO IBC Code Chapter 17: Summary of minimum requirements
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)(English)	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 (English)

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

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

Continued...

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

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)	

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

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

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 ((C12-14)alkylglycidyl ether; bisphenol A/ diglycidyl ether resin, liquid; neopentyl glycol diglycidyl ether; bisphenol A diglycidyl ether; naphtha petroleum, heavy alkylate; carbon black)
China - IECSC	Yes
Europe - EINECS / ELINCS / NLP	Yes
Japan - ENCS	No ((C12-14)alkylglycidyl ether; naphtha petroleum, heavy alkylate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No ((C12-14)alkylglycidyl ether; neopentyl glycol diglycidyl ether; bisphenol A diglycidyl ether)
Vietnam - NCI	Yes
Russia - ARIPS	No (neopentyl glycol diglycidyl ether; naphtha petroleum, heavy alkylate)
Thailand - TECI	No (bisphenol A/ diglycidyl ether resin, liquid; neopentyl glycol diglycidyl ether; bisphenol A diglycidyl ether; naphtha petroleum, heavy alkylate)
Legend:	Yes = All declared ingredients are on the inventory No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	17/03/2020
Initial Date	11/05/2019

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.
H351	Suspected of causing cancer.

Other information**Ingredients with multiple cas numbers**

Name	CAS No
bisphenol A diglycidyl ether	1675-54-3, 116161-20-7, 170962-54-6, 47424-12-4, 85101-00-4, 25068-38-6

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

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-1.02 - Update to the emergency phone number information.



832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)

MG Chemicals UK Limited

Version No: A-1.02

Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date: 14/02/2018

Revision Date: 17/03/2020

L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)
Synonyms	SDS Code: 832HD-Part B; 832HD-25ML, 832HD-50ML, 832HD-400ML, 832HD-1.7L, 832HD-7.4L, 832HD-40L
Other means of identification	Not Available

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

Relevant identified uses	Epoxy hardener for use with resins
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]	H302 - Acute Toxicity (Oral) Category 4, H312 - Acute Toxicity (Dermal) Category 4, H332 - Acute Toxicity (Inhalation) Category 4, H314 - Skin Corrosion/Irritation Category 1A, H317 - Skin Sensitizer Category 1, H341 - Germ cell mutagenicity Category 2, H361 - Reproductive Toxicity Category 2, H335 - Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), H410 - Chronic Aquatic Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

2.2. Label elements

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

H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H332	Harmful if inhaled.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H341	Suspected of causing genetic defects.
H361	Suspected of damaging fertility or the unborn child.
H335	May cause respiratory irritation.

Continued...

832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)

H410	Very toxic to aquatic life with long lasting effects.
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Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

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

Cumulative effects may result following exposure*.

Limited evidence of a carcinogenic effect*.

Possible respiratory sensitizer*.

4-nonylphenol, branched	Listed in the European Chemicals Agency (ECHA) Candidate List of Substances of Very High Concern for Authorisation
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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]
1.84852-15-3 2.284-325-5 3.601-053-00-8 4.01-2119510715-45-XXXX	41	<u>4-nonylphenol, branched</u>	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1B, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H361fd, H302, H314, H410 ^[3]
1.68953-36-6 2.271-417-5 273-201-6 3.Not Available 4.01-2119487006-38-XXXX	37	<u>tall oil/ tetraethylenepentamine polyamides</u>	Metal Corrosion Category 1, Skin Corrosion/Irritation Category 1A, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Reproductive Toxicity Category 1B, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H290, H314, H317, H360, H410 ^[1]
1.6864-37-5 2.229-962-1 3.612-110-00-1 4.01-2119497829-12-XXXX	16	<u>4,4'-methylenebis(2-methylcyclohexanamine)</u>	Acute Toxicity (Inhalation) Category 3, Acute Toxicity (Dermal) Category 3, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1A, Chronic Aquatic Hazard Category 2; H331, H311, H302, H314, H411 ^[3]

Continued...

832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)

1.112-57-2 2.203-986-2 3.612-060-00-0 4. Not Available	3	<u>tetraethylenepentamine</u>	Acute Toxicity (Dermal) Category 4, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1B, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 2; H312, H302, H314, H317, H411 ^[3]
1.64741-65-7. 2.265-067-2 3.649-275-00-4 4.01-2120009436-62-XXXX	2	<u>naphtha petroleum, heavy alkylate</u>	Flammable Liquid Category 3, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1; H226, H336, H304 ^[1]
1.108-95-2 2.203-632-7 3.604-001-00-2 4.01-2119471329-32-XXXX	0.2	<u>phenol</u>	Germ cell mutagenicity Category 2, Acute Toxicity (Inhalation) Category 3, Acute Toxicity (Dermal) Category 3, Acute Toxicity (Oral) Category 3, Specific target organ toxicity - repeated exposure Category 2, Skin Corrosion/Irritation Category 1B; H341, H331, H311, H301, H373 **, H314 ^[3]
Legend:		1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI 4. Classification drawn from C&L	

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay. ▶ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. ▶ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs). ▶ As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested. ▶ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered. This must definitely be left to a doctor or person authorised by him/her. (ICSC13719)
Ingestion	▶ For advice, contact a Poisons Information Centre or a doctor at once. ▶ Urgent hospital treatment is likely to be needed. ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Transport to hospital or doctor without delay.

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.

For acute or short-term repeated exposures to highly alkaline materials:

- ▶ Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- ▶ Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- ▶ Oxygen is given as indicated.
- ▶ The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- ▶ Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- ▶ Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

- ▶ Neutralising agents should never be given since exothermic heat reaction may compound injury.

* Catharsis and emesis are absolutely contra-indicated.

* Activated charcoal does not absorb alkali.

* Gastric lavage should not be used.

Supportive care involves the following:

- ▶ Withhold oral feedings initially.
- ▶ If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- ▶ Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- ▶ Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

- ▶ Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

Continued...

