Ramset Chemset Injection Reo 502

Ramset (ITW Australia Pty Ltd)

Chemwatch: **41-8662**
Version No: **2.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: **3**

Issue Date: **20/05/2014**
Print Date: **20/05/2014**
Initial Date: **Not Available**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Ramset Chemset Injection Reo 502
Chemical Name	Not Applicable
Synonyms	Product Code: REO502J
Proper shipping name	CORROSIVE LIQUID, BASIC, ORGANIC, N.O.S. (contains benzene-1,3-dimethanamine,N-aminoethylpiperazine and nonylphenol)
Chemical formula	Not Applicable
Other means of identification	Not Available
CAS number	Not Applicable

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

Relevant identified uses	Requires that the two parts be mixed by hand or mixer before use, in accordance with manufacturers directions. Mix only as much as is required. **Do not** return the mixed material to the original containers

Details of the supplier of the safety data sheet

Registered company name	Ramset (ITW Australia Pty Ltd)		
Address	1 Ramset Drive Chirnside Park 3116 VIC Australia		
Telephone	1300 780 063		
Fax	Not Available		
Website	www.ramset.com.au		
Email	Not Available		

Emergency telephone number

Association / Organisation	Not Available		
Emergency telephone numbers	1800 039 008 (24 hrs)		
Other emergency telephone numbers	1800 039 008 (24 hrs)		

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	+612 9186 1132	Not Available

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the Model WHS Regulations and the ADG Code.

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	**1**	
Toxicity	**2**	
Body Contact	**3**	
Reactivity	**1**	
Chronic	**2**	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	S5
GHS Classification [1]	Metal Corrosion Category 1, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 1B, Serious Eye Damage Category 1, Respiratory Sensitizer Category 1, Skin Sensitizer Category 1, Germ Cell Mutagen Category 2, Reproductive Toxicity Category 2, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Label elements

GHS label elements	

SIGNAL WORD	DANGER

Hazard statement(s)

H290	May be corrosive to metals
H302	Harmful if swallowed
H312	Harmful in contact with skin
H332	Harmful if inhaled
H314	Causes severe skin burns and eye damage
H318	Causes serious eye damage
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled
H317	May cause an allergic skin reaction
H341	Suspected of causing genetic defects
H361	Suspected of damaging fertility or the unborn child
H401	Toxic to aquatic life
H411	Toxic to aquatic life with long lasting effects

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s): Prevention

P101	If medical advice is needed, have product container or label at hand.
P102	Keep out of reach of children.
P103	Read label before use.
P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P234	Keep only in original container.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s): Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
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
P321	Specific treatment (see advice on this label).
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider
P302+P352	IF ON SKIN: Wash with plenty of water and soap
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P363	Wash contaminated clothing 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.

Precautionary statement(s): Storage

P405	Store locked up.

Precautionary statement(s): Disposal

P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	30-60	Part A

25068-38-6	NotSpec.	bisphenol A/ epichlorohydrin resin
	NotSpec.	ingredients determined not to be hazardous
	NotSpec.	Part B
1477-55-0	15-20	benzene-1,3-dimethanamine
80-05-7	10-20	bisphenol A
140-31-8	12-15	N-aminoethylpiperazine
25154-52-3	<10	nonylphenol
103-83-3	<3	N,N-dimethylbenzylamine
108-95-2	<3	phenol
	NotSpec.	ingredients determined not to be hazardous

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

Treat symptomatically.
For acute or short-term repeated exposures to highly alkaline materials:
Respiratory stress is uncommon but present occasionally because of soft tissue edema.
Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
Oxygen is given as indicated.
The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.
Alkalis continue to cause damage after exposure.
INGESTION:
Milk and water are the preferred diluents
No more than 2 glasses of water should be given to an adult.
Neutralising agents should never be given since exothermic heat reaction may compound injury.
* Catharsis and emesis are absolutely contra-indicated.
* Activated charcoal does not absorb alkali.
* Gastric lavage should not be used.
Supportive care involves the following:
Withhold oral feedings initially.
If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).
SKIN AND EYE:
Injury should be irrigated for 20-30 minutes.
Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

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

Water spray or fog.
Alcohol stable foam.
Dry chemical powder.
Carbon dioxide.

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:
Combustible.
Slight fire hazard when exposed to heat or flame.
Heating may cause expansion or decomposition leading to violent rupture of containers.
On combustion, may emit toxic fumes of carbon monoxide (CO).
May emit acrid smoke.
Mists containing combustible materials may be explosive.
Combustion products include:
,
carbon dioxide (CO_2)
,
nitrogen oxides (NO_x)
,
other pyrolysis products typical of burning organic material
May emit corrosive fumes.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills:
Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.
Check regularly for spills and leaks.
Small spills should be covered with inorganic absorbents and disposed of properly. Organic absorbents have been known to ignite when contaminated with amines in closed containers. Certain cellulosic materials used for spill cleanup such as wood chips or sawdust have shown reactivity with ethyleneamines and should be avoided. Ethyleneamine leaks will frequently be identified by the odor (ammoniacal) or by the formation of a white, solid, waxy substance (amine carbamates). Inorganic absorbents or water may be used to clean up the amine waste.
Clean up all spills immediately.
Avoid breathing vapours and contact with skin and eyes.
Control personal contact with the substance, by using protective equipment.
Contain and absorb spill with sand, earth, inert material or vermiculite.
Wipe up.
Place in a suitable, labelled container for waste disposal.

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

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS

LAND SPILL - SMALL

cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
wood fiber - pillow	1	throw	pitchfork	R, P, DGC, RT
foamed glass - pillow	2	shovel	shovel	R, W, P, DGC
sorbent clay - particulate	2	shovel	shovel	R, I, P
wood fibre - particulate	3	shovel	shovel	R, W, P, DGC

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	R, SS, DGC
wood fiber - particulate	4	blower	skiploader	R, W, P, DGC
expanded moneral - particulate	4	blower	skiploader	R, I, W, P, DGC

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

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.
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 MSDS.

