Hychem Hyflex NS Hardener

Hychem International

Chemwatch Hazard Alert Code: **3**

Chemwatch: **22-7440**
Version No: **5.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **10/12/2021**
Print Date: **09/03/2023**
L.GHS.AUS.EN

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

Product Identifier

Product name	Hychem Hyflex NS Hardener
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S.
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Hardener for coating systems.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Hychem International**
Address	Unit 1, 30 Bluett Drive Smeaton Grange NSW 2567 Australia
Telephone	+61 2 4646 1660
Fax	+61 2 4647 3700
Website	Not Available
Email	Not Available

Emergency telephone number

Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Flammable Liquids Category 2, Corrosive to Metals Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1A, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 4, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	

Signal word	**Danger**

Hazard statement(s)

H225	Highly flammable liquid and vapour.
H290	May be corrosive to metals.
H302	Harmful if swallowed.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H332	Harmful if inhaled.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P233	Keep container tightly closed.
P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P234	Keep only in original packaging.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use alcohol resistant foam or fine spray/water fog to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P390	Absorb spillage to prevent material damage.
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

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
25154-52-3	10-30	nonylphenol
25620-58-0	<10	trimethylhexamethylene diamine
61788-46-3	<10	cocoamine

Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*

SECTION 4 First aid measures

Description of first aid measures

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

Indication of any immediate medical attention and special treatment needed

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

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

Alkalis continue to cause damage after exposure.

INGESTION:

- Milk and water are the preferred diluents

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

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

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

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

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

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	Alert Fire Brigade and tell them location and nature of hazard. Wear 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	Liquid and vapour are highly flammable. Severe fire hazard when exposed to heat, flame and/or oxidisers. Vapour may travel a considerable distance to source of ignition. Heating may cause expansion or decomposition leading to violent rupture of containers.

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	‣ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material. May emit corrosive fumes.
HAZCHEM	2X

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ Remove all ignition sources. ‣ 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 small quantities with vermiculite or other absorbent material. ‣ Wipe up. ‣ Collect residues in a flammable waste container. ‣ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material. ‣ Check regularly for spills and leaks.

Major Spills

Chemical Class: amines, alkyl
For release onto land: recommended sorbents listed in order of priority.

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

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

NOTE:

- 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.

Chemical Class: phenols and cresols
For release onto land: recommended sorbents listed in order of priority.

Continued...

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

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
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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.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	**DO NOT** allow clothing wet with material to stay in contact with skin Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. Avoid contact with moisture. Avoid contact with incompatible materials. When handling, **DO NOT** eat, drink or smoke. Keep containers securely sealed when not in use. Avoid physical damage to containers. Always wash hands with soap and water after handling. Work clothes should be laundered separately. Launder contaminated clothing before re-use. Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	Store in approved flammable liquid storage area. No smoking, naked lights/ignition sources. Keep containers securely sealed.

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	- Store away from incompatible materials in a cool, dry, well-ventilated area. - Protect containers against physical damage and check regularly for leaks. - Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access. - Store in grounded, properly designed and approved vessels and away from incompatible materials - Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances. - Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems. - Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors. - Keep adsorbents for leaks and spills readily available - For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up; storage tanks should be above ground and diked to hold entire contents. - Observe manufacturer's storage and handling recommendations contained within this SDS. - **DO NOT** store near acids, or oxidising agents

Conditions for safe storage, including any incompatibilities

Suitable container	- 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	- Avoid reaction with oxidising agents - Avoid contact with copper, aluminium and their alloys. - 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, bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
nonylphenol	3.9 mg/m3	43 mg/m3	260 mg/m3

Ingredient	Original IDLH	Revised IDLH
nonylphenol	Not Available	Not Available
trimethylhexamethylene diamine	Not Available	Not Available
cocoamine	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit

Continued...