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For acute or short term repeated exposures to phenols/ cresols:

- ▶ Phenol is absorbed rapidly through lungs and skin. [Massive skin contact may result in collapse and death]*
- ▶ [Ingestion may result in ulceration of upper respiratory tract; perforation of oesophagus and/or stomach, with attendant complications, may occur. Oesophageal stricture may occur.]*
- ▶ An initial excitatory phase may present. Convulsions may appear as long as 18 hours after ingestion. Hypotension and ventricular tachycardia that require vasopressor and antiarrhythmic therapy, respectively, can occur.
- ▶ Respiratory arrest, ventricular dysrhythmias, seizures and metabolic acidosis may complicate severe phenol exposures so the initial attention should be directed towards stabilisation of breathing and circulation with ventilation, intubation, intravenous lines, fluids and cardiac monitoring as indicated.
- ▶ [Vegetable oils retard absorption; do NOT use paraffin oils or alcohols. Gastric lavage, with endotracheal intubation, should be repeated until phenol odour is no longer detectable; follow with vegetable oil. A saline cathartic should then be given.]* ALTERNATIVELY: Activated charcoal (1g/kg) may be given. A cathartic should be given after oral activated charcoal.
- ▶ Severe poisoning may require slow intravenous injection of methylene blue to treat methaemoglobinaemia.
- ▶ [Renal failure may require haemodialysis.]*
- ▶ Most absorbed phenol is biotransformed by the liver to ethereal and glucuronide sulfates and is eliminated almost completely after 24 hours. [Ellenhorn and Barceloux: Medical Toxicology]

*[Union Carbide]

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker who has been exposed to the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
1. Total phenol in blood	250 mg/gm creatinine	End of shift	B, NS

B: Background levels occur in specimens collected from subjects **NOT** exposed

NS: Non-specific determinant; also seen in exposure to other materials

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
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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 fire fighting procedures suitable for surrounding area. ▶ Do not approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▶ 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 acid smoke. ▶ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO₂) nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material. May emit corrosive fumes.

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	▶ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material. ▶ Check regularly for spills and leaks. Small spills should be covered with inorganic absorbents and disposed of properly. Organic absorbents have been known to ignite when contaminated with amines in closed containers. Certain cellulosic materials used for spill cleanup such as wood chips or sawdust have shown reactivity with ethyleneamines and should be avoided. Ethyleneamine leaks will frequently be identified by the odor (ammoniacal) or by the formation of a white, solid, waxy substance (amine carbamates). Inorganic absorbents or water may be used to clean up the amine waste. ▶ 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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Continued...

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Chemical Class: phenols and cresols

For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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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

Major Spills

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

- ▶ Clear area of personnel and move upwind.
- ▶ 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.
- ▶ Consider evacuation (or protect in place).
- ▶ Stop leak if safe to do so.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Neutralise/decontaminate residue (see Section 13 for specific agent).
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- ▶ 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. ▶ WARNING: To avoid violent reaction, ALWAYS add material to water and NEVER water to material. ▶ 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. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
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. ▶ DO NOT store near acids, or oxidising agents ▶ No smoking, naked lights, heat or ignition sources.

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7.2. Conditions for safe storage, including any incompatibilities

Suitable container	▶ DO NOT use aluminium or galvanised containers ▶ DO NOT use aluminium, galvanised or tin-plated containers ▶ Lined metal can, lined metal pail/ can. ▶ Plastic pail. ▶ Polyliner drum. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks. For low viscosity materials ▶ Drums and jerricans must be of the non-removable head type. ▶ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ▶ Removable head packaging; ▶ Cans with friction closures and ▶ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	▶ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air. ▶ 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. ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ▶ Avoid contact with copper, aluminium and their alloys. ▶ Avoid reaction with oxidising agents

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)	phenol	Phenol	7.8 mg/m ³ / 2 ppm	16 mg/m ³ / 4 ppm	Not Available	Sk
European Union (EU) Third List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	phenol	Phenol	8 mg/m ³ / 2 ppm	16 mg/m ³ / 4 ppm	Not Available	skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	phenol	Phenol	7.8 mg/m ³ / 2 ppm	Not Available	Not Available	Skin

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
4-nonylphenol, branched	Nonyl phenol, 4- (branched)	0.2 mg/m ³	2.3 mg/m ³	260 mg/m ³
4,4'-methylenebis(2-methylcyclohexanamine)	Laromin C 260; (bis(4-Amino-3-methylcyclohexyl) methane; Dimethyldicyane)	0.28 mg/m ³	3.1 mg/m ³	19 mg/m ³
tetraethylenepentamine	Tetraethylenepentamine	15 mg/m ³	130 mg/m ³	790 mg/m ³
phenol	Phenol	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
4-nonylphenol, branched	Not Available	Not Available
tall oil/ tetraethylenepentamine polyamides	Not Available	Not Available
4,4'-methylenebis(2-methylcyclohexanamine)	Not Available	Not Available
tetraethylenepentamine	Not Available	Not Available
naphtha petroleum, heavy alkylate	Not Available	Not Available
phenol	250 ppm	Not Available

MATERIAL DATA

Continued...

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Polyamide hardeners have much reduced volatility, toxicity and are much less irritating to the skin and eyes than amine hardeners. However commercial polyamides may contain a percentage of residual unreacted amine and all unnecessary contact should be avoided.

Odour Threshold Value for phenol: 0.060 ppm (detection)

NOTE: Detector tubes for phenol, measuring in excess of 1 ppm, are commercially available.

Systemic absorption by all routes may induce convulsions with damage to the lungs and central nervous system.

Exposure at or below the recommended TLV-TWA is thought to protect the worker from respiratory, cardiovascular, hepatic, renal and neurological toxicity. Workers or volunteers exposed at or below 5.2 ppm phenol have experienced no ill-effects. Because phenol as a vapour, liquid or solid can penetrate the skin causing systemic effects, a skin notation is considered necessary. Although ACGIH has not recommended a STEL it is felt that ACGIH excursion limits (15 ppm limited to a total duration of 30 minutes with brief excursions limited to no more than 25 ppm) and NIOSH Ceiling values are sufficiently similar so as to provide the same margin of safety.

Odour Safety Factor(OSF)

OSF=25 (PHENOL)

NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV.

