

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	Hearne 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)









SIGNAL WORD

DANGER

Hazard statement(s)

H302	Harmful if swallowed.			
H312	armful in contact with skin.			
H332	armful if inhaled.			
H314	uses 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.			

H410 Very toxic to aquatic life with long lasting effects.

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	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	P273 Avoid release to the environment.		
P272	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	P308+P313 IF exposed or concerned: Get medical advice/ attention.			
P310 Immediately call a POISON CENTER/doctor/physician/first aider.				
P302+P352	P302+P352 IF ON SKIN: Wash with plenty of water and soap.			
P363	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	4-nonylphenol, branched	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	tall oil/ tetraethylenepentamine polyamides	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	4,4'-methylenebis(2- methylcyclohexanamine)	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]

1.112-57-2 2.203-986-2 3.612-060-00-0 4.Not Available	3	tetraethylenepentamine	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	naphtha petroleum, heavy alkylate	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	phenol	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:	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 furnes 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, furnes) 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

INGESTION:

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.
- Pamage 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.

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]

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]
- Figure 1 (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.
- Filter (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 Fnd 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
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

- Slight fire hazard when exposed to heat or flame.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO). May emit acrid smoke.
- Mists containing combustible materials may be explosive.

Combustion products include:

carbon dioxide (CO2)

nitrogen oxides (NOx)

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

 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.

▶ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.

Minor Spills

- 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

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

R.W. SS cross-linked polymer - particulate 1 blower skiploader 2 R, DGC, RT cross-linked polymer - pillow throw skiploader 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

Safe handling

7.1. Precautions for 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.
- 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

Other information

See section 5

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.
- 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.

Continued...

7.2. Conditions for safe storage, including any incompatibilities

- ▶ 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

Suitable container

- 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/m3 / 2 ppm	16 mg/m3 / 4 ppm	Not Available	Sk
European Union (EU) Third List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	phenol	Phenol	8 mg/m3 / 2 ppm	16 mg/m3 / 4 ppm	Not Available	skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	phenol	Phenol	7.8 mg/m3 / 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/m3	2.3 mg/m3	260 mg/m3
4,4'-methylenebis(2- methylcyclohexanamine)	Laromin C 260; (bis(4-Amino-3-methylcyclohexyl) methane; Dimethyldicyane)	0.28 mg/m3	3.1 mg/m3	19 mg/m3
tetraethylenepentamine	Tetraethylenepentamine	15 mg/m3	130 mg/m3	790 mg/m3
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

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

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.

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

8.2.1. Appropriate engineering controls

Within each range the appropriate value depends on:

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

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

8.2.2. Personal protection











8.2.2. Personal 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.
- Learning 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.

Eye and 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

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-butatoluene 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:

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

Material	СРІ
BUTYL	Α
NEOPRENE	A
VITON	A
BUTYL/NEOPRENE	С
NAT+NEOPR+NITRILE	С
NATURAL RUBBER	С
NATURAL+NEOPRENE	С
NEOPRENE/NATURAL	С
NITRILE	С
PE/EVAL/PE	С
PVA	С
PVC	С
TEFLON	С
VITON/NEOPRENE	С

^{*} 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.

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

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

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

76ak-p()

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

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

Inhaled

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.

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

Ingestion of amine epoxy-curing agents (hardeners) may cause severe abdominal pain, nausea, vomiting or diarrhoea. The vomitus may contain blood and mucous. 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, hypernea, 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.

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

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 oederna 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.

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 (glaucopsia, '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, glaucopsia 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.

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 esophagus 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-methoxphenol (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

Eve

Chronic

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.

	lungs (symptoms and changes in pulmonary function). Asi	imatics appear to be	at particular risk for pairrionary	Circuis.
832HD Black 1:1 Epoxy Potting and Encapsulating Compound (Part B)	TOXICITY Not Available		IRRITATION Not Available	
4-nonylphenol, branched	TOXICITY Oral (rat) LD50: 1300 mg/kg ^[2]	Eye	TATION (rabbit): 100 mg - SEVERE (rabbit): 500 mg/24h-SEVERE	
tall oil/ tetraethylenepentamine polyamides	TOXICITY Oral (rat) LD50: >5000 mg/kg ^[2]		IRRITATION Eyes (rabbit) (-) modera Skin (rabbit) (-) modera	
4,4'-methylenebis(2- methylcyclohexanamine)	TOXICITY Dermal (rabbit) LD50: 200 mg/kg ^[2] Inhalation (rat) LC50: 0.42 mg/l/4h* ^[2] Oral (rat) LD50: 320 mg/kg ^[2]			IRRITATION Not Available

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.

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.

4,4'-METHYLENEBIS(2-METHYLCYCLOHEXANAMINE)

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

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/dav.

The material may produce respiratory tract irritation, Symptoms of pulmonary irritation may include coughing, wheezing, larvngitis, 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.

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 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. **TETRAETHYLENEPENTAMINE**

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 significant 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.

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

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

for petroleum:

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

NAPHTHA PETROLEUM. **HEAVY ALKYLATE**

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 coniunctivitis

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

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

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(2METHYLCYCLOHEXANAMINE) & TETRAETHYLENEPENTAMINE

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

TETRAETHYLENEPEATH 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 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 sensitisers 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

allergic test reaction in more than 1% of the persons tested.

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 sensitiser 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	0
Skin Irritation/Corrosion	✓	Reproductivity	✓
Serious Eye Damage/Irritation	0	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	0
Mutagenicity	✓	Aspiration Hazard	0

Legend:

X - Data available but does not fill the criteria for classification

Data available to make classification

O - Data Not Available to make classification

12.1. Toxicity

2HD Black 1:1 Epoxy Potting d Encapsulating Compound	ENDPOINT TEST DURATION (HR)			SPECIES	VALUE		SOUF	RCE	
(Part B)	Not Available		Not Available		Not Available	Not Avai	lable	Not A	/ailable
	FNIDDOINIT	750	T DUD ATION (UD)	ODEO(E)			\/A111E		SOURCE
	ENDPOINT TEST DURATION (HR)		SPECIES	•		VALUE			
	LC50	96		Fish	_		0.017mg/L		2
A manufabanal basabad	EC50	48		Crustace			0.0844mg/L		2
4-nonylphenol, branched	EC50			-			0.027mg/L		2
	BCF	24		Fish			0.193mg/L		4
	EC10	96			other aquatic plants		0.012mg/L		4
	NOEC	672		Fish			>0.0019mg/L		2
all oil/ tetraethylenepentamine	ENDPOINT		TEST DURATION (HR)		SPECIES		LUE		JRCE
polyamides	LC50		96		Fish		19mg/L	2	
	EC50		48		Crustacea	0.1	I8mg/L	2	
	FNIDDOINT	TE	CT DUD ATION (UD)	CDECIE			VALUE		SOURCE
	ENDPOINT		ST DURATION (HR)	SPECIE	:5		VALUE 21.5mg/L		
	LC50	96			Fish				1
4,4'-methylenebis(2- methylcyclohexanamine)			48		Crustacea		=15.2mg/L		1
mennyicycionexanamine)	EC50				Algae or other aquatic plants		=1.6mg/L		1
	EC10	96			r other aquatic plants		=0.41mg/L		1
	NOEC	72		Algae o	r other aquatic plants	i	0.13mg/L		2
	ENDPOINT	TES	ST DURATION (HR)	SPECIE	·s		VALUE		SOURCE
	EC50	48	or borranore (int)	Crustac			=24.1mg/L		1
tetraethylenepentamine	EC50	72						•	1
	NOEC	72			r other aquatic plants		=2.1mg/L =0.5mg/L		1
	NOEC	12		Algae o	r other aquatic plants	•	=0.5mg/L		<u>'</u>
	ENDPOINT	TE	ST DURATION (HR)	SPECI	ES		VALUE		SOURCE
naphtha petroleum, heavy	EC50	72	. ,	Algae o	or other aquatic plant	s	=13mg/L		1
alkylate	NOEC	72			or other aquatic plant		=0.1mg/L		1
	ENDPOINT	TES	T DURATION (HR)	SPECIES	3		VALUE		SOURCE
	LC50	96		Fish			0.00175mg/L		4
	EC50	48		Crustace	а		=3.1mg/L		1
phenol	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		Crustace	a		0.01mg/L		4

(Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

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

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 environmental 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 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 17beta-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 (20 ug/1) or concentrations of octylphenol as low as 0.1 uM (20 ug/1). 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)	нівн	нівн
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

	P	В	Т
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

► Containers may still present a chemical hazard/ danger when empty. ► Return to supplier for reuse/ recycling if possible. Otherwise: Fig 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. Product / Packaging 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. Freatment 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

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)	Class 8 Subrisk Not Applicable	
14.4.Packing group	II	
14.5.Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Classification code C Hazard Label 8 Special provisions 2	30 C9 3 274

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)	ICAO/IATA Class ICAO / IATA Subrisk ERG Code	8 Not Applicable 8L	
14.4. Packing group	II		
14.5. Environmental hazard	Environmentally hazard	us	

14.6. Special precautions for

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		
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	
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
EU REACH Regulation (EC) No 1907/2006 - Proposals to identify Substances of Very
High Concern: Annex XV reports for commenting by Interested Parties
Europe AeroSpace and Defence Industries Association of Europe (ASD) REACH
Implementation Working Group Priority Declarable Substances List (PDSL)
Europe European Chemicals Agency (ECHA) Candidate List of Substances of Very
High Concern for Authorisation

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

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

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	Υ	
Canada - DSL	Υ	
Canada - NDSL	N (phenol; 4-nonylphenol, branched; tetraethylenepentamine; 4,4'-methylenebis(2-methylcyclohexanamine); naphtha petroleum, heavy alkylate)	
China - IECSC	Υ	
Europe - EINEC / ELINCS / NLP	Y	
Japan - ENCS	N (4-nonylphenol, branched; tall oil/ tetraethylenepentamine polyamides; naphtha petroleum, heavy alkylate)	
Korea - KECI	Υ	
New Zealand - NZIoC	Y	
Philippines - PICCS	Υ	
USA - TSCA	Υ	
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

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

 ${\sf PC-TWA} : {\sf Permissible\ Concentration-Time\ Weighted\ Average}$

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.