Ingredient	**Occupational Exposure Band Rating**	**Occupational Exposure Band Limit**
nonylphenol	E	≤ 0.1 ppm
trimethylhexamethylene diamine	E	≤ 0.1 ppm
cocoamine	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Exposure controls

Appropriate engineering controls

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

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Individual protection measures, such as personal protective equipment

Eye and face protection

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

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Skin protection	See Hand protection below
Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber ‣ 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. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ‣ Leather wear not recommended: Contaminated leather footwear, watch bands, should be destroyed, i.e. burnt, as they cannot be adequately decontaminated
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.

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:
Hychem Hyflex NS Hardener

Material	CPI
NEOPRENE	A
NITRILE	A

* CPI - Chemwatch Performance Index
A: Best Selection

Respiratory protection

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

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

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	AK-AUS / Class1 P2	-

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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.

up to 50	1000	-	AK-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	AK-2 P2
up to 100	10000	-	AK-3 P2
100+			Airline**

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Yellow paste.

Physical state	Liquid	**Relative density (Water = 1)**	0.97 @ 20C
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	~11 @ 20C	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	3000-5000 @ 25C
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	~120 (PMCC)	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	Unstable in the presence of incompatible materials. Product is considered stable. Hazardous polymerisation will not occur.

Continued...

Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Inhalation of epoxy resin amine hardener vapours (including polyamines and amine adducts) may produce bronchospasm and coughing episodes lasting days after cessation of the exposure. Even faint traces of these vapours may trigger an intense reaction in individuals showing "amine asthma". The literature records several instances of systemic intoxications following the use of amines in epoxy resin systems. Excessive exposure to the vapours of epoxy amine curing agents may cause both respiratory irritation and central nervous system depression. Signs and symptoms of central nervous system depression, in order of increasing exposure, are headache, dizziness, drowsiness, and incoordination. In short, a single prolonged (measured in hours) or excessive inhalation exposure may cause serious adverse effects, including death. Inhalation of amine vapours may cause irritation of the mucous membranes of the nose and throat and lung irritation with respiratory distress and cough. Single exposures to near lethal concentrations and repeated exposures to sublethal concentrations produces tracheitis, bronchitis, pneumonitis and pulmonary oedema. Aliphatic and alicyclic amines are generally well absorbed from the respiratory tract. Systemic effects include headache, nausea, faintness and anxiety. These effects are thought to be transient and are probably related to the pharmacodynamic action of the amines. Histamine release by aliphatic amines may produce bronchoconstriction and wheezing. Pulmonary absorption may lead to systemic toxicity affecting the cardiovascular and central nervous system. Inhalation of phenol and some of its derivatives may produce profuse perspiration, intense thirst, nausea, vomiting, diarrhoea, cyanosis, hyperactivity, stupor, falling blood pressure, hyperpnoea, abdominal pain, haemolysis, convulsions, coma and pulmonary oedema with pneumonia. Respiratory failure and kidney damage may follow. Phenols may exhibit local anaesthetic properties and, in general, are central nervous system depressants at high concentrations. The dihydroxy derivatives act as simple phenols but their effects are largely limited to local irritation. Trihydroxy derivatives may reduce the oxygen content of blood at sufficient exposure levels. Methyl phenols (cresols) typically do not pose significant inhalation hazards due to relatively low vapour pressures and objectionable odours. Substituted phenols produce similar effects to phenol although such effects may only be evident at high levels of exposure. Alkyl substitution tends to increase toxicity.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. The material can produce chemical burns within the oral cavity and gastrointestinal tract following 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. Aliphatic and alicyclic amines are generally well absorbed from the gut. Corrosive action may cause tissue damage throughout the gastrointestinal tract. Detoxification is thought to occur in the liver, kidney and intestinal mucosa with the enzymes, monoamine oxidase and diamine oxidase (histaminase) having a significant role. 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	The material can produce chemical burns following direct contact with the skin. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. 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

Continued...