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

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

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

8.2. Exposure controls

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 '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. Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. 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. <table border="1" data-bbox="391 1010 1490 1267"> <thead> <tr> <th>Type of Contaminant:</th><th>Air Speed:</th></tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td><td>0.25-0.5 m/s (50-100 f/min.)</td></tr> <tr> <td>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)</td><td>0.5-1 m/s (100-200 f/min.)</td></tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 f/min.)</td></tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td><td>2.5-10 m/s (500-2000 f/min.)</td></tr> </tbody> </table> Within each range the appropriate value depends on: <table border="1" data-bbox="391 1323 1490 1491"> <thead> <tr> <th>Lower end of the range</th><th>Upper end of the range</th></tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td><td>2: Contaminants of high toxicity</td></tr> <tr> <td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr> <tr> <td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr> </tbody> </table> 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.	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.)	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
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3: Intermittent, low production.	3: High production, heavy use																				
4: Large hood or large air mass in motion	4: Small hood-local control only																				
8.2.2. Personal protection																					
Eye and face protection	▶ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ▶ Chemical goggles whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ▶ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ▶ Alternatively a gas mask may replace splash goggles and face shields. ▶ 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																				

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Hands/feet protection	▶ Elbow length PVC gloves ▶ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. NOTE: ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. ▶ When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butadiene rubber), boots and aprons. ▶ DO NOT use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber 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.
Body protection	See Other protection below
Other protection	▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

'Forsberg Clothing Performance Index'.

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

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Material	CPI
BUTYL	A
NEOPRENE	A
VITON	A
BUTYL/NEOPRENE	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
PVC	C
TEFLON	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the 'Exposure Standard' (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - Full-face

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

76ak-p()

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	clear, amber		
Physical state	Liquid	Relative density (Water = 1)	0.95
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	321
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	2300
Initial boiling point and boiling range (°C)	>93	Molecular weight (g/mol)	Not Available
Flash point (°C)	150	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available

Continued...

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Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	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	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales. Inhalation of epoxy resin amine hardener vapours (including polyamines and amine adducts) may produce bronchospasm and coughing episodes lasting days after cessation of the exposure. Even faint traces of these vapours may trigger an intense reaction in individuals showing 'amine asthma'. The literature records several instances of systemic intoxications following the use of amines in epoxy resin systems. Excessive exposure to the vapours of epoxy amine curing agents may cause both respiratory irritation and central nervous system depression. Signs and symptoms of central nervous system depression, in order of increasing exposure, are headache, dizziness, drowsiness, and incoordination. In short, a single prolonged (measured in hours) or excessive inhalation exposure may cause serious adverse effects, including death. Inhalation of quantities of liquid mist may be extremely hazardous, even lethal due to spasm, extreme irritation of larynx and bronchi, chemical pneumonitis and pulmonary oedema. Pulmonary absorption may lead to systemic toxicity affecting the cardiovascular and central nervous system. Inhalation of phenol and some of its derivatives may produce profuse perspiration, intense thirst, nausea, vomiting, diarrhoea, cyanosis, hyperactivity, stupor, falling blood pressure, hyperpnoea, abdominal pain, haemolysis, convulsions, coma and pulmonary oedema with pneumonia. Respiratory failure and kidney damage may follow. Phenols may exhibit local anaesthetic properties and, in general, are central nervous system depressants at high concentrations. The dihydroxy derivatives act as simple phenols but their effects are largely limited to local irritation. Trihydroxy derivatives may reduce the oxygen content of blood at sufficient exposure levels. Methyl phenols (cresols) typically do not pose significant inhalation hazards due to relatively low vapour pressures and objectionable odours. Substituted phenols produce similar effects to phenol although such effects may only be evident at high levels of exposure. Alkyl substitution tends to increase toxicity.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation. Ingestion of amine epoxy-curing agents (hardeners) may cause severe abdominal pain, nausea, vomiting or diarrhoea. The vomitus may contain blood and mucus. If death does not occur within 24 hours there may be an improvement in the patients condition for 2-4 days only to be followed by the sudden onset of abdominal pain, board-like abdominal rigidity or hypo-tension; this indicates that delayed gastric or oesophageal corrosive damage has occurred. Some phenol derivatives may produce mild to severe damage within the gastrointestinal tract. Absorption may result in profuse perspiration, intense thirst, nausea, vomiting, diarrhoea, cyanosis (following the formation of methaemoglobin), hyperactivity, stupor, falling blood pressure, hyperaemia, abdominal pain, haemolysis, convulsions, coma and pulmonary oedema followed by pneumonia. Respiratory failure and kidney damage may follow. Severe phenol ingestions cause hypotension, coma, ventricular dysrhythmias, seizures and white coagulative chemical burns. Phenol does not uncouple oxidative phosphorylation like dinitrophenol and pentachlorophenol and thus does not cause a heat exhaustion-like syndrome. Phenolic groups with ortho and para positions free from substitution are reactive; this is because the ortho and para positions on the aromatic ring are highly activated by the phenolic hydroxyl group and are therefore readily substituted.