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 MSDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	for bulk storages: If slight coloration of the ethyleneamine is acceptable, storage tanks may be made of carbon steel or black iron, provided they are free of rust and mill scale. However, if the amine is stored in such tanks, color may develop due to iron contamination. If iron contamination cannot be tolerated, tanks constructed of types 304 or 316 stainless steel should be used. (Note: Because they are quickly corroded by amines, do not use copper, copper alloys, brass, or bronze in tanks or lines.) This product should be stored under a dry inert gas blanket, such as nitrogen, to minimize contamination resulting from contact with air and water Store in original containers. Keep containers securely sealed. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this MSDS. **DO NOT** store near acids, or oxidising agents No smoking, naked lights, heat or ignition sources.

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	N-aminoethylpiperazine: is a strong base in aqueous solutions is incompatible wit strong oxidisers, organic anhydrides, acrylates, alcohols, aldehydes, alkylene oxides, substituted allyls, cellulose nitrate, cresols, caprolactam solution, epichlorohydrin, ethylene dichloride, isocyanates, ketones, glycols, nitrates, organic halides, phenols, vinyl acetate decomposes exothermically with maleic anhydride may increase the explosive sensitivity of nitromethane attacks aluminium, copper, magnesium, nickel, zinc, or their alloys, and galvanised steel 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 oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates. Avoid contact with copper, aluminium and their alloys.

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	benzene-1,3-dimethanamine	m-Xylene-a,a'-diamine	Not Available	Not Available	0.1 (mg/m3)	Not Available

Australia Exposure Standards	phenol	Phenol	4 (mg/m3) / 1 (ppm)	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	TEEL-0	TEEL-1	TEEL-2	TEEL-3
bisphenol A/ epichlorohydrin resin	125 / 4 / 50(ppm)	350 / 150 / 12.5(ppm)	500 / 100(ppm)	500(ppm)
bisphenol A	5(ppm)	15(ppm)	100(ppm)	500(ppm)
N-aminoethylpiperazine	2.5(ppm)	7.5(ppm)	50(ppm)	500(ppm)
nonylphenol	5 / 6(ppm)	15 / 20(ppm)	125 / 100(ppm)	500(ppm)
N,N-dimethylbenzylamine	1(ppm)	3(ppm)	20(ppm)	200(ppm)
phenol	5(ppm)	15(ppm)	23(ppm)	200(ppm)

Ingredient	Original IDLH	Revised IDLH
phenol	250(ppm)	250 [Unch](ppm)

MATERIAL DATA

Odour Threshold Value for phenol: 0.060 ppm (detection)
NOTE: Detector tubes for phenol, measuring in excess of 1 ppm, are commercially available.
Systemic absorption by all routes may induce convulsions with damage to the lungs and central nervous system.
Exposure at or below the recommended TLV-TWA is thought to protect the worker from respiratory, cardiovascular, hepatic, renal and neurological toxicity. Workers or volunteers exposed at or below 5.2 ppm phenol have experienced no ill-effects. Because phenol as a vapour, liquid or solid can penetrate the skin causing systemic effects, a skin notation is considered necessary. Although ACGIH has not recommended a STEL it is felt that ACGIH excursion limits (15 ppm limited to a total duration of 30 minutes with brief excursions limited to no more than 25 ppm) and NIOSH Ceiling values are sufficiently similar so as to provide the same margin of safety.
Odour Safety Factor(OSF)
OSF=25 (PHENOL)
For benzene-1,3-dimethanamine (m-xylene-alpha,alpha'-diamine)
Saturates in air at 219.5 mg/m3 (39.5 ppm) at 25 deg C.
The substance is a gastrointestinal irritant and skin sensitiser in humans. Its actions are similar to p-phenylenediamine and the recommendation for a TLV-C is derived by analogy. Exposure at or below this value is thought to protect workers against the risk of skin irritation, percutaneous absorption and systemic injury. It should be noted however that individuals might be hypersusceptible or otherwise unusually responsive to the certain chemicals and this value may not be adequate to provide effective protection against adverse health effects.
The skin notation is currently undergoing review.
The TLV value is listed only in mg/m3 although it is anticipated that at this concentration the compound should exist largely as vapour.

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.

Personal protection

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]
Skin protection	See Hand protection below
Hand 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. 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. 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.
Body protection	See Other protection below
Other protection	Overalls. PVC Apron. PVC protective suit may be required if exposure severe. Eyewash unit. Ensure there is ready access to a safety shower.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
***"*Forsberg Clothing Performance Index".**
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Ramset Chemset Injection Reo 502

Material	CPI
BUTYL	A

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

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

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Supplied in a plastic coaxial tube - containing both parts. Part A is a white viscous liquid; not miscible with water. Part B is a black viscous liquid; not miscible with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.4

Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	>11 (Part B)	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	>149 (Part A)	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	>100	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	VOC = 0 g/l (mixed and hardened)
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water (g/L)	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	Presence of incompatible materials. Product is considered stable. Hazardous polymerisation will not occur.
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. In clinical observation of workers, at a producer of benzene-1,3-dimethanamine (m-xylene-alpha,alpha'- diamine), the compound produced gastrointestinal irritation which was attributed to its caustic nature. Exposure of rats for 1 hour to an aerosol at concentrations ranging from 1.74 - 6.04 mg/l (1740 - 6040 mg/m3) resulted in frank ocular irritation, lachrymation and dyspnea. Although no fatalities occurred during the period of exposure several animals died within 48 hours. Necroscopy revealed macroscopic abnormalities involving the lungs. Liver and kidney changes were also noted. Guinea pigs exposed for three 2-hour periods for 3 days at approximately 50 ppm vapour, showed impaired appetite, reduced reaction to stimuli, with reduced alertness and dyspnea (progressing in severity with prolongation of exposure) occurred in all exposed animals. Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales. Inhalation hazard is increased at higher temperatures. 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. 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	Skin contact with the material may be harmful; systemic effects may result following absorption. The material can produce chemical burns following direct contact with the skin. Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur. Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions. Individuals exhibiting "amine dermatitis" may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis. NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided. Undiluted benzene-1,3-dimethanamine (m-xylene-alpha,alpha'- diamine) is corrosive to guinea pig skin. A 10 % aqueous solution of the material produces severe erythema, irritation. Repeated applications of a 5% aqueous solution produce local oedema redness. One test showed evidence of mild sensitisation following repeated guinea pig skin application. The result was not duplicated in another test. It has been reported that the material is a potent skin sensitiser of workers in plastics manufacturing. 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
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 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. Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. 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. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. 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. Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Prolonged exposure to some derivatives of phenol may produce dermatitis, anorexia, weight loss, weakness, muscle aches and pain, liver damage, dark urine, ochronosis, skin eruptions, diarrhoea, nervous disorders with headache, salivation, fainting, increased skin and scleral pigmentation, vertigo and mental disorders. Liver and kidney damage may also ensue. Chronic phenol toxicity was first noted in medical personnel in the late 1800s when 5 and 10% phenol was used as a skin disinfectant. The term carbolic (phenol) marasmus was given to this syndrome. Addition of structurally related phenolic compounds to the diet of Syrian golden hamsters induced forestomach hyperplasia and tumours. These compounds included 2(3)-tert-butyl-4-methoxphenol (BHA) (CAS RN: 25013-16-5), 2-tert-butyl-4-methylphenol (TBMP) (29759-28-2) and p-tert-butylphenol (PTBP) (98-54-4); less active were catechol (154-23-4), p-methylphenol (331-39-5), methylhydroquinone (MHQ) (95-71-6) and pyrogallol (87-66-1), whilst no activity was seen with resorcinol (108-46-3), hydroquinone (123-31-9), propylparaben (94-13-3) and tert-butylhydroquinone (TBHQ) (1948-33-0). In autoradiographic studies, intake of BHA, TBMP, catechol, PMOP, PTBP and MHQ resulted in a significant increase in the labelling index of the forestomach epithelium, whilst PMOP induced epithelial damage and pyloric regenerative hyperplasia. Catechol, CA and PYMP induced similar but less marked alterations. Both catechol and PMOP increased the labelling index in the glandular stomach. The urinary bladder was free from histo-pathological lesions, but propylparabene, catechol, TBHQ and MHQ increased the labelling index. The authors of this study concluded that long term administration of PTBP and TBMP may be carcinogenic for hamster forestomach and that both 1-hydroxy and tert-butyl substituents may play a role in the induction of forestomach tumours. Hiros, M., et al: Carcinogenesis, Vol 7, pp 1285-1289; 1986 On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Solid phenol is highly toxic via ingestion, inhalation and skin contact. Chronic phenol poisoning is very rarely reported, but symptoms include vomiting, difficulty in swallowing, diarrhoea, lack of appetite, headache, fainting, dizziness, dark urine, mental disturbances, and possibly skin rash. Death due to liver and kidney damage may occur. Repeated exposure of animals to phenol vapour at concentrations ranging from 26 to 52 ppm has produced respiratory, cardiovascular, hepatic, renal and neurologic toxicity. Administration of phenol in the drinking water of mice (2500 ppm for 103 weeks) produced an increased incidence of leukemia and lymphomas. Phenol has been studied in initiation/promotion protocols with a number of polycyclic hydrocarbons and has been shown to have promoting activity in the two-stage skin model 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.

Ramset Chemset Injection Reo 502	TOXICITY	IRRITATION
	Not Available	Not Available

bisphenol A/ epichlorohydrin resin	TOXICITY	IRRITATION
	Intraperitoneal (mouse) LD50: 4000 mg/kg	Eye (rabbit): 100 mg - mild
	Intraperitoneal (rat) LD50: 2400 mg/kg	Nil reported
	Oral (mouse) LD50: 15600 mg/kg	
	Oral (rat) LD50: 11400 mg/kg	
	Oral (rat) LD50: 13600 mg/kg	
	Not Available	Not Available

benzene-1,3-dimethanamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2000 mg/kg	Eye (rabbit): 0.05 mg/24h SEVERE
	Inhalation (rat) LC50: 700 ppm/1h	Skin (rabbit): 0.75 mg/24h SEVERE
	Oral (rat) LD50: 930 mg/kg	
	Not Available	Not Available

bisphenol A	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3000 mg/kg	Eye (rabbit): 0.25 mg/24h-SEVERE
	Inhalation (rat) LC50: 200 ppm	Skin (rabbit): 250 mg open - mild
	Intraperitoneal (Mouse) LD50: 150 mg/kg	Skin (rabbit): 500 mg/24h - mild
	Oral (Mouse) LD50: 2400 mg/kg	
	Oral (Rabbit) LD50: 2230 mg/kg	
	Oral (Rat) LD50: 1200 mg/kg	
	Oral (rat) LD50: 3250 mg/kg	
	Oral (Rat) TDLo: 1000 mg/kg	
	Subcutaneous (Mouse) LD: 2500 mg/kg	
	Subcutaneous (Rat) TDLo: 5.9 mg/kg	
	Not Available	Not Available

N-aminoethylpiperazine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 880 mg/kg	Eye (rabbit): 20 mg/24h - mod
	Intraperitoneal (Mouse) LD50: 250 mg/kg	Skin (rabbit): 0.1 mg/24h - mild
	Oral (rat) LD50: 2410 mg/kg	Skin (rabbit): 5 mg/24h - SEVERE
	Not Available	Not Available

nonylphenol	TOXICITY	IRRITATION
	Oral (rat) LD50: 1620 mg/kg	Skin(rabbit):10mg/24h(open)-SEVERE
	Not Available	Not Available