	the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided. Volatile amine vapours produce primary skin irritation and dermatitis. Direct local contact, with the lower molecular weight liquids, may produce skin burns. Percutaneous absorption of simple aliphatic amines is known to produce lethal effects often the same as that for oral administration. Cutaneous sensitisation has been recorded chiefly due to ethyleneamines. Histamine release following exposure to many aliphatic amines may result in "triple response" (white vasoconstriction, red flare and wheal) in human skin. Phenol and some of its derivatives may produce mild to severe skin irritation on repeated or prolonged contact, producing second and third degree chemical burns. Rapid cutaneous absorption may lead to systemic toxicity affecting the cardiovascular and central nervous system. Absorption through the skin may result in profuse perspiration, intense thirst, nausea, vomiting, diarrhoea, cyanosis (following the formation of methaemoglobin), hyperactivity, stupor, falling blood pressure, hyperpnoea, abdominal pain, haemolysis, convulsions, coma and pulmonary oedema followed by pneumonia. Respiratory failure and kidney damage may follow. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation. 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. 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.
Chronic	Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. 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. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. 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. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or 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. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.

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Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.
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

	TOXICITY	IRRITATION
Hychem Hyflex NS Hardener	Not Available	Not Available
nonylphenol	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (rabbit): 0.5 mg (open)-SEVERE
	Oral (Rat) LD50: 1000-2500 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 500 mg(open)-mod
		Skin(rabbit):10mg/24h(open)-SEVERE
		Skin: adverse effect observed (corrosive)[1]
trimethylhexamethylene diamine	Oral (Rat) LD50: 910 mg/kg[2]	Eye (rabbit): Corrosive *Sensitiser ** [* = Manufacturer CG] [** = Manufacturer Degussa]
		Skin (rabbit): Corrosive *
cocoamine	dermal (rat) LD50: >2000 mg/kg[2]	Nil reported [AKZO] Corrosive (Eye) Corrosive (Skin) [ICI]
	Oral (Rat) LD50: 1300 mg/kg[2]	

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

NONYLPHENOL

For nonylphenol and its compounds:
Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor),. Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogeous hormone for binding with the estrogen receptors ERalpha and ERbeta.
Effects in pregnant women.
Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These “low doses” ranged from 10-13-10-9 M, which is lower than what is generally found in the environment.
Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications.
Effects on metabolism
Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol

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	because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats. Cancer Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.
TRIMETHYLHEXAMETHYLENE DIAMINE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
COCOAMINE	For Fatty Nitrogen-Derived ether amines and Fatty Nitrogen-derived amines (FND ether amines and FND amines): FND ether amines and FND amines are very similar in structure and function. . The minimal difference among the alkyl substituents and the large database for the FND categories indicates that the structural differences in these large alkyl chains do not result in differences in toxicity or mutagenicity. The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals The available acute oral LD50 study for the propanamine derivative with the extensive data for the other supporting chemicals provides adequate evidence that the FND ether amines are only moderately to slightly toxic via this route and exposure period. Acute dermal studies for the supporting chemicals indicate these chemicals can be classified as minimally toxic. Acute inhalation studies did not result in deaths under normal exposure conditions for two chemicals. Repeated dose toxicity studies had similar NOAELs (12.5 to 50 mg/kg/day for rats and 3 or 13 mg/kg/day for dogs). Importantly because the highest exposure potential for some of the FND ether amines is via skin contact, a number of repeat dose dermal studies indicate the chemicals are highly irritating. No clear organ-specific toxicity occurred in any of the repeat dose studies with the supporting chemicals in the FND ether amines category. In addition, available data indicate that the FND ether amines are unlikely to be mutagenic and that they are not reproductive or developmental toxins In evaluating potential toxicity of the FND Amines chemicals, it is also useful to review the available data for the related FND Cationic and FND Amides Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (*in vitro* bacterial and mammalian cells as well as *in vivo* studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
Hychem Hyflex NS Hardener & TRIMETHYLHEXAMETHYLENE DIAMINE & COCOAMINE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
Hychem Hyflex NS Hardener & NONYLPHENOL	for alkylphenolics category: The alkylphenolics may be divided into three groups. Group I: ortho-substituted mono-alkylphenols: Group II para-substituted mono-alkylphenols Group III: di- and tri-substituted mixed alkyl phenols