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Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material can produce severe chemical burns following direct contact with the skin. Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur. Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions. Individuals exhibiting 'amine dermatitis' may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis. NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided. Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep. Phenol and some of its derivatives may produce mild to severe skin irritation on repeated or prolonged contact, producing second and third degree chemical burns. Rapid cutaneous absorption may lead to systemic toxicity affecting the cardiovascular and central nervous system. Absorption through the skin may result in profuse perspiration, intense thirst, nausea, vomiting, diarrhoea, cyanosis (following the formation of methaemoglobin), hyperactivity, stupor, falling blood pressure, hyperpnoea, abdominal pain, haemolysis, convulsions, coma and pulmonary oedema followed by pneumonia. Respiratory failure and kidney damage may follow. 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	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight. Some phenol derivatives may produce mild to severe eye irritation with redness, pain and blurred vision. Permanent eye injury may occur; recovery may also be complete or partial. Vapours of volatile amines cause eye irritation with lachrymation, conjunctivitis and minor transient corneal oedema which results in 'halos' around lights (glauropsia, 'blue haze', or 'blue-grey haze'). Vision may become misty and halos may appear several hours after workers are exposed to the substance. This effect generally disappears spontaneously within a few hours of the end of exposure, and does not produce physiological after-effects. However oedema of the corneal epithelium, which is primarily responsible for vision disturbances, may take more than one or more days to clear, depending on the severity of exposure. Photophobia and discomfort from the roughness of the corneal surface also may occur after greater exposures. Although no detriment to the eye occurs as such, glauropsia predisposes an affected individual to physical accidents and reduces the ability to undertake skilled tasks such as driving a vehicle. Direct local contact with the liquid may produce eye damage which may be permanent in the case of the lower molecular weight species.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Secondary amines may react in the acid conditions of the stomach with oxidants or preservatives to form potentially carcinogenic N-nitrosamines. The formation of nitrosamines from such amines has not only been observed in animals models but, at least for certain compounds, in the workplace. The amine-containing substances and end products handled at work can themselves be contaminated to a degree with corresponding nitrosamines. Under conditions encountered in practice nitrosation is to be expected with secondary amines and to a limited extent with primary and tertiary amines. Nitrogen oxides are the most probable nitrosating agents. Nitrosyl chloride, nitrite esters, metal nitrites and nitroso compounds may also be involved. Several factors such as pH, temperature, catalysts and inhibitors influence the extent of nitrosation. Two precautionary measures are therefore necessary when handling amines at the workplace. - Simultaneous exposure to nitrosating agents should be reduced to minimum. This can be out into practice by eliminating nitrosating agents or, if they play a role in the actual process, replacing them with substances that do not lead to the formation of carcinogenic nitrosamines. In particular the level of nitrogen oxides at the workplace should be monitored and reduced when necessary. - The levels of nitrosamines in the workplace and in substances containing amines should be monitored. Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Report No. 31, DFG, 1995 In animal experiments the oesophagus is shown to be the most important target organ for nitrosamines, independent of the route of application. The mechanism of this organotrophy cannot be explained sufficiently. The high oesophageal epithelium metabolic activation of nitrosamines, together with a comparatively low DNA repair, probably plays the most important role. In addition chronic stress factors, which lead to high stimulation of epithelial turnover, are a pacemaker for malignant progression. In some countries, the traditional consumption of extremely hot drinks leads to constant burns of the oesophagus, which increases the risk. Mate, a non-alcoholic brew, frequently consumed as tea in Uruguay, appears to be a high risk factor for oesophageal cancer Prolonged exposure to some derivatives of phenol may produce dermatitis, anorexia, weight loss, weakness, muscle aches and pain, liver damage, dark urine, ochronosis, skin eruptions, diarrhoea, nervous disorders with headache, salivation, fainting, increased skin and scleral pigmentation, vertigo and mental disorders. Liver and kidney damage may also ensue. Chronic phenol toxicity was first noted in medical personnel in the late 1800s when 5 and 10% phenol was used as a skin disinfectant. The term carbolic (phenol) marasmus was given to this syndrome. Addition of structurally related phenolic compounds to the diet of Syrian golden hamsters induced forestomach hyperplasia and tumours. These compounds included 2(3)-tert-butyl-4-methoxyphenol (BHA) (CAS RN: 25013-16-5), 2-tert-butyl-4-methylphenol (TBMP) (29759-28-2) and p-tert-butylphenol (PTBP) (98-54-4); less active were catechol (154-23-4), p-methylphenol (331-39-5), methylhydroquinone (MHQ) (95-71-6) and pyrogallol (87-66-1), whilst no activity was seen with resorcinol (108-46-3), hydroquinone (123-31-9), propylparaben (94-13-3) and tert-butylhydroquinone (TBHQ) (1948-33-0). In autoradiographic studies, intake of BHA, TBMP, catechol, PMOP, PTBP and MHQ resulted in a significant increase in the labelling index of the forestomach epithelium, whilst PMOP induced epithelial damage and pyloric regenerative hyperplasia. Catechol, CA and PYMP induced similar but less marked alterations. Both catechol and PMOP increased the labelling index in the glandular stomach. The urinary bladder was free from histo-pathological lesions, but propylparabene, catechol, TBHQ and MHQ increased the labelling index. The authors of this study concluded that long term administration of PTBP and TBMP may be carcinogenic for hamster forestomach and that both 1-hydroxy and tert-butyl substituents may play a role in the induction of forestomach tumours. Hiros, M., et al: Carcinogenesis, Vol 7, pp 1285-1289; 1986 On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Repeated or prolonged exposure to acids may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will

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	be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). Asthmatics appear to be at particular risk for pulmonary effects.	
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	Not Available	Not Available
4-nonylphenol, branched	TOXICITY	IRRITATION
	Oral (rat) LD50: 1300 mg/kg ^[2]	Eye (rabbit): 100 mg - SEVERE
		Skin (rabbit): 500 mg/24h-SEVERE
tall oil/ tetraethylenepentamine polyamides	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg ^[2]	Eyes (rabbit) (-) moderate
		Skin (rabbit) (-) moderate
4,4'-methylenebis(2-methylcyclohexanamine)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 200 mg/kg ^[2]	Not Available
	Inhalation (rat) LC50: 0.42 mg/l/4h ^[2]	
	Oral (rat) LD50: 320 mg/kg ^[2]	
tetraethylenepentamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 660 mg/kg ^[2]	Eye (rabbit): 100 mg/24h moderate
	Oral (rat) LD50: 3990 mg/kg ^[2]	Eye (rabbit): 5 mg moderate
		Skin (rabbit): 495 mg SEVERE
		Skin (rabbit): 5 mg/24h SEVERE
naphtha petroleum, heavy alkylate	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >3.83 mg/l/4H ^[2]	Not Available
phenol	TOXICITY	IRRITATION
	dermal (rat) LD50: 525 mg/kg ^[1]	Eye(rabbit): 100 mg rinse - mild
	Inhalation (rat) LC50: 0.316 mg/l/4H ^[2]	Eye(rabbit): 5 mg - SEVERE
	Oral (rat) LD50: 317 mg/kg ^[2]	Skin(rabbit): 500 mg open -SEVERE
		Skin(rabbit): 500 mg/24hr - SEVERE

