Ameroyal® CF

Drew Marine

Chemwatch Hazard Alert Code: **4**

Chemwatch: **23-9021**
Version No: **3.1.1.1**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **01/01/2013**
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Initial Date: **Not Available**
S.GHS.USA.EN

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

Product Identifier

Product name	Ameroyal® CF
Chemical Name	Not Applicable
Proper shipping name	Caustic alkali liquids, n.o.s. (contains potassium hydroxide and sodium hydroxide)
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	Use according to manufacturer's directions.

Details of the manufacturer/importer

Registered company name	Drew Marine
Address	100 South Jefferson Road Whippany 07981 NJ United States
Telephone	973 526-5700.
Fax	Not Available
Website	Not Available
Email	Not Available

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	The numbers below are for EMERGENCY USE ONLY. Use the corporate number above for all other calls.
Other emergency telephone numbers	CHEMWATCH: From within the US and CANADA: 1 877-715-9305 OR call + 613 9573 3112. From outside the US and Canada: + 800 2436 2255 (+800 CHEMCALL) or +613 9573 3112

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
877 715 9305	+612 9186 1132	Not Available

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

Una vez conectado y si el mensaje no está en su idioma preferido, por favor marque 02

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

GHS Classification	Metal Corrosion Category 1, Skin Corrosion/Irritation Category 1A, Serious Eye Damage Category 1

Label elements

GHS label elements	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H290	May be corrosive to metals
H314	Causes severe skin burns and eye damage
H318	Causes serious eye damage

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Supplementary statement(s)

Not Applicable

Precautionary statement(s): Prevention

P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P234	Keep only in original container.

Precautionary statement(s): Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider
P363	Wash contaminated clothing before reuse.
P390	Absorb spillage to prevent material damage.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

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
1310-58-3	5-10	potassium hydroxide
1310-73-2	1-5	sodium hydroxide
		Note: Manufacturer has supplied full ingredient
		information to allow CHEMWATCH assessment.
7440-38-2	trace	arsenic
107-21-1	trace	ethylene glycol
	trace	mercury
7440-47-3	trace	chromium
7439-89-6	trace	iron
7440-02-0	trace	nickel
75-56-9	trace	propylene oxide
123-91-1	trace	1,4-dioxane
75-21-8	trace	ethylene oxide
7439-92-1	trace	lead

Note: Manufacturer has supplied full ingredient information to allow CHEMWATCH assessment.

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, without delay. ▸ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. ▸ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).

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	▸ 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]

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

	The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas. Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances. In such an event consider: ▸ foam. ▸ dry chemical powder. ▸ carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

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), other pyrolysis products typical of burning organic materialMay 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. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.

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Major Spills	▸ 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. ▸ 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. ▸ **WARNING:** To avoid violent reaction, **ALWAYS** add material to water and **NEVER** water to material. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this MSDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this 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	Sodium hydroxide/ potassium hydroxide: ▸ reacts with water evolving heat and corrosive fumes ▸ reacts violently with acids, trans-acetylene dichloride, aminotetrazole, p-bis(1,3-dibromoethyl), benzene, bromoform, halogenated compounds, nitrogen-containing compounds, organic halogens, chlorine dioxide ((explodes), chloroform, cresols, cyclopentadiene, 4-chloro-2-methylphenol, cis-dichloroethylene, 2,2-dichloro-3,3-dimethylbutane, ethylene chlorohydrin, germanium, iodine pentafluoride, maleic anhydride, p-nitrotoluene,nitrogen trichloride, o-nitrophenol, phosphonium iodide, potassium peroxodisulfate, propylene oxide, 1,2,4,5-tetrachlorobenzene (highly toxic substance is forme), 2,2,3,3-tetrafluoro-1-propanol, tetrahydrofuran, thorium dicarbide, trichloroethanol, 2,4,6-trinitrotoluene, vinyl acetate ▸ reacts with fluorine, nitroalkanes, (forming explosive compounds) ▸ incompatible with acetic acid, acetaldehyde, acetic anhydride, acrolein, acrylonitrile, allyl chloride, organic anhydride, acrylates, alcohols, aldehydes, alkylene oxides, substituted allyls, ammonium chloroplatinate, benzanthrone, bromine, benzene-1,4-diol, carbon dioxide, cellulose nitrate, chlorine trifluoride, 4-chlorobutyronitrile, chlorohydrin, chloronitrotoluenes, chlorosulfonic acid, cinnamaldehyde, caprolactam solution, chlorocresols, 1,2-dichloroethylene, epichlorohydrin, ethylene cyanohydrin, formaldehyde (forms formic acid and flammable hydrogen gas), glycols, glyoxal, hexachloroplatinate, hydrogen sulfide, hydroquinone, iron-silicon, isocyanates, ketones, methyl azide, 4-methyl-2-nitrophenol, mineral acids (forming corresponding salt),nitrobenzene, N-nitrosohydroxylamine, nitrates pentol, phenols, phosphorus, phosphorus pentaoxide, beta-propiolactone, sodium, sulfur dioxide, tetrahydroborate, 1,1,1,2-tetrachloroethane, 2,2,2-trichloroethanol, trichloronitromethane, zirconium ▸ ignites on contact with cinnamaldehyde or zinc and reacts explosively with a mixture of chloroform and methane ▸ forms heat-, friction-, and/ or shock-sensitive- explosive salts with nitro-compounds, cyanogen azide, 3-ethyl-4-hydroxy-1,2,5-oxadiazole, 3-methyl-2-penten-4-yn-1-ol, N,N'-bis(2,2,2-trinitroethyl)urea, trichloroethylene (forms dichloroacetylene) ▸ increase the explosive sensitivity of nitromethane ▸ attacks some plastics, rubber, coatings and metals: aluminium, tin, zinc,etc, and their alloys, producing flammable hydrogen gas ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ▸ Avoid contact with copper, aluminium and their alloys.