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	The subdivision of the category alkylphenols into *ortho*, *para* and the di/tri-substituted mixed members is supported by several published investigations. In assessing antimicrobial and antifouling activity of twenty-three alkylphenols, a significant difference was noted between *para* and *ortho*-substituted materials. In particular, biological activity was found to vary parabolically with increasing hydrophobicity of the *para*-substituent while introduction of a bulky substituent at the *ortho*-position resulted in a very significant decrease in antimicrobial, antifouling, and membrane-perturbation potency. Several alkylphenolic analogs of butylated hydroxytoluene (BHT) were examined for hepatotoxicity in mice depleted of hepatic glutathione. The structural requirement of both hepatic and pulmonary toxicity was a phenol ring having benzylic hydrogen atoms at the para position and an ortho-alkyl group(s) that moderately hinders the phenolic hydroxyl group. It is noteworthy that in this model, neither of the Group III members TTBP (2,4,6-tri-tert-butylphenol) nor 2,6-DTBP (2,6-di-tert-butylphenol) showed either hepatic or pulmonary toxicity. Lastly, important differences were observed in gene activation (recombinant yeast cell assay – Lac-Z reporter gene) between *ortho*-substituted and *para*-substituted alkylphenol **Acute toxicity:** The acute (single-dose) toxicity of alkylphenols examined to date shows consistency, with LD50 values ranging from approximately 1000 mg/kg to over 2000 mg/kg. These data demonstrate a very low level of acute systemic toxicity and do not suggest any unique structural specificity, despite the general tendency for the chemicals to be, at least, irritants to skin **Repeat dose toxicity**: The available studies for members drawn from the three groups range from 28-day and 90-day general toxicity studies, through developmental toxicity and reproductive/developmental screening, to multigeneration reproductive studies are available for some category members For the overall category of alkylphenols, the dosage at which the relatively mild general toxicity appears tends only to fall below 100 mg/kg/day with extended treatment, with an overall NOAEL for the category of approximately 20 mg/kg/day. No unusual and no apparent structurally unique toxicity is evident Repeat dose studies on OTBP (o-tert-butylphenol; Group I) and PTBP (p-tert-butylphenol; Group II) suggest the forestomach to be the main organ affected. OTBP also appears to have a mild (though statistically significant) protective effect against benzo[a]pyrene induced forestomach tumors. Long-term treatment with high dietary dose levels of PTBP caused hyperplastic changes in the forestomach epithelium of rats and hamsters, a likely consequence of the irritancy of the material. The relevance of this for human hazard is doubtful, particularly since there is no analogous structure in humans to the forestomach of rodents. There was no evidence of an effect on reproductive function at dosages up to 150 mg/kg. One reproductive screening study reported increased ‘breeding loss and also reduced pup weight gain and survival in early lactation at 750 mg/kg/day. It is reasonable to assume that these effects were secondary to “severe toxic symptoms” reported in the dams at this dosage. Other than an indication of a very mildly oestrogenic effect of PNP (p-nonylphenol; Group II) at a high dose levels (200-300 mg/kg/day) no effect on development was seen in a multigeneration study. By means of the classification method of Verhaar * all the alkylphenols would be classified as Type 2 compounds (polar narcotics). Narcosis, a non-specific mode of toxicity is caused by disruption (perturbation) of the cell membrane. The ability to induce narcosis is dependent on the hydrophobicity of the substance with biochemical activation or reaction involved. Such narcotic effects are also referred to as minimum or base-line toxicity. Polar narcotics such as the category phenols are usually characterised by having hydrogen bond donor activity and are thought to act by a similar mechanism to the inert, narcotic compounds but exhibit above base-line toxicity. In fact, a large number of alkylphenols have been evaluated as intravenous anesthetic agents. While the structure-activity relationships were found to be complex, the anesthetic potency and kinetics appeared to be a function of both the lipophilic character and the degree of steric hindrance exerted by ortho substituents. Less steric hindrance resulted in lower potency, while greater crowding led to complete loss of anesthetic activity and greater lipophilicity resulted in slower kinetics. These data support the notion that the alkylphenols behave as polar narcotics. In addition, the anaesthetic activity/potency differences seen with varying structure and placement of substituents strongly supports the division of alkylphenols category into the ortho, para, and di/tri-substituted groups (i.e. Group I, II and III, respectively). **Genotoxicity: I**t reasonable to consider the mutagenic potential of all the alkylphenols together because only functional group is the phenolic, which is not a structural alert for mutagenicity. The data support this, since the results of genotoxicity testing are uniformly negative for all category substances examined * Verhaar, H.J.M. van Leeuwen, C.J. and Hermens, J.L.M., Classifying Environmental Pollutants. 1: Structure-Activity Relationships for Prediction of Aquatic Toxicity, Chemosphere (25), pp 471 – 491 (1992). for nonylphenol: Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study. Nonylphenol was not mutagenic to Salmonella typhimurium, TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA, with or without an exogeneous metabolic activation system. Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.
Hychem Hyflex NS Hardener & NONYLPHENOL & TRIMETHYLHEXAMETHYLENE DIAMINE & COCOAMINE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented

Continued...

	exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
TRIMETHYLHEXAMETHYLENE DIAMINE & COCOAMINE	While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects. ‣ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis. ‣ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient. Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion. **Inhalation:** Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies. While most polyurethane amine catalysts are not sensitisers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease. Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema. **Skin Contact:** Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis. Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient. **Eye Contact:** Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. Direct contact with the liquid amine may cause severe irritation and tissue injury, and the “burning” may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling. The corneal swelling may manifest itself in visual disturbances such as blurred or “foggy” vision with a blue tint (“blue haze”) and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation. **Ingestion:** The oral toxicity of amine catalysts varies from moderately to very toxic. Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus,and gastrointestinal tract. Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs. Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death. **Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000** **Alliance for Polyurethanes Industry** The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence). The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

Continued...

	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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

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

SECTION 12 Ecological information

Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
Hychem Hyflex NS Hardener	Not Available	Not Available	Not Available	Not Available	Not Available

	Endpoint	Test Duration (hr)	Species	Value	Source
nonylphenol	BCF	1344h	Fish	90-220	7
	NOEC(ECx)	672h	Crustacea	0.0039mg/l	1
	EC50	72h	Algae or other aquatic plants	0.056mg/l	4
	EC50	96h	Algae or other aquatic plants	0.027mg/l	1
	LC50	96h	Fish	0.098-0.187mg/l	4
	EC50	48h	Crustacea	0.14mg/l	1

	Endpoint	Test Duration (hr)	Species	Value	Source
trimethylhexamethylene diamine	EC50(ECx)	72h	Algae or other aquatic plants	29.5mg/l	Not Available
	EC50	72h	Algae or other aquatic plants	29.5mg/l	Not Available

	Endpoint	Test Duration (hr)	Species	Value	Source
cocoamine	NOEC(ECx)	96h	Algae or other aquatic plants	<0.001mg/l	1
	LC50	96h	Fish	0.1mg/l	1
	EC50	96h	Algae or other aquatic plants	0.001mg/l	1
	EC50	48h	Crustacea	0.045mg/l	1

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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
for alkylphenols:
The alkylphenolics may be divided into three groups.
Group I: ortho-substituted mono-alkylphenols:
Group II para-substituted mono-alkylphenols
Group III: di- and tri-substituted mixed alkyl phenols
All the phenols have a single, common functional group the phenolic hydroxyl. Because alkyl and benzyl groups have a small positive inductive effect all the group phenols are expected to have slightly higher acid dissociation constants (pKa) than phenol (pKa 10.0 at 25.C). Data in a review of the physical chemistry properties of substituted phenols confirms a limited pKa range of 9.9 to 10.9. Thus, none of the alkylphenols will be ionised significantly at environmental or physiological pH s.
Although the overall category phenols do not form a homologous series, values for several of the more important physical chemistry parameters do correlate with molecular weight. In particular water solubility and vapour pressure decrease with increasing molecular weight, and the octanol/water partition coefficient (log Kow) increases. This trend is unmistakable in Group II and Group III substances while the ortho-substituted materials of the same molecular weight are similar

Continued...