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

4-NONYLPHENOL, BRANCHED	Gastrointestinal changes, liver changes, effects on newborn recorded.
4,4'-METHYLENEBIS(2-METHYLCYCLOHEXANAMINE)	For 4,4'-methylenebis(2-methylcyclohexanamine) (DMD): Acute toxicity: In humans (epoxy resins production workers) scleroderma-like skin changes have been described revealing 4,4'-methylenebis(2-methylcyclohexanamine) as most probable causative agent. In DMD production workers unspecific skin changes, but no scleroderma-like symptoms were seen. DMD is harmful via the oral route and toxic via the dermal and inhalation route: LD50 rat (oral): > 320 < 460 mg/kg bw, symptoms: unspecific; LC50 rat (inhalation, liquid aerosol): 420 mg/m³/4h, symptoms: irritation of the airways; LD50 rabbit (dermal): > 200 < 400 mg/kg bw, symptoms: cyanosis, necrotic changes at the test site. The substance is highly corrosive to skin (full thickness necrosis after 3 minutes of exposure) and may cause severe damage to eyes. In the guinea pig maximization test the substance showed no sensitising effect. In a well conducted rat 90-day inhalation study (OECD TG 413) body weight development was impaired, local irritative effects observed for the skin and upper airways (nasal mucosa) and target organ toxicity indicative of a mild anaemic effect as well as effects on the liver, testes and kidneys were seen at 48 mg/m³. No histopathological correlate was found with respect to increased absolute lung weights. At 12 mg/m³ the only effect seen was an increase in GPT levels in males. The NOAEC was 2 mg/m³. Subchronic toxicity: The substance may cause local damage as well as systemic toxicity including histopathological changes in several target organs (damage to haematological system, liver, kidney, adrenal gland and heart) after repeated oral uptake and to a lesser extent after inhalative exposure as shown in animal studies. In a subchronic oral toxicity study with rats (OECD TG 408), the animals were exposed to 0, 2.5, 12 and 60 mg/kg bw/day by gavage over 3 months. Liver, white and red blood cells, kidneys, adrenal glands and heart were the target organs for toxic effect showing also histopathological alterations. At the high dose level (60 mg/kg bw/day) body weight development/food consumption were clearly impaired and the general state of health was poor. The absolute testes weight was decreased and an atrophy of the seminiferous tubuli and a reduced content of the seminal vesicle were noted. These changes were interpreted as consequence of the marked impairment on body weight. While the toxic effects at the mid dose of 12 mg/kg bw/day were generally less pronounced, a NOAEL was achieved at 2.5 mg/kg bw/day. Genotoxicity: The substance showed no genotoxic effects in the Ames test (OECD TG 471), cytogenetic assay with CHO cells (OECD TG 473) and

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	HGPRT assay (OECD TG 476) when tested up to the cyto-/bacteriotoxic range. Reproductive toxicity: In rat 90-day oral and inhalation studies the substance showed no direct adverse effects to the male and female reproductive organs (testes, ovaries and uterus examined). The observed effects on testes being a secondary nonspecific consequence of the severe systemic toxicity (e.g. decrease in body weight) seen at the same dose level. Developmental toxicity: In a developmental toxicity study (OECD TG 414) the DMD (0, 5, 15 or 45 mg/kg bw/day) was administered from day 6 to 19 post-coitum orally by gavage to rats. The NOAEL for maternal toxicity was 5 mg/kg bw/day. Slight foetotoxicity (retardation of ossification of skull bones) without teratogenicity was observed at 45 mg/kg bw/day, together with severely reduced body weight of the dams. The NOAEL for developmental toxicity was 15 mg/kg bw/day. The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence). The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties. * [BASF]
TETRAETHYLENEPENTAMINE	Triethylenetetramine (TETA) is a severe irritant to skin and eyes and induces skin sensitisation. TETA is of moderate acute toxicity: LD50(oral, rat) > 2000 mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure to saturated vapour via inhalation was tolerated without impairment. Exposure to aerosol leads to reversible irritations of the mucous membranes in the respiratory tract. Following repeated oral dosing via drinking water only in mice but not in rats at concentration of 3000 ppm there were signs of impairment. The NOAEL is 600 ppm [92 mg/kg bw (oral, 90 days)]. Lifelong dermal application to mice (1.2 mg/mouse) did not result in tumour formation. There are differing results of the genetic toxicity for TETA. The positive results of the in vitro tests may be the result of a direct genetic action as well as a result of an interference with essential metal ions. Due to this uncertainty of the in vitro tests, the genetic toxicity of TETA has to be assessed on the basis of in vivo tests. The in vivo micronucleus tests (i.p. and oral) and the SLRL test showed negative results. There are no human data on reproductive toxicity (fertility assessment). The analogue diethylenetriamine had no effects on reproduction. TETA shows developmental toxicity in animal studies if the chelating property of the substance is effective. The NOEL is 830 mg/kg bw (oral). Experience with female patients suffering from Wilson's disease demonstrated that no miscarriages and no foetal abnormalities occur during treatment with TETA.. In rats, there are several studies concerning developmental toxicity. The oral treatment of rats with 75, 375 and 750 mg/kg resulted in no effects on dams and fetuses, except slight increased fetal body weight. After oral treatment of rats with 830 or 1670 mg/kg bw only in the highest dose group increased foetal abnormalities in 27/44 fetus (69,2 %) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significantly the fetal abnormalities of the highest dose group to 3/51 (6,5 % foetus. These findings suggest that the developmental toxicity is produced as a secondary consequence of the chelating properties of TETA.
NAPHTHA PETROLEUM, HEAVY ALKYLATE	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption is known as the 'hydrocarbon continuum hypothesis', and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. for petroleum: This product contains 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.
PHENOL	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.
4-NONYLPHENOL, BRANCHED & PHENOL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
4-NONYLPHENOL, BRANCHED & TETRAETHYLENEPENTAMINE & PHENOL	The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
4-NONYLPHENOL, BRANCHED & TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & 4,4'-METHYLENEBIS(2-	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