PACKAGE MATERIAL INCOMPATIBILITIES

Continued...

Not Available

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US ACGIH Threshold Limit Values (TLV)	potassium hydroxide	Potassium hydroxide	Not Available	Not Available	2 mg/m3	TLV® Basis: URT, eye, & skin irr
US NIOSH Recommended Exposure Limits (RELs)	potassium hydroxide	Caustic potash, Lye, Potassium hydrate	Not Available	Not Available	2 mg/m3	Not Available
US OSHA Permissible Exposure Levels (PELs) - Table Z1	sodium hydroxide	Sodium hydroxide	2 mg/m3	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	sodium hydroxide	Sodium hydroxide	Not Available	Not Available	2 mg/m3	TLV® Basis: URT, eye, & skin irr
US NIOSH Recommended Exposure Limits (RELs)	sodium hydroxide	Caustic soda, Lye, Soda lye, Sodium hydrate	Not Available	Not Available	2 mg/m3	Not Available
US OSHA Permissible Exposure Levels (PELs) - Table Z1	arsenic	Arsenic-inorganic compounds	0.01 mg/m3	Not Available	Not Available	see 1910.1018;(as As)
US ACGIH Threshold Limit Values (TLV)	arsenic	Arsenic and inorganic compounds, as As	0.01 mg/m3	Not Available	Not Available	TLV® Basis: Lung cancer; BEI
US NIOSH Recommended Exposure Limits (RELs)	arsenic	Arsenic metal: Arsenia	Not Available	Not Available	0.002 mg/m3	Ca See Appendix A
US ACGIH Threshold Limit Values (TLV)	ethylene glycol	‡ Ethylene glycol	Not Available	Not Available	100 mg/m3	TLV® Basis: URT & eye irr
US NIOSH Recommended Exposure Limits (RELs)	ethylene glycol	1,2-Dihydroxyethane; 1,2-Ethanediol; Glycol; Glycol alcohol; Monoethylene glycol	Not Available	Not Available	Not Available	See Appendix D
US OSHA Permissible Exposure Levels (PELs) - Table Z1	chromium	Chromium metal and insol. salts	1 mg/m3	Not Available	Not Available	(as Cr)
US ACGIH Threshold Limit Values (TLV)	chromium	Chromium, and inorganic compounds, as Cr - Metal and Cr III compounds	0.5 mg/m3	Not Available	Not Available	TLV® Basis: URT & skin irr
US NIOSH Recommended Exposure Limits (RELs)	chromium	Chrome, Chromium	0.5 mg/m3	Not Available	Not Available	See Appendix C
US OSHA Permissible Exposure Levels (PELs) - Table Z3	iron	Inert or Nuisance Dust	5 mg/m3 / 15 mg/m3 / 15 mppcf / 50 mppcf	Not Available	Not Available	Respirable fraction;All inert or nuisance dusts, whether mineral, inorganic, or organic, not listed specifically by substance name are covered by this limit, which is the same as the Particulates Not Otherwise Regulated (PNOR) limit in Table Z-1. / Total dust;All inert or nuisance dusts, whether mineral, inorganic, or organic, not listed specifically by substance name are covered by this limit, which is the same as the Particulates Not Otherwise Regulated (PNOR) limit in Table Z-1.
US OSHA Permissible Exposure Levels (PELs) - Table Z1	nickel	Nickel, metal and insoluble compounds	1 mg/m3	Not Available	Not Available	(as Ni)
US ACGIH Threshold Limit Values (TLV)	nickel	Nickel and inorganic compounds including Nickel subsulfide, as Ni - Elemental	1.5 mg/m3	Not Available	Not Available	TLV® Basis: Dermatitis; pneumoconiosis
US NIOSH Recommended Exposure Limits (RELs)	nickel	Nickel metal: Elemental nickel, Nickel catalyst	0.015 mg/m3	Not Available	Not Available	Ca See Appendix A [*Note: The REL does not apply to Nickel carbonyl.]
US OSHA Permissible Exposure Levels (PELs) - Table Z1	propylene oxide	Propylene oxide	240 mg/m3 / 100 ppm	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	propylene oxide	* Propylene oxide	2 ppm	Not Available	Not Available	TLV® Basis: Eye & URT irr
US NIOSH Recommended Exposure Limits (RELs)	propylene oxide	1,2-Epoxy propane; Methyl ethylene oxide; Methyloxirane; Propene oxide; 1,2-Propylene oxide	Not Available	Not Available	Not Available	Ca See Appendix A
US OSHA Permissible Exposure Levels (PELs) - Table Z1	1,4-dioxane	Dioxane (Diethylene dioxide)	360 mg/m3 / 100 ppm	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	1,4-dioxane	1.4-Dioxane	20 ppm	Not Available	Not Available	TLV® Basis: Liver dam
US NIOSH Recommended Exposure Limits (RELs)	1,4-dioxane	Diethylene dioxide; Diethylene ether; Dioxan; p-Dioxane; 1,4-Dioxane	Not Available	Not Available	3.6 mg/m3 / 1 ppm	Ca See Appendix A