N,N-dimethylbenzylamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1660 mg/kg	Eye (rabbit): 5 mg - SEVERE
	Inhalation (Mouse) LC: 1200 mg/m3/2h	Skin (rabbit): 500 mg/4h-SEVERE
	Inhalation (mouse) LC50: 1800 mg/m3/2h	
	Inhalation (Rat) LC: 1200 mg/m3/2h	
	Oral (rat) LD50: 265 mg/kg	
	Not Available	Not Available

phenol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 850 mg/kg	Eye(rabbit): 100 mg rinse - mild
	Inhalation (rat) LC50: 316 mg/m3	Eye(rabbit): 5 mg - SEVERE
	Oral (human) LDLo: 140 mg/kg	Skin(rabbit): 500 mg open -SEVERE
	Oral (rat) LD50: 317 mg/kg	Skin(rabbit): 500 mg/24hr - SEVERE

	Not Available	Not Available

** Value obtained from manufacturer's msds*
unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances

Ramset Chemset Injection Reo 502	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. Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. No significant acute toxicological data identified in literature search. The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities. Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor. Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds. Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines. In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper 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. Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
BISPHENOL A/ EPICHLOROHYDRIN RESIN	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. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. for RTECS No: SL 6475000: (liquid grade) Equivocal tumourigen by RTECS criteria Somnolence, dyspnea, peritonitis

BENZENE-1,3-DIMETHANAMINE	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. Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. For benzene-1,3-dimethanamine (m-xylene-alpha,alpha'- diamine) The toxicity via oral administration and inhalation was tissue damage in the digestive and respiratory organs, respectively, which are the first contact sites. The chemical is corrosive to rat and mouse skin and a sensitiser in the guinea pig maximisation test. In the 28-day repeated dose toxicity study [OECD TG 407], the chemical was given to rats by gavage at doses of 0, 10, 40, 150 and 600 mg/kg b.w/day. One male and four females died, and salivation, low locomotor activity and piloerection were noted in the 600 mg/kg group. Furthermore, ulceration, acanthosis with hyperkeratosis and submucosal inflammation were observed in the forestomach. No adverse effects were observed in the 150 mg/kg and the lower dose groups. A reproductive /developmental toxicity screening test [OECD TG 421] of rats by gavage at 50, 150 and 450 mg/kg b.w/day for at least 41 days resulted in death in one male in the 150 mg/kg group, and three males and one female in the 450 mg/kg group. In almost all 450 mg/kg animals, the same histopathological changes as the above 28-day study were observed in the forestomach. No adverse effects were found at 50 mg/kg b.w/day. Based on this information, the NOAEL for repeated dose toxicity is considered to be 50 mg/kg b.w/day. In the above reproductive/developmental toxicity screening test [OECD TG 421] the substance was administered from 14 days before mating to 20 days after mating in males and to day 3 of lactation in females. No adverse effects were observed in terms of copulation, fertility, delivery and nursing of parents, and the viability, body weight and morphology of offspring. The NOAEL for reproductive/developmental toxicity (F1 offspring) was 450 mg/kg b.w/day. The chemical was not mutagenic in bacteria [OECD TG 471 & 472]. It induced neither chromosomal aberrations in mammalian cells *in vitro* [OECD TG 473] nor micronuclei in mouse bone marrow *in vivo* [OECD TG 474]. In clinical observation of workers during the manufacturing process, the chemical appears to act as a gastrointestinal irritant. It has also been shown to cause contact sensitisation reactions in workers at concentrations equal to and below 0.1 mg/m3 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.
BISPHENOL A	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. Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. No significant acute toxicological data identified in literature search. The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities. Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by

proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.
For bisphenol A (BPA)
Following oral administration absorption of BPA is rapid and extensive while dermal absorption is limited. Extensive first pass metabolism occurs following absorption from the gastrointestinal tract with glucuronide conjugation being the major metabolic pathway. Bisphenol A is of low acute toxicity (rodent oral LD50 values from 3300-4100 mg/kg, a rabbit oral LD50 value 2230 mg/kg and a rat acute inhalation 6-hour LC50 value >170 mg/m3). Bisphenol A is not a skin irritant, however, it is severely irritating to the eyes. BPA was negative in gene mutation and clastogenicity assays in cultured mammalian cells, as well as in a micronucleus test for clastogenicity *in vivo*; therefore, BPA is considered not to present a genotoxic concern for human health. BPA results in minimal effects on the liver and kidney (LOAEL from chronic exposure in the diet was 50 mg/kg/day). For reproductive toxicity, data from a three-generation study in the rat, BPA was not a selective reproductive toxicant at doses ranging from 0.001 to 500 mg/kg/day. BPA is not a developmental toxicant in rats or mice.
Inconsistent findings are reported in the "low dose" literature for bisphenol A. The inherent challenge of conducting these types of studies may be exacerbated with bisphenol A because the endpoints of concern are endocrine-mediated and potentially impacted by factors that include phytoestrogen content of the animal feed, extent of bisphenol A exposure from caging or water bottles, and the alleged sensitivity of the animal model to oestrogens.
High-dose studies are less susceptible to these types of influences because the toxicologic response should be more robust and less variable . Several large, robust, well designed studies with multiple dose groups using several strains of rats and mice have been conducted and none of these detected any adverse reproductive effects at low to moderate dosage levels of BPA administered via the relevant route of human exposures. Further, none of these studies detected changes in prostate weight, age at puberty (rat), pathology or tumors in any tissue, or reproductive tract malformations.
Every chemical that produces low dose cellular and molecular alterations of endocrine function also produces a cascade of effects increasing in severity resulting in clearly adverse alterations at higher doses, albeit the effects can be different from those seen at low doses. With these endocrine disrupters, but not BPA, the low dose effects are often causally linked to the high-dose adverse effects of the chemical. This is true for androgens like testosterone and trenbolone, estrogens like DES, 17beta-oestradiol and ethinyl oestradiol, xenoestrogens like methoxychlor and genistein, and antiandrogens like vinclozolin, for example. Hence, the failure of BPA to produce reproducible adverse effects via a relevant route of exposure, coupled with the lack of robustness of the many of the low dose studies (sample size, dose range, statistical analyses and experimental design, GLP) and the inability to reproduce many of these effects of any adverse effect strains the credibility of some of these study results.
The lack of reproducibility of the low dose effects, the absence of toxicity in those low-dose-affected tissues at high-doses, and the uncertain adversity of the reported effects lead to the conclusion that there is "minimal" concern for reproductive effects.
In contrast, the literature on bisphenol A effects on neural and behavioral response is more consistent with respect to the number of "positive"' studies although it should be noted that the high-dose studies that proved to be the most useful for evaluating reproductive effects did not adequately assess neural and behavioral responses. In addition, even though different investigators assessed different neural and behavioral endpoints, an expert Panel concluded that the overall findings suggest that bisphenol A may be associated with neural changes in the brain and behavioral alterations related to sexual dimorphism in rodents. For this reason, the Panel expressed "some" concern for these effects even though it is not clear the reported effects constitute an adverse toxicological response. In summary:
For pregnant women and foetuses, the Expert Panel has different levels of concern for the different developmental endpoints that may be susceptible to bisphenol A disruption, as follows:
- For neural and behavioral effects, the Expert Panel has some concern;
- For prostate effects, the Expert Panel has minimal concern;
- For the potential effect of accelerated puberty, the Expert Panel has minimal concern; and
- For birth defects and malformations, the Expert Panel has negligible concern.

For infants and children, the Expert Panel has the following levels of concern for biological processes that might be altered by Bisphenol A, as follows:
- Some concern for neural and behavioral effects; and
- Minimal concern for the effect of accelerated puberty.

For adults, the Expert Panel has negligible concern for adverse reproductive effects following exposures in the general population to Bisphenol A. For highly exposed subgroups, such as occupationally exposed populations, the level of concern is elevated to minimal.
NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Bisphenol A National Toxicology Program US Department of Health and Human Services September 2008 NTP Publication No 08-5994
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.

N-AMINOETHYLPIPERAZINE

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.
Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds.
Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines.
In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper

The material may produce moderate eye irritation leading to 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.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented

	exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. for piperazine: Exposure to piperazine and its salts has clearly been demonstrated to cause asthma in occupational settings. No NOAEL can be estimated for respiratory sensitisation (asthma). Although the LD50 levels indicate a relatively low level of oral acute toxicity (LD50 1-5 g/kg bw), signs of neurotoxicity may appear in humans after exposure to lower doses. Based on exposure levels of up to 3.4 mg/kg/day piperazine base and a LOAEL of 110 mg/kg, there is no concern for acute toxicity In pigs, piperazine is readily absorbed from the gastrointestinal tract, and the major part of the resorbed compound is excreted as unchanged piperazine during the first 48 hours. The principal route of excretion of piperazine and its metabolites is via urine, with a minor fraction recovered from faeces (16%). In humans the kinetics of the uptake and excretion of piperazine and its metabolites with urine appear to be roughly similar to that in the pig, and the nature and extent of conversion to metabolites has not been determined. Piperazine has demonstrated a low acute toxicity (LD50 = 1-5 g/kg bw) by the oral, dermal, and subcutaneous route of administration to rodents, whereas adequate inhalation toxicity data have not been found. However, there are findings of EEG (electroencephalogram) changes in 37% of 89 children administrated 90-130 mg/kg piperazine (two doses during one day), corroborated by a proposed GABA (gamma-aminobutyric acid) receptor agonism exerted by piperazine. Since clinical symptoms of neurotoxicity may occur after exposure to higher doses, a LOAEL of 110 mg/kg piperazine base for acute neurotoxicity in humans after acute exposure is proposed. Piperazine, as concentrated aqueous solution, has strongly irritating properties with regard to skin, and should be regarded as corrosive with respect to the eye. Exposure to piperazine and it salts has been demonstrated to cause allergic dermatitis as well as respiratory sensitisation in humans. As shown by the LLNA, piperazine has a sensitising potential in animals. Although piperazine is clearly sensitising, no NOAEL can be set for this effect from the present database. A NOAEL of 25 mg/kg/day of piperazine for liver toxicity in the beagle dog has been chosen after repeated exposure. A LOAEL of 30 mg/kg/day of piperazine for neurotoxicity is proposed based on documentation of (rare cases) of neurotoxicity from human clinical practice. Neurotoxicity also appears in other species (e.g., rabbits, dogs, cats, tigers, and horses), but not in rodents. For reproductive effects of piperazine, there is a NOAEL of 125 mg/kg/day for effects on fertility, i.e., reduced pregnancy index, decreased number of implantation sites, and decreased litter sizes in rats. The teratogenic properties have been investigated in rats and rabbits in adequate studies. In rabbit, such effects may be elicited at a dose level that is also toxic to the dam. The LOAEL is 94 mg/kg/day, and the NOAEL 42 mg/kg/day piperazine base (maternal and embryotoxic). In the rat study, there were decreases in body weight of both dams and offspring at the top dose (2,100 mg/kg/day piperazine base), but there were no signs of any malformations. The genotoxic properties have been investigated both *in vitro* (in the Ames test, in a nonstandard study on Saccharomyces cervisiae and in Chinese hamster ovary cells) and *in vivo*, in a micronuclei assay on mice, all with negative results. There are no solid indications of a carcinogenic effect of piperazine, neither in animal studies, nor from the investigation on humans. In view of lack of genotoxic action, it appears unlikely that piperazine poses a carcinogenic risk. There seems to be an additional cancer risk due to the formation of N-mononitrosopiperazine (NPZ) from piperazine. It is possible to calculate a hypothetical additional cancer risk posed by NPZ after exposure to piperazine, but the calculation would depend on several assumptions. We conclude that there seems to be an additional cancer risk due to the formation of NPZ from piperazine, and although it is difficult to estimate, it is probably small.
NONYLPHENOL	Skin (rabbit) LD50: 2140 mg/kg Skin (rabbit): 500 mg(open)-mod Eye (rabbit): 0.5 mg (open)-SEVERE 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. Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. 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. 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 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: It 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).