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METHYLCYCLOHEXANAMINE) & TETRAETHYLENEPENTAMINE & PHENOL	bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TETRAETHYLENEPENTAMINE	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.
TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TETRAETHYLENEPENTAMINE	Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds. Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines. In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper
TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & 4,4'-METHYLENEBIS(2-METHYLCYCLOHEXANAMINE) & TETRAETHYLENEPENTAMINE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & 4,4'-METHYLENEBIS(2-METHYLCYCLOHEXANAMINE)	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TETRAETHYLENEPENTAMINE	For alkyl polyamines: The alkyl polyamines cluster consists of organic compounds containing two terminal primary amine groups and at least one secondary amine group. Typically these substances are derivatives of ethylenediamine, propylenediamine or hexanediamine. The molecular weight range for the entire cluster is relatively narrow, ranging from 103 to 232 Acute toxicity of the alkyl polyamines cluster is low to moderate via oral exposure and a moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin sensitizers in experimental animals. Repeated exposure in rats via the oral route indicates a range of toxicity from low to high hazard. Most cluster members gave positive results in tests for potential genotoxicity. Limited carcinogenicity studies on several members of the cluster showed no evidence of carcinogenicity. Unlike aromatic amines, aliphatic amines are not expected to be potential carcinogens because they are not expected to undergo metabolic activation, nor would activated intermediates be stable enough to reach target macromolecules. Polyamines potentiate NMDA induced whole-cell currents in cultured striatal neurons
TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TETRAETHYLENEPENTAMINE	Tetraethylenepentamine (TEPA) has a low acute toxicity when administered orally to rats (LD50 =3250 mg/kg). In an acute inhalation toxicity study with saturated vapor and whole body exposure, the LC50 was calculated to be >9.9 ppm (highest dose tested). TEPA is corrosive to the skin and eyes of rabbits. TEPA is a skin sensitizer in the guinea pig. Dermal acute toxicity LD50 values in the rabbit range from 660 - 1260 mg/kg. The higher toxicity via the dermal route is most likely due to the corrosive nature of TEPA to the skin whereas TEPA would be neutralized by stomach acid. The results of a 28-day repeated dose dermal toxicity study of TEPA indicated a systemic toxicity NOEL of 200 mg/kg/day and a dermal toxicity NOEL (local) of 50 mg/kg/day. The dermal LOAEL was 100 mg/kg/day. In addition, in a repeat dose study of TETA administered in drinking water to male and female rats for 90-92 days, the NOEL was 276 mg/kg/day in males and 352 mg/kg/day in females, the highest dose administered with the NIH-31 diet (several diets were used to study the effects of copper deficiency versus toxicity directly to TEPA). In this same study in mice the NOEL was 487 mg/kg/day in males and 551 mg/kg/day in females, the highest dose administered. A lifetime study was conducted via dermal administration in fifty male mice with a solution of 35% TEPA. There were 20 cases of hyperkeratosis, 13 cases of epidermal necrosis and no evidence of dermal hyperplasia. There were no data available for TEPA for reproductive and developmental toxicity. As a result, data on triethylenetetramine (TETA) was used to address these endpoints. TETA data showed no effects on reproductive organs in rats up to 276 mg/kg/day (males) and 352 mg/kg/day (females) and in mice (up to 500 mg/kg/day) when administered in drinking water. TETA was not considered a developmental toxicant via dermal administration in rabbits at maternally toxic doses up to 125 mg/kg/day but showed developmental toxicity in rats at maternally toxic doses of 830 or 1660 mg/kg/day via drinking water. The maternal and foetal toxicity was most likely due to copper deficiency and zinc toxicity at these levels. Subsequent studies where the diet was supplemented with copper resulted in a decrease of foetal abnormalities. There were no standard fertility studies available. However, there were no effects on the gonads observed in a 90-day drinking water study in rats and mice as described above. In the Ames Salmonella assay, TEPA was found to be positive both with and without metabolic activation. TEPA was found to increase sister chromatid exchange in CHO cells and was considered positive in a UDS assay using rat hepatocytes. TEPA was not considered genotoxic in the mouse micronucleus assay and had equivocal results in the two dominant lethal assays in Drosophila melanogaster. Again, it is believed that the positive results are based upon TEPA's ability to chelate copper.

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 available but does not fill the criteria for classification
✓ – Data available to make classification
⊘ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Continued...

832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)

12.1. Toxicity

832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

4-nonylphenol, branched	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.017mg/L	2
	EC50	48	Crustacea	0.0844mg/L	2
	EC50	96	Algae or other aquatic plants	0.027mg/L	2
	BCF	24	Fish	0.193mg/L	4
	EC10	96	Algae or other aquatic plants	0.012mg/L	4
	NOEC	672	Fish	>0.0019mg/L	2

tall oil/ tetraethylenepentamine polyamides	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.19mg/L	2
	EC50	48	Crustacea	0.18mg/L	2

4,4'-methylenebis(2-methylcyclohexanamine)	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	21.5mg/L	1
	EC50	48	Crustacea	≈15.2mg/L	1
	EC50	96	Algae or other aquatic plants	≈1.6mg/L	1
	EC10	96	Algae or other aquatic plants	≈0.41mg/L	1
	NOEC	72	Algae or other aquatic plants	0.13mg/L	2

tetraethylenepentamine	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	48	Crustacea	≈24.1mg/L	1
	EC50	72	Algae or other aquatic plants	≈2.1mg/L	1
	NOEC	72	Algae or other aquatic plants	≈0.5mg/L	1

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

phenol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.00175mg/L	4
	EC50	48	Crustacea	≈3.1mg/L	1
	EC50	96	Algae or other aquatic plants	0.0611mg/L	4
	BCF	24	Fish	60mg/L	4
	EC10	0.5	Algae or other aquatic plants	0.076mg/L	4
	NOEC	144	Crustacea	0.01mg/L	4

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

Environmental fate:

DMD has a water solubility of 3.6 g/l, a vapour pressure of 0.08 Pa and a measured log Kow of 2.51. However, due to the Lewis base character of the substance the experimental determination of the log Kow is inaccurate.

From the physico-chemical properties the hydrosphere is identified as target compartment for the substance.

Biodegradability: <10% DOC Reduction (OECD 302B/Iso 9888/EEC 88/302.C)

According to OECD criteria the substance is not biodegradable even with adapted inoculum (OECD TG 302B <1 % after 28 days) and can only be poorly eliminated in sewage water treatment plants. Due to the chemical structure of DMD hydrolysis is not likely to occur under environmental conditions.

In the atmosphere the substance is quickly degraded by photochemical attack (half life ≈3.1 hours). The log Koc was calculated to 3.26. It has to be considered however, that as a basic compound cyclohexylamine can additionally be bound to the soil by ion exchange.