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US OSHA Permissible Exposure Levels (PELs) - Table Z1	ethylene oxide	Ethylene oxide	1 ppm	5 ppm	Not Available	see 1910.1012;(STEL (Excursion limit)(as averaged over a sampling period of 15 minutes))
US ACGIH Threshold Limit Values (TLV)	ethylene oxide	Ethylene oxide	1 ppm	Not Available	Not Available	TLV® Basis: Cancer; CNS impair
US NIOSH Recommended Exposure Limits (RELs)	ethylene oxide	Dimethylene oxide; 1,2-Epoxy ethane; Oxirane	0.18 mg/m3 / 0.1 ppm	Not Available	9 mg/m3 / 5 ppm	Ca See Appendix A
US OSHA Permissible Exposure Levels (PELs) - Table Z1	lead	Lead, inorganic	0.05 mg/m3	Not Available	Not Available	(as Pb);see 1910.1025;If an employee is exposed to lead for more than 8 hours in any work day, the permissible exposure limit, as a time weighted average (TWA) for that day, shall be reduced according to the following formula: Maximum permissible limit (in µg/m3)=400÷hours worked in the day.
US ACGIH Threshold Limit Values (TLV)	lead	Lead and inorganic compounds, as Pb	0.05 mg/m3	Not Available	Not Available	TLV® Basis: CNS & PNS impair; hematologic eff; BEI
US NIOSH Recommended Exposure Limits (RELs)	lead	Lead metal, Plumbum	0.050 mg/m3	Not Available	Not Available	See Appendix C [*Note: The REL also applies to other lead compounds (as Pb) -- see Appendix C.]

EMERGENCY LIMITS

Ingredient	TEEL-0	TEEL-1	TEEL-2	TEEL-3
Ameroyal® CF	Not Available	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
potassium hydroxide	Not Available	Not Available
sodium hydroxide	250 mg/m3	10 mg/m3
arsenic	100 mg/m3	5 mg/m3
ethylene glycol	Not Available	Not Available
chromium	N.E. mg/m3 / N.E. ppm	250 mg/m3
iron	Not Available	Not Available
nickel	N.E. mg/m3 / N.E. ppm	10 mg/m3
propylene oxide	2,000 ppm	400 ppm
1,4-dioxane	2,000 ppm	500 ppm
ethylene oxide	800 ppm	800 [Unch] ppm
lead	700 mg/m3	100 mg/m3

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.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection.
Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
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.

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Personal protection	
Eye and face protection	▸ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ▸ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ▸ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ▸ Alternatively a gas mask may replace splash goggles and face shields. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ Elbow length PVC gloves ▸ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. 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:
Ameroyal® CF

Material	CPI
BUTYL	A
NEOPRENE	A
NATURAL RUBBER	C

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

Respiratory protection

Type AX-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 5 x ES	AX-AUS / Class 1 P2	-	AX-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	AX-2 P2	AX-PAPR-2 P2
up to 50 x ES	-	AX-3 P2	-
50+ x ES	-	Air-line**	-

* - Continuous-flow; ** - Continuous-flow or positive pressure demand
^ - 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	Light yellow corrosive liquid; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.125
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available

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Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	13	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	0	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	100	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	93.4	**Taste**	Not Available
Evaporation rate	>1 Ether = 1	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	2.3 @ 20C	**Gas group**	Not Available
Solubility in water (g/L)	Miscible	**pH as a solution(1%)**	Not Available
Vapour density (Air = 1)	>1	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation. Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	The material can produce severe chemical burns following direct contact with the skin. Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight.
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. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

Ameroyal® CF	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

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	TOXICITY	IRRITATION
potassium hydroxide	Oral (rat) LD50: 273 mg/kg	Eye (rabbit):1mg/24h rinse-moderate
		Skin (human): 50 mg/24h SEVERE
		Skin (rabbit): 50 mg/24h SEVERE
	Not Available	Not Available
sodium hydroxide	**TOXICITY**	**IRRITATION**
		Eye (rabbit): 0.05 mg/24h SEVERE
		Eye (rabbit):1 mg/24h SEVERE
		Eye (rabbit):1 mg/30s rinsed-SEVERE
		Skin (rabbit): 500 mg/24h SEVERE
	Not Available	Not Available
arsenic	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
ethylene glycol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 9530 mg/kg	Eye (rabbit): 100 mg/1h - mild
	Inhalation (rat) LC50: 50100 mg/m3/8 hr	Eye (rabbit): 12 mg/m3/3D
	Oral (rat) LD50: 4700 mg/kg	Eye (rabbit): 1440mg/6h-moderate
		Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 555 mg(open)-mild
	Not Available	Not Available
chromium	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
iron	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 98600 mg/kg	[Patty]
	Not Available	Not Available
nickel	**TOXICITY**	**IRRITATION**
	Intravenous (dog) LD50: 40 mg/kg	
	Not Available	Not Available
propylene oxide	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 1245 mg/kg	Eye (rabbit): 20 mg/24h moderate
	Oral (rat) LD50: 380 mg/kg	Eye (rabbit): 5 mg SEVERE
		Skin (rabbit): 50 mg/6m SEVERE
		Skin (rabbit):415 mg open moderate
	Not Available	Not Available
1,4-dioxane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 7600 mg/kg	Eye(human): 300 ppm/15m
	Oral (rat) LD50: 4200 mg/kg	Eye(rabbit): 21 mg (int)-irritant
		Skin(rabbit): 515 mg (open)-mild
	Not Available	Not Available
ethylene oxide	**TOXICITY**	**IRRITATION**
	Inhalation (rat) LC50: 800 ppm/4 hr	Eye (rabbit): 18 mg/6h - moderate
	Oral (rat) LD50: 72 mg/kg	Skin (human): 1%/7 sec - irritant
	Not Available	Not Available
lead	**TOXICITY**	**IRRITATION**
		Nil Reported
	Not Available	Not Available

Not available. Refer to individual constituents.