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

N,N-DIMETHYLBENZYLAMINE

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.

N, N-Dimethylbenzylamine was studied for oral toxicity in a 28 day repeat dose toxicity test. Mortalities of both males and females receiving 400 mg/kg were observed from week 2. Clinical observation revealed miosis in both sexes receiving 100 mg/kg and miosis and salivation in those receiving 200 and 400 mg/kg. Body weight gain was suppressed in males receiving 400 mg/kg. Slight increases in total cholesterol were observed in males receiving 200 mg/kg. Pathological examination revealed no abnormalities attributable to the test substance treatment. The NOEL for repeat dose toxicity is considered to be 50 mg/kg/day for both sexes. N, N-Dimethylbenzylamine was not mutagenic to Salmonella typhimurium TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA and with or without an exogeneous metabolic activation system. N, N-Dimethylbenzylamine induced structural chromosomal aberrations with an exogeneous metabolic activation system.

PHENOL

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.

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

nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
The substance is classified by IARC as Group 3:
NOT classifiable as to its carcinogenicity to humans.
Evidence of carcinogenicity may be inadequate or limited in animal testing.

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

CMR STATUS

SKIN	benzene-1,3-dimethanamine	Australia Exposure Standards - Skin	Sk
	phenol	Australia Exposure Standards - Skin	Sk

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
Ramset Chemset Injection Reo 502	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

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
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 bisphenol A and related bisphenols:

In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms. Among freshwater organisms, fish appear to be the most sensitive species. Evidence of endocrine-related effects in fish, aquatic invertebrates, amphibians and reptiles has been reported at environmentally relevant exposure levels lower than those required for acute toxicity. There is a widespread variation in reported values for endocrine-related effects, but many fall in the range of 1 ug/L to 1 mg/L
Bisphenol A, its derivatives and analogues, can be released from polymers, resins and certain substances by metabolic products
As an environmental contaminant, bisphenol A interferes with nitrogen fixation at the roots of leguminous plants associated with the bacterial symbiont Sinorhizobium meliloti. Despite a half-life in the soil of only 1-10 days, its ubiquity makes it an important pollutant. According to Environment Canada, "initial assessment shows that at low levels, bisphenol A can harm fish and organisms over time. Studies also indicate that it can currently be found in municipal wastewater." However, a study conducted in the United States found that 91-98% of bisphenol A may be removed from water during treatment at municipal water treatment plants.
A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations.
A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish, while females made up only 55 per cent in uncontaminated areas.
Although abundant data are available on the toxicity of bisphenol-A (2,2-bis (4-hydroxydiphenyl)propane;(BPA) A variety of BPs were examined for their acute toxicity against Daphnia magna, mutagenicity, and oestrogenic activity using the Daphtoxkit (Creasel Ltd.), the umu test system, and the yeast two-hybrid system, respectively, in comparison with BPA. BPA was moderately toxic to D. magna (48-h EC50 was 10 mg/l) according to the current U.S. EPA acute toxicity evaluation standard, and it was weakly oestrogenic with 5 orders of magnitude lower activity than that of the natural estrogen 17 beta-oestradiol in the yeast screen, while no mutagenicity was observed. All seven BPs tested here showed moderate to slight acute toxicity, no mutagenicity, and weak oestrogenic activity as well as BPA. Some of the BPs showed considerably higher oestrogenic activity than BPA, and others exhibited much lower activity. Bisphenol S (bis(4-hydroxydiphenyl)sulfone) and bis(4-hydroxyphenyl)sulfide) showed oestrogenic activity.
Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F ([bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism.

Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem,
Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe3+ ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.
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.
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.
For benzene-1,3-dimethanamine (m-xylene-alpha,alpha'- diamine)
Environmental fate:
The chemical has a log Pow value of 0.18 at 2 a vapour pressure 5 C, of 0.04 hPa at 25 C, and a water solubility of > 100 000 mg/L. Fugacity model Mackay level III calculations suggest that the majority of the chemical would distribute to soil if released to soil and/or air compartment(s), and water if released to aquatic compartment.
The chemical is not readily biodegradable (49% after 28 d) or inherently biodegradable (BOD = 22%, TOC = 6% and analysis in HPLC = 21%) and it does not hydrolyse (half-life >1 y at 25 C). However, the chemical does not bioaccumulate (BCF < 2.7 at 0.2 mg/L). The chemical will react with carbon dioxide to form the carbamate acid, and will undergo indirect photo-oxidation with hydroxy radicals (T1/2= 5.39 h), and will therefore not persist in the atmosphere.
Ecotoxicity:
Fish LC50 (96 h): Medaka 87.6 mg/l; golden orfe 75 mg/l; rainbow trout >100 mg/l
Daphnia magna EC50 (48 h): 15.2 - 16 mg/l
Daphnia magna EC50 (21 d): 6.77 mg/l (reproduction inhibition); NOEC 4.7 mg/l (reproduction inhibition)
Daphnia magna LC50 (21 d): 8.4 mg/l (parental toxicity)
Algae EbC50: Scenedesmus subspicatus 12 g/l; NOEC 6.25 mg/l; EbC50 Selenastrum capricornutum 20.3 mg/l; NOEC (0-72 h) 10.5 mg/l
For bisphenol A
log Kow 3.32
Kow : 314-1524
Half-life (hr) air : 4
Half-life (hr) H2O surface water : 96
BCF : 42-96