Ecotoxicity:

DMD is considered as toxic to aquatic organisms

Fish LC50 (96 h): *Leusiscus idus* >22<46 mg/l

Daphnia magna EC50 (48 h): 15.2 mg/l

Green alga *ErC50* (72 h): *Scenedesmus subspicatus* > 5 mg/l; *Ebc50* 2.1 mg/l

832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)

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.).

for alkylphenols and their ethoxylates, or propoxylates:

Environmental fate: Alkylphenols are ubiquitous in the environment after the introduction, generally as wastes, of their alkoxylated forms (ethoxylates and propoxylates, for example); these are extensively used throughout industry and in the home.

Alkylphenol ethoxylates are widely used surfactants in domestic and industrial products, which are commonly found in wastewater discharges and in sewage treatment plant (STP) effluents.

Degradation of APES in wastewater treatment plants or in the environment generates more persistent shorter-chain APES and alkylphenols (APs) such as nonylphenol (NP), octylphenol (OP) and AP mono- to triethoxylates (NPE1, NPE2 and NPE3). There is concern that APE metabolites (NP, OP, NPE1-3) can mimic natural hormones and that the levels present in the environment may be sufficient to disrupt endocrine function in wildlife and humans. The physicochemical properties of the APE metabolites (NP, NPE1-4, OP, OPE1-4), in particular the high Kow values, indicate that they will partition effectively into sediments following discharge from STPs. The aqueous solubility data for the APE metabolites indicate that the concentration in water combined with the high partition coefficients will provide a significant reservoir (load) in various environmental compartments. Data from studies conducted in many regions across the world have shown significant levels in samples of every environmental compartment examined. In the US, levels of NP in air ranged from 0.01 to 81 ng/m³, with seasonal trends observed. Concentrations of APE metabolites in treated wastewater effluents in the US ranged from < 0.1 to 369 ug/l, in Spain they were between 6 and 343 ug/l and concentrations up to 330 ug/l were found in the UK. Levels in sediments reflected the high partition coefficients with concentrations reported ranging from < 0.1 to 13,700 ug/kg for sediments in the US. Fish in the UK were found to contain up to 0.8 ug/kg NP in muscle tissue. APES degraded faster in the water column than in sediment. Aerobic conditions facilitate easier further biotransformation of APE metabolites than anaerobic conditions.

Nonylphenols are susceptible to photochemical degradation. Using natural, filtered, lake water it was found that nonylphenol had a half-life of approximately 10-15 h under continuous, noon, summer sun in the surface water layer, with a rate approximately 1.5 times slower at depths 20-25 cm. Photolysis was much slower with ethoxylated nonylphenol, and so it is unlikely to be a significant event in removal of the ethoxylates.

Air: Alkylphenols released to the atmosphere will exist in the vapour phase and is thought to be degraded by reaction with photochemically produced hydroxyl radicals, with a calculated half-life, for nonylphenol, of 0.3 days.

Water: Abiotic degradation of alkylphenol is negligible. Biodegradation does not readily take place. The half-life in surface water may be around 30 days.

Degradation: Alkylphenol ethoxylates (APES) may abiotically degrade into the equivalent alkylphenol. During degradation ethylene oxide units are cleaved off the ethylene oxide chain until only short-chain alkylphenol ethoxylates remain, typically mono- and diethylene oxides. Oxidation of these oligomers creates the corresponding carboxylic acids. This leaves several degradation products: short-chain ethoxylates, their carboxylic acids, and alkylphenols.

Biodegradation: Alkylphenols are not readily biodegradable. Several mechanisms of microbial aromatic ring degradation have been reported, the most common being formation of catechol from phenol, followed by ring scission between or adjacent to the two hydroxyl groups.

The full breakdown pathway for APES has not yet been determined, and all studies have so far focused on identification of intermediates in bacterial culture media, rather than studying cell-free systems or purified enzymes. It is, however, likely that microbial metabolism usually starts by an attack on the ethoxylate chain, rather than on the ring or the hydrophobic chain. The ethoxylate groups are progressively removed, either by ether cleavage, or by terminal alcohol oxidation followed by cleavage of the resulting carboxylic acid.

Biodegradation of APES produces less biodegradable products: alkylphenol mono- and di-ethoxylates, alkylphenoxy acetic and alkylphenoxypolyethoxy acetic acids, and alkylphenols. These metabolites frequently persist through sewage treatment and in rivers. Anaerobic conditions generally lead to the accumulation of alkylphenols. The rate of biodegradation seems to decrease with increasing length of the ethylene oxide chain.

Bioaccumulation: Metabolites of APES accumulate in organisms, with bioconcentration factors varying from ten to several thousand, depending on species, metabolite and organ.

The metabolites of APES are generally more toxic than the original compounds. APES have LC50s above about 1.5 mg/l, whereas alkylphenols, such as nonylphenol, have LC50s are generally around 0.1 mg/l.

Oestrogenic activity: The role of alkyl chain length and branching, substituent position, number of alkylated groups, and the requirement of a phenolic ring structure was assessed in fish. The results showed that most alkylphenols were oestrogenic, although with 3-300 thousand times lower potency than the endogenous estrogen 17β-estradiol. Mono-substituted tertiary alkylphenols with moderate (C4-C5) and long alkyl chain length (C8-C9) in the para position exhibited the highest oestrogenic potency. Substitution with multiple alkyl groups, presence of substituents in the ortho- and meta-position and lack of a hydroxyl group on the benzene ring reduced the oestrogenic activity, although several oestrogenic alkylated non-phenolics were identified.

Human exposure: Alkylphenols were first found to be oestrogenic (oestrogen-mimicking) in the 1930s, but more recent research has highlighted the implications of these effects. The growth of cultured human breast cancer cells is affected by nonylphenol at concentrations as low as 1 uM (220 ug/l) or concentrations of octylphenol as low as 0.1 uM (20 ug/l). Oestrogenic effects have also been shown on rainbow trout hepatocytes, chicken embryo fibroblasts and a mouse oestrogen receptor.

The insecticide chlordecone (Kepone) shows similar behaviour to alkylphenols, accumulating in liver and adipose tissue, and eliciting oestrogenic activity. Workers exposed to this insecticide can suffer reproductive effects such as low sperm counts and sterility. In addition, the oestrogenic effects of chlordecone on MCF7 cells occur at similar concentrations to those of alkylphenols, suggesting that alkylphenols will be a similar health hazard if target cells are exposed to uM levels of these compounds.