POTASSIUM HYDROXIDE	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

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	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 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.
SODIUM HYDROXIDE	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.
ARSENIC	Arsenic compounds are classified by the European Union as toxic by inhalation and ingestion and toxic to aquatic life and long lasting in the environment. IARC classify arsenic in drinking water as a confirmed human carcinogen (IARC 1). The main inorganic forms of arsenic relevant for human exposures are pentavalent arsenic (also called arsenate, As(V), or As+5) and trivalent arsenic (also called arsenite, As(III), or As+3). These inorganic species undergoes a series of reduction and oxidative/methylation steps in human liver and other tissues to form tri- and pentavalent methylated metabolites of methylarsonite [MA(III)], methylarsonate [MA(V)], dimethylarsinite [DMA(III)], and dimethylarsinate [DMA(V)]. Some mammalian species also produce trimethylated metabolites, trimethylarsine oxide The distinction between inorganic and organic forms is important because it is generally accepted that the organic species are excreted more quickly from the body and generally considered less toxic, with a relative rank order of As(III) > As(V) >> MA(V), DMA(V) >> arsenobetaine. However, the methylated trivalent metabolites, MA(III) and DMA(III), are significantly more toxic than their pentavalent counterpart and either As(III) or As(V) . In many cases, biomonitoring or environmental occurrence data are reported as total arsenic and do not distinguish between the different species. In those situations, understanding the relevant sources of arsenic is essential to evaluate potential arsenic related health effects, especially those related to inorganic arsenic exposure. **WARNING:** This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**. Tumorigenic - Carcinogenic by RTECS criteria.
ETHYLENE GLYCOL	For ethylene glycol: Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol. dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO_2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO_2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested. **Respiratory Effects.** Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases). **Cardiovascular Effects.** Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases.Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown. **Gastrointestinal Effects.** Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition. **Musculoskeletal Effects.** Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia. **Hepatic Effects.** Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. **Renal Effects.** Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads

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	to haematuria, proteinuria, decreased renal function, oliguria, anuria , and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy. **Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate). **Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion. Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months. **Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration. **Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight. **Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. **Genotoxic Effects**: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol. [Estimated Lethal Dose (human) 100 ml; RTECS quoted by Orica] Substance is reproductive effector in rats (birth defects). Mutagenic to rat cells.
CHROMIUM	For chrome(III) and other valence states (except hexavalent): For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases. The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells. enter cells The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental, divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter though these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures. The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage. Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms. No significant acute toxicological data identified in literature search. 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. Tenth Annual Report on Carcinogens: Substance known to be Carcinogenic [*National Toxicology Program: U.S. Dep. of Health and Human Services 2002*] Gastrointestinal tumours, lymphoma, musculoskeletal tumours and tumours at site of application recorded.
NICKEL	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. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*] Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m3/24H/17W-C