Biodegradation dominant (half-life <= 4 days; not expected to bioaccumulate significantly in aquatic organisms
Processes Abiotic: violent,fast decomposition.in H2O
Environmental fate:
If released to soil, bisphenol A is expected to have moderate to low mobility. This compound may biodegrade under aerobic conditions following acclimation. If released to acclimated water, biodegradation would be the dominant fate process (half-life less than or equal to 4 days). In nonacclimated water, bisphenol A may biodegrade after a sufficient adaption period, it may adsorb extensively to suspended solids and sediments, or it may photolyse. If released to the atmosphere, bisphenol A is expected to exist almost entirely in the particulate phase. Bisphenol A in particulate form may be removed from the atmosphere by dry deposition or photolysis. The small fraction of bisphenol A which would exist in the vapor phase may react with photochemically generated hydroxyl radicals (half-life 4 hours) or it may photolyse. Photodegradation products of bisphenol A vapor are phenol, 4-isopropylphenol, and a semiquinone derivative of bisphenol A.
Terrestrial Fate: If released to soil, bisphenol A is expected to have moderate to low mobility. Bisphenol A may biodegrade under aerobic conditions following acclimation. This compound is not expected to undergo chemical hydrolysis or volatilise significantly from soil surfaces.
Aquatic Fate: If released to acclimated water, biodegradation would be the dominant fate process (half-life less than or equal to 4 days). In nonacclimated waters, bisphenol A may biodegrade after a sufficient adaption period, it may adsorb extensively to suspended solids and sediments, or it may photolyse. This compound is not expected to bioaccumulate significantly in aquatic organisms, volatilise, or undergo chemical hydrolysis.
Atmospheric Fate: Based on the estimated vapor pressure bisphenol A is expected to exist almost entirely in the particulate phase in the atmosphere. Bisphenol A particles may be removed from the atmosphere by dry deposition or photolysis. The small fraction of bisphenol A which would exist in the vapor phase may react with photochemically generated hydroxyl radicals (half-life 4 hours) or it may photolyse. Photodegradation products of bisphenol A vapor are phenol, 4-isopropylphenol, and a semiquinone derivative of bisphenol A.
Biodegradation: Undergoes rapid biodegradation in laboratory tests using acclimated microorganisms.
Bisphenol A is "inherently biodegradable"
In waste-water treatment systems 92-98% removal is reported in activated sludges.
Bioconcentration: A bioconcentration factor (BCF) of <100 was measured for bisphenol A in carp . BCF of 42 and 196 were estimated for bisphenol A based on the water solubility and the log Kow, respectively . These BCF values indicate that bisphenol A should not bioaccumulate significantly in aquatic organisms.
Soil Adsorption/Mobility: Soil adsorption coefficients (Koc) of 314 and 1524 have been estimated for bisphenol A based on (be water solubility and the log Kow, respectively . These Koc values suggest that mobility of bisphenol A in soil would be moderate to low and that adsorption to suspended solids would be moderate to extensive .
Volatilisation from Water/Soil: The value of Henry's law constant suggests that volatilisation would be insignificant from all bodies of water . Due to its relatively low vapor pressure and its tendency to adsorb to soil, bisphenol A is not expected to volatilize significantly from wet or dry soil surfaces.
Ecotoxicity:
Moderately toxic to aquatic organisms
Fish LC50 (96 h): 42 mg/l
Environmental toxicity is a function of the n-octanol/ water partition coefficient (log Pow, log Kow). Phenols with log Pow >7.4 are expected to exhibit low toxicity to aquatic organisms. However the toxicity of phenols with a lower log Pow is variable, ranging from low toxicity (LC50 values >100 mg/l) to highly toxic (LC50 values <1 mg/l) dependent on log Pow, molecular weight and substitutions on the aromatic ring. Dinitrophenols are more toxic than predicted from QSAR estimates. Hazard information for these groups is not generally available.
For ethyleneamines:
Adsorption of the ethyleneamines correlates closely with both the cation exchange capacity (CEC) and organic content of the soil. Soils with increased CEC and organic content exhibited higher affinities for these amines. This dependence of adsorption on CEC and organic content is most likely due to the strong electrostatic interaction between the positively charged amine and the negatively charged soil surface.
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
Not Available	Not Available	Not Available

Bioaccumulative potential

Ingredient	Bioaccumulation
Not Available	Not Available

Mobility in soil

Ingredient	Mobility
Not Available	Not Available

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 MSDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: Reduction Reuse Recycling Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. **DO NOT** allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. 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 licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material). 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)

UN number	3267
Packing group	III
UN proper shipping name	CORROSIVE LIQUID, BASIC, ORGANIC, N.O.S. (contains benzene-1,3-dimethanamine,N-aminoethylpiperazine and nonylphenol)
Environmental hazard	No relevant data
Transport hazard class(es)	Class: 8 Subrisk:
Special precautions for user	Special provisions: 223 274 limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