By comparing environmental concentrations, bioconcentration factors and *in vitro* oestrogenic effect levels, current environmental levels of alkylphenolic compounds are probably high enough to affect the hormonal control systems of some organisms. It is also possible that human health could be being affected.

Prevent, by any means available, spillage from entering drains or water courses.

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
4-nonylphenol, branched	HIGH	HIGH
4,4'-methylenebis(2-methylcyclohexanamine)	HIGH	HIGH
tetraethylenepentamine	LOW	LOW
phenol	LOW (Half-life = 10 days)	LOW (Half-life = 0.95 days)

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
4-nonylphenol, branched	LOW (BCF = 271)
4,4'-methylenebis(2-methylcyclohexanamine)	LOW (BCF = 60)
tetraethylenepentamine	LOW (LogKOW = -3.1604)
phenol	LOW (BCF = 17.5)

12.4. Mobility in soil

Ingredient	Mobility
4-nonylphenol, branched	LOW (KOC = 56010)
4,4'-methylenebis(2-methylcyclohexanamine)	LOW (KOC = 1838)
tetraethylenepentamine	LOW (KOC = 1098)
phenol	LOW (KOC = 268)

12.5. Results of PBT and vPvB assessment

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	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT Criteria fulfilled?	Not Available	Not Available	Not Available

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

SECTION 14 TRANSPORT INFORMATION

Labels Required

	 Limited Quantity: 832HD-25ML, 832HD-50ML, 832HD-400ML, 832HD-1.7L
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Land transport (ADR)

14.1.UN number	1760										
14.2.UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains tetraethylenepentamine and 4-nonylphenol, branched)										
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>Class</td><td>8</td></tr> <tr> <td>Subrisk</td><td>Not Applicable</td></tr> </table>	Class	8	Subrisk	Not Applicable						
Class	8										
Subrisk	Not Applicable										
14.4.Packing group	II										
14.5.Environmental hazard	Environmentally hazardous										
14.6. Special precautions for user	<table border="1"> <tr> <td>Hazard identification (Kemler)</td><td>80</td></tr> <tr> <td>Classification code</td><td>C9</td></tr> <tr> <td>Hazard Label</td><td>8</td></tr> <tr> <td>Special provisions</td><td>274</td></tr> <tr> <td>Limited quantity</td><td>1 L</td></tr> </table>	Hazard identification (Kemler)	80	Classification code	C9	Hazard Label	8	Special provisions	274	Limited quantity	1 L
Hazard identification (Kemler)	80										
Classification code	C9										
Hazard Label	8										
Special provisions	274										
Limited quantity	1 L										

Air transport (ICAO-IATA / DGR)

14.1. UN number	1760						
14.2. UN proper shipping name	Corrosive liquid, n.o.s. * (contains tetraethylenepentamine and 4-nonylphenol, branched)						
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>ICAO/IATA Class</td><td>8</td></tr> <tr> <td>ICAO / IATA Subrisk</td><td>Not Applicable</td></tr> <tr> <td>ERG Code</td><td>8L</td></tr> </table>	ICAO/IATA Class	8	ICAO / IATA Subrisk	Not Applicable	ERG Code	8L
ICAO/IATA Class	8						
ICAO / IATA Subrisk	Not Applicable						
ERG Code	8L						
14.4. Packing group	II						
14.5. Environmental hazard	Environmentally hazardous						

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14.6. Special precautions for user	Special provisions	A3 A803
	Cargo Only Packing Instructions	855
	Cargo Only Maximum Qty / Pack	30 L
	Passenger and Cargo Packing Instructions	851
	Passenger and Cargo Maximum Qty / Pack	1 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y840
	Passenger and Cargo Limited Maximum Qty / Pack	0.5 L

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1760	
14.2. UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains tetraethylenepentamine and 4-nonylphenol, branched)	
14.3. Transport hazard class(es)	IMDG Class	8
	IMDG Subrisk	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-A , S-B
	Special provisions	274
	Limited Quantities	1 L

Inland waterways transport (ADN)

14.1. UN number	1760	
14.2. UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains tetraethylenepentamine and 4-nonylphenol, branched)	
14.3. Transport hazard class(es)	8	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Classification code	C9
	Special provisions	274
	Limited quantity	1 L
	Equipment required	PP, EP
	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

4-NONYLPHENOL, BRANCHED(84852-15-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances	European Customs Inventory of Chemical Substances ECICS (English)
EU REACH Regulation (EC) No 1907/2006 - Proposals to identify Substances of Very High Concern: Annex XV reports for commenting by Interested Parties	European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
Europe AeroSpace and Defence Industries Association of Europe (ASD) REACH Implementation Working Group Priority Declarable Substances List (PDSL)	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
Europe European Chemicals Agency (ECHA) Candidate List of Substances of Very High Concern for Authorisation	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

TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES(68953-36-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)

4,4'-METHYLENEBIS(2-METHYLCYCLOHEXANAMINE)(6864-37-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 European Customs Inventory of Chemical Substances ECICS (English)
 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

TETRAETHYLENEPENTAMINE(112-57-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

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

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

PHENOL(108-95-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 European Customs Inventory of Chemical Substances ECICS (English)
 European List of Notified Chemical Substances (ELINCS)
 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) Third List of Indicative Occupational Exposure Limit Values (IOELVs) (English)
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
 UK Workplace Exposure Limits (WELs)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments

15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (phenol; 4-nonylphenol, branched; tetraethylenepentamine; 4,4'-methylenebis(2-methylcyclohexanamine); naphtha petroleum, heavy alkylate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (4-nonylphenol, branched; tall oil/ tetraethylenepentamine polyamides; naphtha petroleum, heavy alkylate)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = 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**Full text Risk and Hazard codes**

H226	Flammable liquid and vapour.
H290	May be corrosive to metals.
H301	Toxic if swallowed.
H304	May be fatal if swallowed and enters airways.
H311	Toxic in contact with skin.
H331	Toxic if inhaled.
H336	May cause drowsiness or dizziness.
H360	May damage fertility or the unborn child.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.

Other information**Ingredients with multiple cas numbers**

832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)

Name	CAS No
tall oil/ tetraethylenepentamine polyamides	68513-05-3, 68953-36-6, 68555-22-6, 1226892-45-0

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-1.02 - Update to the emergency phone number information.