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PROPYLENE OXIDE	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 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. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
1,4-DIOXANE	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 1,4-dioxane: Acute toxic effects reported in animals are mainly CNS depression, kidney and liver damage. Overt CNS effects (including convulsions) have been reported in rabbits administered (i.v.) 5 ml (2060 mg/kg) of 1,4-dioxane in solution. Subtle effects on CNS function, as assessed by perturbations of certain neurotransmitters in male rats (Sprague-Dawley) have been reported following an oral dose of 1050 mg/kg 1,4-dioxane . A study of the effects of 1,4-dioxane on electrically evoked seizure discharge, considered to be a sensitive indicator of neurotropic effects, revealed that a 30% depression in response following inhalation of 1860 ppm (4 hr) in rats and 2400 ppm (2 hr) in mice Slight dermal erythema and severe scale formation were reported in rabbits up to 8 days after dermal application of 1,4-dioxane (dose not reported). Mild irritation was also observed in rabbit skin following an application of 515 mg 1,4-dioxane in an open Draize Test . However, skin irritation was not seen in rats exposed (unoccluded) to 8,300 mg/kg 1,4-dioxane. Evidence of skin irritation was not seen in guinea pigs, rabbits or mice following repeated (studies ranging from 50-100 days) dermal exposure to 1,4-dioxane (above 50 mg) applied two or three times per day 1,4-Dioxane has been reported to have a miotic effect in rabbits at concentrations below that causing alterations in the conjunctiva or cornea, with pupils returning to normal 10 to 15 minutes after administration . Liquid 1,4-dioxane has been reported to cause eye irritation in rabbits Irritation of the nose and lung has been reported following inhalation of 1,4-dioxane (>2000 ppm) in guinea pigs, mice and cats An inhalation study has been carried out in human volunteers exposed to 50 ppm (180 mg/m3) 1,4-dioxane for 6 hr. In this study absorption of 1,4-dioxane was estimated at around 80% of dose. The maximum uptake (10.9 mg/kg) was around 50% of that measured in rats following similar exposure . Because of its rapid biotransformation to b-hydroxyethoxyacetic acid (HEAA), the body burden of 1,4-dioxane was estimated to be no more than 1.2 mg/kg at steady state. No data were available for dermal uptake for 1,4-dioxane in humans (*in vivo*), although skin absorption was considered a potential route of exposure in case reports of human fatalities from short term exposures . Significant differences exist for dermal penetration (in diffusion cell studies on human skin) of 1,4-dioxane under occluded and non-occluded conditions. Up to 3.2% of applied 1,4-dioxane (dissolved in lotion) was absorbed under occlusion for 3.5 hr, whereas only 0.3% absorption occurred under non-occluded conditions. Differences in the amount of absorption were accounted for by the high volatility of 1,4-dioxane . A permeability constant (Kp) of 2.7 x 10-4 cm/hr was determined for the occluded test system which is similar to that calculated for undiluted 1,4-dioxane. The absorption rate for 1,4-dioxane (under occlusion) was calculated to be approximately 0.3 mg/cm2/hr which compares with other solvents reported as being readily absorbed in *in vitro* skin (human) tests. From the solubility characteristics alone, it is predicted that 'considerable uptake' by the skin could be expected for 1,4-dioxane, but that oxidation and evaporation from the skin surface would limit the total amount absorbed. Almost 90% (as a percentage of applied dose) evaporation of 1,4-dioxane in a lotion was demonstrated within 15 minutes of application (to a non-absorbent test material), with the remainder evaporating over the next 24 hr. Sub-acute and sub-chronic studies for 1,4-dioxane have been carried out in rats, mice, guinea pigs, rabbits, dogs and cats. Effects reported include narcosis, behavioural changes, haematological effects, cardiac effects and histopathological lesions of the kidneys, liver and brain. In general, the doses used in these studies are very high and as such provide little useful information on critical effects (i.e., most sensitive effects) and no observed adverse effect levels (NOAELs). **Metabolism and elimination** The major metabolite of 1,4-dioxane in humans is beta-hydroxyethanoxyacetic acid (HEAA). Four volunteers were exposed to 50 ppm 1,4-dioxane for 6 hr A steady state plasma level of 10 ug/m 1,4-dioxane was reached after 3 hr inhalation exposure, with a steady state plasma concentration of 8 mg/ml HEAA reached 1 hr after cessation of exposure (i.e., after 7 hr). The plasma half-lives for 1,4-dioxane and HEAA were around 1 and 2.5 hr respectively. HEAA accounted for around 99% of recovered 1,4-dioxane in urine. Clearance of 1,4-dioxane from kidneys was around 400 times slower than HEAA. 1,4-Dioxane may inhibit the oxidative metabolism of other substances as it has been shown to inhibit human CYP2A6 activity in liver microsomes *in vitro* 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. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Brain degenerative changes, kidney tubule changes, urine volume changes, lymphoma including Hodgkin's disease recorded.
ETHYLENE OXIDE	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

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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 material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative.
for 1,2-butylene oxide (ethyloxirane):
Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic
for ethylene oxide:
Ethylene oxide is very soluble in blood. Therefore, pulmonary uptake is expected to be fast and to depend only on the alveolar ventilation rate and the concentration of ethylene oxide in the inspired air. The rate of uptake of ethylene oxide in mice was 1.1 ug/kg body weight, per min, at an exposure level of 1 mg/m3. This corresponds to nearly 100% absorption of ethylene oxide from 1.1 litre of air per min and per kg body weight, which is the reported rate of alveolar ventilation in resting mice. No specific information pertaining to skin absorption is available, but accidental exposure of the skin of 3 industrial workers to 1% aqueous solution of ethylene oxide was reported to have resulted in marked nausea and profuse vomiting
Human exposure mainly occurs through inhalation in sterilisation facilities and in production plants. In sterilisation facilities, 8-h time-weighted average levels have usually been below 36 mg/m3, with short-term exposures of about 100 mg/m3, and peak levels of up to 1800 mg/m3. In production plants, the time-weighted average has usually been below 4 mg/m3. Ambient levels at a distance from point sources of emission have been estimated to be below the limit of detection. Exposure to residues of ethylene oxide or its reaction products, halohydrins and ethylene glycol, also occurs from fumigated foods, pharmaceutical products, and sterilised medical equipment. 2-Chloroethanol levels as high as several g/kg have been measured in food and levels of several hundred mg/kg in medical equipment.
When inhaled, ethylene oxide is readily absorbed, distributed throughout the body, and rapidly metabolized. Accordingly, most organs receive equivalent doses of the chemical and its metabolites. The degree of alkylation of proteins and DNA varies slightly between the different organs and blood. In man and rodents, the half-life of the compound in tissues has been estimated to be 9 - 10 min. Two metabolic pathways have been identified including hydrolysis to 1,2-ethanediol and conjugation with glutathione. Excretion is primarily via the urine. Ethylene oxide is moderately toxic for mammals (the LD50 for the rat is 280 - 365 mg/kg body weight; the 4-h LC50 is 2630 mg/m3). Both experimental animal and human data show that aqueous solutions of ethylene oxide are irritating for the skin and eyes; the irritant effects of ethylene oxide vapour or residues in medical equipment on the eyes and the respiratory tract have also been observed. These effects are often delayed. Severe skin irritation is characterized by the formation of vesicles. A concentration of 10 mg/litre produced mild irritation of the human skin; a concentration of 500 g/litre was most injurious to the human skin. Allergic contact dermatitis has been reported; systemic immunologically mediated allergy is considered rare. Respiratory tract irritation increases with inhaled vapour concentration and may result in severe life-threatening pulmonary disease. Repeated exposure (2 - 8 weeks) to ethylene oxide vapour at or above 900 mg/m3 produced sensory and motor neurological impairment and may result in a peripheral neuropathy. In animals, the latter was often accompanied by muscular atrophy. Lesions in the medulla oblongata of monkeys, following 2 years of intermittent exposure (7 h/day, 5 days/week) to 90 and 180 mg/m3 indicated neuropathy in the brain, which may be related to the neuropathies observed in man and other animal species. Cardiovascular collapse and renal failure have been attributed to residues of ethylene oxide in medical equipment. Ethylene oxide alkylates DNA and is mutagenic for plants, microorganisms, insects, and mammals. Cytogenetic studies on man have shown dose-related increased frequencies of both sister chromatid exchanges (SCEs) and chromosomal aberrations; in one study, SCEs developed following daily exposure for less than 5 min per day.
The evidence that ethylene oxide is a reproductive toxin is less conclusive. Where foetal developmental effects have occurred, the doses of ethylene oxide approached or equalled those producing maternal toxicity. To date, impaired male reproductive function in animals has been demonstrated only at concentrations of 90 mg/m3 or more in long-term intermittent exposures or at higher air concentrations for brief exposures. In pregnant women, the results of one study suggest that occupational exposure estimated to be an 8-h time-weighted average of 0.18 - 0.90 mg/m3, with peak concentrations up to 450 mg/m3, was associated with spontaneous abortions. However, limited exposure data prevents the establishment of a relationship between abortion rates and exposure levels. Ethylene oxide is carcinogenic for animals when administered by the intragastric, subcutaneous injection, and inhalation routes of exposure. In man, 2 studies have shown an association between ethylene oxide exposure and an excess risk of cancer, but both studies have limitations. Airborne concentrations of ethylene oxide in the 2 studies were reported to be time-weighted averages of 36 +/-18 mg/m3 and 10 - 50 mg/m3, with occasional brief exposures in excess of the odour threshold (900 - 1260 mg/m3).