Environmental fate:

Direct photolysis is not expected to be a significant route of loss for any of the alkylphenols because of limited absorbance above 290 nm. However, indirect photolysis (atmospheric oxidation) has been estimated for all substances. None of the alkylphenols are expected to be susceptible to abiotic hydrolysis under environmental conditions.

Level I fugacity modelling reveals that the vast majority of the alkylphenols will be located primarily in the soil compartment with a few exceptions. This is especially evident in the Group II and Group III materials. The model also suggests that a few lower molecular weight phenols, with correspondingly higher water solubility and vapour pressures, will also be present in significant quantities (>10%) in the air and water compartments

Ecotoxicity:

The aquatic toxicity of alkylphenols has been extensively investigated. As might be expected for polar narcotic type substances, the aquatic toxicities for the alkylphenols appears to be related to their degree of lipophilicity and increases basically in line with log Kow. Given the apparent lack of structural specificity associated with these endpoints, it is reasonable to assume (where experimentally determined data are not available) that the toxicity of a particular alkylphenol will be comparable to that of another with like lipophilicity.

For Fatty Nitrogen-derived ether amines and Fatty Nitrogen-Derived amines (FND ether amines and FND amines):

Environmental fate:

Consistent with the size and nature of these molecules, measured and modeled vapor pressures are very low, and the FND ether amines are considered to be essentially nonvolatile. Measurement and prediction of physical/chemical properties for surfactants are complicated by their behavior in test systems and the environment, and the log Kow is not an appropriate hydrophobicity parameter for reliably predicting environmental behavior .Prediction of physical/chemical properties, including strong adsorption and absorption properties (log Koc) and surface tension activity is unreliable for the same reasons.

Water solubility estimates varied from slightly soluble to very insoluble.

Fugacity models predict virtually no occurrence of the FND ether amines in air, which is consistent with the very low vapor pressures. Nonetheless, modeling indicates that they would be expected to degrade relatively rapidly upon exposure to light (t1/2 values ranging from approximately 1.0 to 2.8 hours).

Distribution to air and soil were < 1% for all of the chemicals that could be modeled while distribution to the water compartment varied from 5 to 90% with the remainder in the sediment.

Overall, FND ether amines have been shown to be either readily biodegradable or to attain degradation close to meeting the “readily biodegradable” criteria.

Ecotoxicity:

A number of studies evaluating the toxicity of the structurally related chemicals, to fish, have been reported. Three assays for one such chemical indicated substantial differences in the measured toxicity ranging from 23 to > 1000 mg/L. Again, this range shows the complexity of testing these types of chemicals.

It is likely that the range of values represents bioavailability or physical availability (important because many surfactant-like chemicals are known to kill aquatic organisms via a physical rather than chemical mechanism) of the test chemical to the fish. Except where bioavailability questions arose, reported LC50 values rang from 0.11 to 9.3 mg/L. These findings are consistent with the FND surfactants (cationics, amides, nitriles, ether nitriles) in general and support the conclusion that the FND chemicals are toxic to fish when bioavailable, presumably due to their surfactant properties.

Similar to the fish testing, a series of studies evaluating toxicity to aquatic invertebrates is available for structurally related chemicals with EC50 values ranging between 0.011 and 21 mg/L, except where bioavailability questions arose. Acute toxicity to daphnia was confounded by solubility problems and yielded higher than expected EC50 values In addition, a study examining a mixture of an FND active ingredient with inert materials (e.g. as used in soap) and using two water sources, indicated that river water reduced the toxicity compared to well water (EC50 = 60 vs 22 mg/L, respectively) and that the inert ingredients tended to reduce toxicity (EC50 = 6.5 mg/L for the 83.5% material vs 22 mg/L for the 63% material).