UN number	3267
Packing group	III
UN proper shipping name	Corrosive liquid, basic, organic, n.o.s. * (contains benzene-1,3-dimethanamine,N-aminoethylpiperazine and nonylphenol)
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class: 8 ICAO / IATA Subrisk: ERG Code: 8L
Special precautions for user	Special provisions: A3A803 Cargo Only Packing Instructions: 856 Cargo Only Maximum Qty / Pack: 60 L 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	3267
Packing group	III
UN proper shipping name	CORROSIVE LIQUID, BASIC, ORGANIC, N.O.S. (contains benzene-1,3-dimethanamine,N-aminoethylpiperazine and nonylphenol)
Environmental hazard	
Transport hazard class(es)	IMDG Class: 8 IMDG Subrisk:
Special precautions for user	EMS Number: F-A,S-B Special provisions: 223 274 Limited Quantities: 5 L

SECTION 15 REGULATORY INFORMATION

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

bisphenol A/ epichlorohydrin resin(25068-38-6) is found on the following regulatory lists	"Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)","International Maritime Dangerous Goods Requirements (IMDG Code)","Australia - Victoria Occupational Health and Safety Regulations - Schedule 9: Materials at Major Hazard Facilities (And Their Threshold Quantity) Table 2","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5","WHO Model List of Essential Medicines - Adults","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","OECD List of High

	Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","Australia National Pollutant Inventory","Sigma-AldrichTransport Information","Australia High Volume Industrial Chemical List (HVICL)","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","OECD Existing Chemicals Database","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","Australia Hazardous Substances Information System - Consolidated Lists","International Air Transport Association (IATA) Dangerous Goods Regulations","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","Australia - New South Wales Protection of the Environment Operations (Waste) Regulation 2005 - Characteristics of trackable wastes"
benzene-1,3-dimethanamine(1477-55-0) is found on the following regulatory lists	"International Maritime Dangerous Goods Requirements (IMDG Code)","International Council of Chemical Associations (ICCA) - High Production Volume List","Australia Exposure Standards","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","FisherTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","Australia Inventory of Chemical Substances (AICS)","OECD List of High Production Volume (HPV) Chemicals","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","Australia National Pollutant Inventory","Sigma-AldrichTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","Australia High Volume Industrial Chemical List (HVICL)","OECD Existing Chemicals Database","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","Australia Hazardous Substances Information System - Consolidated Lists","International Air Transport Association (IATA) Dangerous Goods Regulations"
bisphenol A(80-05-7) is found on the following regulatory lists	"OSPAR List of Substances of Possible Concern","FisherTransport Information","OECD List of High Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","International Chemical Secretariat (ChemSec) SIN List (*Substitute It Now!)","Sigma-AldrichTransport Information","OECD Existing Chemicals Database","Australia Hazardous Substances Information System - Consolidated Lists"
N-aminoethylpiperazine(140-31-8) is found on the following regulatory lists	"International Maritime Dangerous Goods Requirements (IMDG Code)","International Council of Chemical Associations (ICCA) - High Production Volume List","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","OECD List of High Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","OSPAR National List of Candidates for Substitution – United Kingdom","Australia National Pollutant Inventory","Sigma-AldrichTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","OECD Existing Chemicals Database","GESAMP/EHS Composite List - GESAMP Hazard Profiles","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","Australia Hazardous Substances Information System - Consolidated Lists","International Air Transport Association (IATA) Dangerous Goods Regulations","IMO IBC Code Chapter 17: Summary of minimum requirements","Acros Transport Information"
nonylphenol(25154-52-3) is found on the following regulatory lists	"International Maritime Dangerous Goods Requirements (IMDG Code)","OSPAR List of Substances of Possible Concern","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","International Maritime Dangerous Goods Requirements (IMDG Code) - Marine Pollutants","United Nations Consolidated List of Products Whose Consumption and/or Sale Have Been Banned, Withdrawn, Severely Restricted or Not Approved by Governments","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","OECD List of High Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","International Chemical Secretariat (ChemSec) SIN List (*Substitute It Now!)","Sigma-AldrichTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","OECD Existing Chemicals Database","GESAMP/EHS Composite List - GESAMP Hazard Profiles","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","Australia Hazardous Substances Information System - Consolidated Lists","International Air Transport Association (IATA) Dangerous Goods Regulations","IMO IBC Code Chapter 17: Summary of minimum requirements","International Fragrance Association (IFRA) Survey: Transparency List"
N,N-dimethylbenzylamine(103-83-3) is found on the following regulatory lists	"Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)","International Maritime Dangerous Goods Requirements (IMDG Code)","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","Australia Inventory of Chemical Substances (AICS)","OSPAR National List of Candidates for Substitution – Norway","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","Australia National Pollutant Inventory","Sigma-AldrichTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","Australia Hazardous Substances Information System - Consolidated Lists","International Air Transport Association (IATA) Dangerous Goods Regulations","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","Acros Transport Information"
phenol(108-95-2) is found on the following regulatory lists	"International Maritime Dangerous Goods Requirements (IMDG Code)","Australia - Victoria Occupational Health and Safety Regulations - Schedule 9: Materials at Major Hazard Facilities (And Their Threshold Quantity) Table 2","IOFI Global Reference List of Chemically Defined Substances","Australia Approved Active Constituents for Agricultural Chemical Products","Australia Exposure Standards","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","FisherTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","OECD List of High Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (Aquatic habitat)","UNECE - Kiev Protocol on Pollutant Release and Transfer Registers - Annex II","Australia National Pollutant Inventory","Sigma-AldrichTransport Information","Australia Australian Pesticides and Veterinary Medicines Authority (APVM) Record of approved active constituents","Australia High Volume Industrial Chemical List (HVICL)","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","OECD Existing Chemicals Database","GESAMP/EHS Composite List - GESAMP Hazard Profiles","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","International Air Transport Association (IATA) Dangerous Goods Regulations","Australia Hazardous Substances Information System - Consolidated Lists","Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (AQUA/1 to 6 - non-pesticide anthropogenic organics)","IMO IBC Code Chapter 17: Summary of minimum requirements","International Fragrance Association (IFRA) Survey: Transparency List","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6"

SECTION 16 OTHER INFORMATION

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.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net/references

The (M)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.