WARNING: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**.
Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen
[*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]

LEAD	WARNING: Lead is a cumulative poison and has the potential to cause abortion and intellectual impairment to unborn children of pregnant workers.

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

Legend: ✔ *– Data required to make classification available*
✘ *– Data available but does not fill the criteria for classification*
⊘ *– Data Not Available to make classification*

CMR STATUS

REPROTOXIN	arsenic	ILO Chemicals in the electronics industry that have toxic effects on reproduction	H s
	nickel	ILO Chemicals in the electronics industry that have toxic effects on reproduction	A
	ethylene oxide	ILO Chemicals in the electronics industry that have toxic effects on reproduction	A
	lead	ILO Chemicals in the electronics industry that have toxic effects on reproduction	H A si
CARCINOGEN	arsenic	US NIOSH Recommended Exposure Limits (RELs) - Carcinogens|US Environmental Defense Scorecard Recognized Carcinogens|US Environmental Defense Scorecard Suspected Carcinogens	Ca See Appendix A|P65-MC|P65|HAZMAP, IARC

Continued...

	chromium	US Environmental Defense Scorecard Suspected Carcinogens	HAZMAP, SCDM|P65-MC|HAZMAP, P65-MC
	iron	US Environmental Defense Scorecard Suspected Carcinogens	P65-MC
	nickel	US NIOSH Recommended Exposure Limits (RELs) - Carcinogens|US Environmental Defense Scorecard Suspected Carcinogens|US Environmental Defense Scorecard Recognized Carcinogens|US Air Toxics Hot Spots TSD for Describing Available Cancer Potency Factors	Ca See Appendix A [*Note: The REL does not apply to Nickel carbonyl.]|P65|P65-MC|1,2B(N2)
	propylene oxide	US NIOSH Recommended Exposure Limits (RELs) - Carcinogens	Ca See Appendix A|2B|P65
	1,4-dioxane	US NIOSH Recommended Exposure Limits (RELs) - Carcinogens	Ca See Appendix A|2B|P65
	ethylene oxide	US NIOSH Recommended Exposure Limits (RELs) - Carcinogens	Ca See Appendix A|1|P65
	lead	US Environmental Defense Scorecard Recognized Carcinogens|US Environmental Defense Scorecard Suspected Carcinogens|US Air Toxics Hot Spots TSD for Describing Available Cancer Potency Factors	P65|P65-MC|2B

RESPIRATORY	sodium hydroxide	US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs) - Respiratory	X
	arsenic	US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs) - Respiratory	X
	ethylene glycol	US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs) - Respiratory	X
	iron	US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs) - Respiratory	X
	nickel	US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs) - Respiratory	X
	propylene oxide	US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs) - Respiratory	X
	1,4-dioxane	US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs) - Respiratory	X

SKIN	sodium hydroxide	US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs) - Skin	X
	arsenic	US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs) - Skin	X
	1,4-dioxane	US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants - Skin|US - Alaska Limits for Air Contaminants - Skin Designation	X|Yes|S

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

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
sodium hydroxide	high	high
ethylene glycol	low (Half-life = 24 days)	low (Half-life = 3.46 days)
propylene oxide	high	high
1,4-dioxane	high (Half-life = 360 days)	low (Half-life = 3.38 days)
ethylene oxide	low (Half-life = 11.88 days)	high (Half-life = 381.96 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
sodium hydroxide	low (BCF = 3.162)
ethylene glycol	low (BCF = 3.162)
propylene oxide	low (BCF = 3.162)
1,4-dioxane	low (BCF = 0.7)
ethylene oxide	low (BCF = 3.162)

Mobility in soil

Ingredient	Mobility
sodium hydroxide	low (KOC = 14.3)
ethylene glycol	high (KOC = 1)
propylene oxide	medium (KOC = 2.324)
1,4-dioxane	high (KOC = 1)
ethylene oxide	high (KOC = 1.435)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	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

Continued...

- 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	NO

Land transport (DOT)

UN number	1719
Packing group	II
UN proper shipping name	Caustic alkali liquids, n.o.s. (contains potassium hydroxide and sodium hydroxide)
Environmental hazard	No relevant data
Transport hazard class(es)	Class: 8
Special precautions for user	Special provisions: B2, IB2, T11, TP2, TP27

Air transport (ICAO-IATA / DGR)

UN number	1719
Packing group	II
UN proper shipping name	Caustic alkali liquid, n.o.s. * (contains potassium hydroxide and sodium hydroxide)
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class: 8 ICAO / IATA Subrisk: Not Applicable ERG Code: 8L
Special precautions for user	Special provisions: A3A803 Cargo Only Packing Instructions: 855 Cargo Only Maximum Qty / Pack: 30 L Passenger and Cargo Packing Instructions: 851 Passenger and Cargo Maximum Qty / Pack: 1 L Passenger and Cargo Limited Quantity Packing Instructions: Y840 Passenger and Cargo Limited Maximum Qty / Pack: 0.5 L

Sea transport (IMDG-Code / GGVSee)

UN number	1719
Packing group	II
UN proper shipping name	CAUSTIC ALKALI LIQUID, N.O.S. (contains potassium hydroxide and sodium hydroxide)
Environmental hazard	No relevant data
Transport hazard class(es)	IMDG Class: 8 IMDG Subrisk: Not Applicable
Special precautions for user	EMS Number: F-A , S-B Special provisions: 274 Limited Quantities: 1 L

Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code

Continued...

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	potassium hydroxide	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	ethylene glycol	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	propylene oxide	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	1,4-dioxane	Y

SECTION 15 REGULATORY INFORMATION

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

potassium hydroxide(1310-58-3) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - California Permissible Exposure Limits for Chemical Contaminants","US - Hawaii Air Contaminant Limits","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - Michigan Exposure Limits for Air Contaminants","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US - Minnesota Permissible Exposure Limits (PELs)","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory"
sodium hydroxide(1310-73-2) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - California Permissible Exposure Limits for Chemical Contaminants","US - Idaho - Limits for Air Contaminants","US - Hawaii Air Contaminant Limits","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - Michigan Exposure Limits for Air Contaminants","US - Oregon Permissible Exposure Limits (Z-1)","US - Washington Toxic air pollutants and their ASIL, SQER and de minimis emission values","US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs)","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US - Minnesota Permissible Exposure Limits (PELs)","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants","US OSHA Permissible Exposure Levels (PELs) - Table Z1"
arsenic(7440-38-2) is found on the following regulatory lists	"US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)","US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - California Permissible Exposure Limits for Chemical Contaminants","US - Idaho - Limits for Air Contaminants","US - Hawaii Air Contaminant Limits","US ACGIH Threshold Limit Values (TLV) - Carcinogens","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - Michigan Exposure Limits for Air Contaminants","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","US - Oregon Permissible Exposure Limits (Z-1)","US - Washington Toxic air pollutants and their ASIL, SQER and de minimis emission values","US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs)","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US National Toxicology Program (NTP) 12th Report Part A Known to be Human Carcinogens","US - Minnesota Permissible Exposure Limits (PELs)","US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs)","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants","US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Carcinogens","US OSHA Permissible Exposure Levels (PELs) - Table Z1"
ethylene glycol(107-21-1) is found on the following regulatory lists	"US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)","US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - California Permissible Exposure Limits for Chemical Contaminants","US - Hawaii Air Contaminant Limits","US ACGIH Threshold Limit Values (TLV) - Carcinogens","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - Michigan Exposure Limits for Air Contaminants","US - Oregon Permissible Exposure Limits (Z-1)","US - Washington Toxic air pollutants and their ASIL, SQER and de minimis emission values","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US ACGIH Threshold Limit Values (TLV) - Notice of Intended Changes","US Spacecraft Maximum Allowable Concentrations (SMACs) for Airborne Contaminants","US - Minnesota Permissible Exposure Limits (PELs)","US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs)","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory"
chromium(7440-47-3) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - California Permissible Exposure Limits for Chemical Contaminants","US - Idaho - Limits for Air Contaminants","US - Hawaii Air Contaminant Limits","US ACGIH Threshold Limit Values (TLV) - Carcinogens","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - Michigan Exposure Limits for Air Contaminants","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","US - Oregon Permissible Exposure Limits (Z-1)","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants","US OSHA Permissible Exposure Levels (PELs) - Table Z1"
iron(7439-89-6) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - California Permissible Exposure Limits for Chemical Contaminants","US - Hawaii Air Contaminant Limits","US - Michigan Exposure Limits for Air Contaminants","US OSHA Permissible Exposure Levels (PELs) - Table Z3","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","US - Oregon Permissible Exposure Limits (Z-1)","US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs)","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants"
nickel(7440-02-0) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)","US - Idaho - Limits for Air Contaminants","US - Hawaii Air Contaminant Limits","US - California Permissible Exposure Limits for Chemical Contaminants","US ACGIH Threshold Limit Values (TLV) - Carcinogens","US - Michigan Exposure Limits for Air Contaminants","US National Toxicology Program (NTP) 12th Report Part B. Reasonably Anticipated to be a Human Carcinogen","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - Oregon Permissible Exposure Limits (Z-1)","US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs)","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US - California Proposition 65 - Carcinogens","US National Toxicology Program (NTP) 12th Report Part A Known to be Human Carcinogens","US Priority List for the Development of Proposition 65 Safe Harbor Levels - No Significant Risk Levels (NSRLs) for Carcinogens and Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity","US - Minnesota Permissible Exposure Limits (PELs)","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs)","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants","US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Carcinogens","US OSHA Permissible Exposure Levels (PELs) - Table Z1"

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propylene oxide(75-56-9) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - Idaho - Limits for Air Contaminants","US - Hawaii Air Contaminant Limits","US - California Permissible Exposure Limits for Chemical Contaminants","US ACGIH Threshold Limit Values (TLV) - Carcinogens","US - Michigan Exposure Limits for Air Contaminants","US EPA Carcinogens Listing","US National Toxicology Program (NTP) 12th Report Part B. Reasonably Anticipated to be a Human Carcinogen","US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Mutagens","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","US - Oregon Permissible Exposure Limits (Z-1)","US - Washington Toxic air pollutants and their ASIL, SQER and de minimis emission values","US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs)","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US - California Proposition 65 - Carcinogens","US National Toxicology Program (NTP) 12th Report Part A Known to be Human Carcinogens","US Priority List for the Development of Proposition 65 Safe Harbor Levels - No Significant Risk Levels (NSRLs) for Carcinogens and Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity","US - Minnesota Permissible Exposure Limits (PELs)","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs)","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants","US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Carcinogens","US OSHA Permissible Exposure Levels (PELs) - Table Z1"
1,4-dioxane(123-91-1) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)","US - Idaho - Limits for Air Contaminants","US - Hawaii Air Contaminant Limits","US - California Permissible Exposure Limits for Chemical Contaminants","US - California Proposition 65 - No Significant Risk Levels (NSRLs) for Carcinogens","US ACGIH Threshold Limit Values (TLV) - Carcinogens","US - Michigan Exposure Limits for Air Contaminants","US EPA Carcinogens Listing","US National Toxicology Program (NTP) 12th Report Part B. Reasonably Anticipated to be a Human Carcinogen","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - California - Proposition 65 - Priority List for the Development of MADLs for Chemicals Causing Reproductive Toxicity","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","US - Oregon Permissible Exposure Limits (Z-1)","US - Washington Toxic air pollutants and their ASIL, SQER and de minimis emission values","US - California OEHHA/ARB - Acute Reference Exposure Levels and Target Organs (RELs)","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US - California Proposition 65 - Carcinogens","US National Toxicology Program (NTP) 12th Report Part A Known to be Human Carcinogens","US - Minnesota Permissible Exposure Limits (PELs)","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs)","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants","US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Carcinogens","US OSHA Permissible Exposure Levels (PELs) - Table Z1"
ethylene oxide(75-21-8) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - California Proposition 65 - Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity","US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)","US - Idaho - Limits for Air Contaminants","US - Hawaii Air Contaminant Limits","US - California Permissible Exposure Limits for Chemical Contaminants","US - California Proposition 65 - No Significant Risk Levels (NSRLs) for Carcinogens","US ACGIH Threshold Limit Values (TLV) - Carcinogens","US - California Proposition 65 - Reproductive Toxicity","US - Michigan Exposure Limits for Air Contaminants","US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Mutagens","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - California - Proposition 65 - Priority List for the Development of MADLs for Chemicals Causing Reproductive Toxicity","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","US - Oregon Permissible Exposure Limits (Z-1)","US - Washington Toxic air pollutants and their ASIL, SQER and de minimis emission values","US OSHA Carcinogens Listing","International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US - California Proposition 65 - Carcinogens","US National Toxicology Program (NTP) 12th Report Part A Known to be Human Carcinogens","US - Minnesota Permissible Exposure Limits (PELs)","US ACGIH Threshold Limit Values (TLV)","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs)","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants","US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Carcinogens","US OSHA Permissible Exposure Levels (PELs) - Table Z1"
lead(7439-92-1) is found on the following regulatory lists	"US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - California Proposition 65 - Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity","US - Idaho - Limits for Air Contaminants","US - Hawaii Air Contaminant Limits","US - California Permissible Exposure Limits for Chemical Contaminants","US - California Proposition 65 - No Significant Risk Levels (NSRLs) for Carcinogens","US ACGIH Threshold Limit Values (TLV) - Carcinogens","US - California Proposition 65 - Reproductive Toxicity","US EPA Carcinogens Listing","US National Toxicology Program (NTP) 12th Report Part B. Reasonably Anticipated to be a Human Carcinogen","US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants","US - California - Proposition 65 - Priority List for the Development of MADLs for Chemicals Causing Reproductive Toxicity","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","US - Washington Toxic air pollutants and their ASIL, SQER and de minimis emission values","US NIOSH Recommended Exposure Limits (RELs)","US - Alaska Limits for Air Contaminants","US - Washington Permissible exposure limits of air contaminants","US - California Proposition 65 - Carcinogens","US National Toxicology Program (NTP) 12th Report Part A Known to be Human Carcinogens","US - Minnesota Permissible Exposure Limits (PELs)","US ACGIH Threshold Limit Values (TLV)","US - Idaho - Acceptable Maximum Peak Concentrations","US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Carcinogens","US OSHA Permissible Exposure Levels (PELs) - Table Z1"

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.

end of SDS