Toxicity to aquatic plants for structurally related chemicals indicates that these amine surfactants are highly toxic to algae (EbC50 and ErC50 values ranging from 0.00075 to 0.17 mg/L).

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

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

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

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

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

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

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

Continued...

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 (220 ug/ l) 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.

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.

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
nonylphenol	HIGH	HIGH
trimethylhexamethylene diamine	HIGH	HIGH
cocoamine	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
nonylphenol	LOW (BCF = 271)
trimethylhexamethylene diamine	LOW (LogKOW = 1.6347)
cocoamine	HIGH (LogKOW = 5.7458)

Mobility in soil

Ingredient	Mobility
nonylphenol	LOW (KOC = 56010)
trimethylhexamethylene diamine	LOW (KOC = 1101)
cocoamine	LOW (KOC = 27640)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible. Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

Continued...

A Hierarchy of Controls seems to be common - the user should investigate:

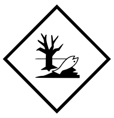
- Reduction
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

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

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	2X

Land transport (ADG)

<table>
<tr><td>UN number or ID number</td><td>2735</td></tr>
<tr><td>UN proper shipping name</td><td>AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S.</td></tr>
<tr><td>Transport hazard class(es)</td><td><table><tr><td>Class</td><td>8</td></tr><tr><td>Subsidiary risk</td><td>Not Applicable</td></tr></table></td></tr>
<tr><td>Packing group</td><td>III</td></tr>
<tr><td>Environmental hazard</td><td>Environmentally hazardous</td></tr>
<tr><td>Special precautions for user</td><td><table><tr><td>Special provisions</td><td>223 274</td></tr><tr><td>Limited quantity</td><td>5 L</td></tr></table></td></tr>
</table>

Air transport (ICAO-IATA / DGR)

<table>
<tr><td>UN number</td><td>2735</td></tr>
<tr><td>UN proper shipping name</td><td>Polyamines, liquid, corrosive, n.o.s. *; Amines, liquid, corrosive, n.o.s. *</td></tr>
<tr><td>Transport hazard class(es)</td><td><table><tr><td>ICAO/IATA Class</td><td>8</td></tr><tr><td>ICAO / IATA Subrisk</td><td>Not Applicable</td></tr><tr><td>ERG Code</td><td>8L</td></tr></table></td></tr>
<tr><td>Packing group</td><td>III</td></tr>
<tr><td>Environmental hazard</td><td>Environmentally hazardous</td></tr>
<tr><td>Special precautions for user</td><td><table><tr><td>Special provisions</td><td>A3 A803</td></tr><tr><td>Cargo Only Packing Instructions</td><td>856</td></tr><tr><td>Cargo Only Maximum Qty / Pack</td><td>60 L</td></tr></table></td></tr>
</table>

Continued...

	Passenger and Cargo Packing Instructions	852
	Passenger and Cargo Maximum Qty / Pack	5 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y841
	Passenger and Cargo Limited Maximum Qty / Pack	1 L

Sea transport (IMDG-Code / GGVSee)

UN number	2735
UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S.
Transport hazard class(es)	IMDG Class: 8 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-A, S-B Special provisions: 223 274 Limited Quantities: 5 L

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

Not Applicable

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

Product name	Group
nonylphenol	Not Available
trimethylhexamethylene diamine	Not Available
cocoamine	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
nonylphenol	Not Available
trimethylhexamethylene diamine	Not Available
cocoamine	Not Available

SECTION 15 Regulatory information

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

nonylphenol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

trimethylhexamethylene diamine is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)

cocoamine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia	Yes

Continued...

National Inventory	Status
Non-Industrial Use	
Canada - DSL	Yes
Canada - NDSL	No (trimethylhexamethylene diamine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (cocoamine)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	10/12/2021
Initial Date	14/12/2009

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
5.1	10/12/2021	Classification change due to full database hazard calculation/update.

Other information

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

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC－TWA: Permissible Concentration-Time Weighted Average
PC－STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances

Continued...

ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances