

NV Hot Stripper

N.V. Chemicals (Aust) P/L

Chemwatch Hazard Alert Code: 4

Chemwatch: 5498-22

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Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

L.GHS.AUS.EN.E

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

Product Identifier

Product name	NV Hot Stripper
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	CORROSIVE LIQUID, TOXIC, N.O.S. (contains ammonium fluoride and hydrochloric acid)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	N.V. Chemicals (Aust) P/L	Re-Stox Business Supplies & Ranges Coffee
Address	24 Lisa Place Coolaroo VIC 3048 Australia	14 Melba Avenue Victoria 3140 Australia
Telephone	+61 3 9351 1100	+61 39738 7730
Fax	+61 3 9351 1077	Not Available
Website	http://www.nvchemicals.com.au/	Not Available
Email	info@nvchemicals.com.au	gwilliams@restox.com.au

Emergency telephone number

Association / Organisation	N.V.Chemicals(Aust) P/L	Re-Stox Business Supplies & Ranges Coffee
Emergency telephone numbers	0411 387 097	+61 409 866 355
Other emergency telephone numbers	Not Available	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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


Chemwatch Hazard Ratings

	Min	Max	
Flammability	0		
Toxicity	3		
Body Contact	4		
Reactivity	1		
Chronic	2		

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

Poisons Schedule	Not Applicable
Classification ^[1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1A, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 3, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
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Signal word	Danger
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Hazard statement(s)

H302	Harmful if swallowed.
H314	Causes severe skin burns and eye damage.
H331	Toxic if inhaled.
H335	May cause respiratory irritation.
H412	Harmful to aquatic life with long lasting effects.
AUH019	May form explosive peroxides.

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting. If more than 15 mins from Doctor, INDUCE VOMITING (if conscious).
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P363	Wash contaminated clothing before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
7647-01-0	>20	<u>hydrochloric acid</u>
6540-99-4	>10	<u>lauryl alcohol, ethoxylated</u>
34590-94-8	>10	<u>dipropylene glycol monomethyl ether</u>
12125-01-8	>10	<u>ammonium fluoride</u>
7732-18-5	balance	<u>water</u>

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

SECTION 4 First aid measures**Description of first aid measures**

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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	<p>If there is evidence of severe skin irritation or skin burns:</p> <ul style="list-style-type: none"> ▶ Avoid further contact. Immediately remove contaminated clothing, including footwear. ▶ Flush skin under running water for 15 minutes. ▶ Avoiding contamination of the hands, massage calcium gluconate gel into affected areas, pay particular attention to creases in skin.

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	<ul style="list-style-type: none"> ▶ Contact the Poisons Information Centre. ▶ Continue gel application for at least 15 minutes after burning sensation ceases. ▶ If pain recurs, repeat application of calcium gluconate gel or apply every 20 minutes. ▶ If no gel is available, continue washing for at least 15 minutes, using soap if available. If patient is conscious, give six calcium gluconate or calcium carbonate tablets in water by mouth. ▶ Transport to hospital, or doctor, urgently.
Inhalation	<ul style="list-style-type: none"> ▶ 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. <p>For massive exposures:</p> <ul style="list-style-type: none"> ▶ If dusts, vapours, aerosols, 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. ▶ If victim is conscious, give six calcium gluconate or calcium carbonate tablets in water by mouth. ▶ Transport to hospital, or doctor, urgently.
Ingestion	<ul style="list-style-type: none"> ▶ 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 strong acids:

- ▶ Airway problems may arise from laryngeal edema and inhalation exposure. Treat with 100% oxygen initially.
- ▶ Respiratory distress may require cricothyroidotomy if endotracheal intubation is contraindicated by excessive swelling
- ▶ Intravenous lines should be established immediately in all cases where there is evidence of circulatory compromise.
- ▶ Strong acids produce a coagulation necrosis characterised by formation of a coagulum (eschar) as a result of the desiccating action of the acid on proteins in specific tissues.

INGESTION:

- ▶ Immediate dilution (milk or water) within 30 minutes post ingestion is recommended.
- ▶ **DO NOT attempt to neutralise the acid since exothermic reaction may extend the corrosive injury.**
- ▶ Be careful to avoid further vomit since re-exposure of the mucosa to the acid is harmful. Limit fluids to one or two glasses in an adult.
- ▶ Charcoal has no place in acid management.
- ▶ Some authors suggest the use of lavage within 1 hour of ingestion.

SKIN:

- ▶ Skin lesions require copious saline irrigation. Treat chemical burns as thermal burns with non-adherent gauze and wrapping.
- ▶ Deep second-degree burns may benefit from topical silver sulfadiazine.

EYE:

- ▶ Eye injuries require retraction of the eyelids to ensure thorough irrigation of the conjunctival cul-de-sacs. Irrigation should last at least 20-30 minutes. **DO NOT use neutralising agents or any other additives.** Several litres of saline are required.
- ▶ Cycloplegic drops, (1% cyclopentolate for short-term use or 5% homatropine for longer term use) antibiotic drops, vasoconstrictive agents or artificial tears may be indicated dependent on the severity of the injury.
- ▶ Steroid eye drops should only be administered with the approval of a consulting ophthalmologist).

[Ellenhorn and Barceloux: Medical Toxicology]

For acute or short term repeated exposures to ammonia and its solutions:

- ▶ Mild to moderate inhalation exposures produce headache, cough, bronchospasm, nausea, vomiting, pharyngeal and retrosternal pain and conjunctivitis. Severe inhalation produces laryngospasm, signs of upper airway obstruction (stridor, hoarseness, difficulty in speaking) and, in excessively, high doses, pulmonary oedema.
- ▶ Warm humidified air may soothe bronchial irritation.
- ▶ Test all patients with conjunctival irritation for corneal abrasion (fluorescein stain, slit lamp exam)
- ▶ Dyspneic patients should receive a chest X-ray and arterial blood gases to detect pulmonary oedema.

SECTION 5 Firefighting measures**Extinguishing media**

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered to be a significant fire risk. ▶ Acids may react with metals to produce hydrogen, a highly flammable and explosive gas.

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	<ul style="list-style-type: none"> ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ May emit corrosive, poisonous fumes. May emit acrid smoke. <p>carbon dioxide (CO₂) hydrogen chloride phosgene hydrogen fluoride nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material.</p>
HAZCHEM	2X

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ 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.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin <p>The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe</p> <ul style="list-style-type: none"> ▶ DO NOT concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential. ▶ Any static discharge is also a source of hazard. ▶ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina. ▶ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage. ▶ Add inhibitor to any distillate as required. ▶ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ WARNING: To avoid violent reaction, ALWAYS add material to water and NEVER water to material. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<p>20L/5L.</p> <ul style="list-style-type: none"> ▶ DO NOT use aluminium or galvanised containers ▶ Check regularly for spills and leaks
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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.

- ▶ Polyethylene or polypropylene container.
- ▶ Packing as recommended by manufacturer.
- ▶ Check all containers are clearly labelled and free from leaks.

- ▶ Inorganic acids are generally soluble in water with the release of hydrogen ions. The resulting solutions have pH's of less than 7.0.
- ▶ Inorganic acids neutralise chemical bases (for example: amines and inorganic hydroxides) to form salts - neutralisation can generate dangerously large amounts of heat in small spaces.
- ▶ The dissolution of inorganic acids in water or the dilution of their concentrated solutions with additional water may generate significant heat.
- ▶ The addition of water to inorganic acids often generates sufficient heat in the small region of mixing to cause some of the water to boil explosively. The resulting "bumping" can spatter the acid.
- ▶ Inorganic acids react with active metals, including such structural metals as aluminum and iron, to release hydrogen, a flammable gas.
- ▶ Inorganic acids can initiate the polymerisation of certain classes of organic compounds.
- ▶ Inorganic acids react with cyanide compounds to release gaseous hydrogen cyanide.
- ▶ Inorganic acids generate flammable and/or toxic gases in contact with dithiocarbamates, isocyanates, mercaptans, nitrides, nitriles, sulfides, and strong reducing agents. Additional gas-generating reactions occur with sulfites, nitrites, thiosulfates (to give H₂S and SO₃), dithionites (SO₂), and even carbonates.
- ▶ Acids often catalyse (increase the rate of) chemical reactions.

Hydrogen chloride:

- ▶ reacts strongly with strong oxidisers (releasing chlorine gas), acetic anhydride, caesium cyanotridecahydrodecaborate(2-), ethylidene difluoride, hexalithium disilicide, metal acetylide, sodium, silicon dioxide, tetraselenium tetranitride, and many organic materials
- ▶ is incompatible with alkaline materials, acetic anhydride, acetylides, aliphatic amines, alkanolamines, alkylene oxides, aluminium, aluminium-titanium alloys, aromatic amines, amines, amides, 2-aminoethanol, ammonia, ammonium hydroxide, borides, calcium phosphide, carbides, carbonates, cyanides, chlorosulfonic acid, ethylenediamine, ethyleneimine, epichlorohydrin, formaldehyde, isocyanates, metals, metal oxides, metal hydroxides, metal acetylides, metal carbides, oleum, organic anhydrides, potassium permanganate, perchloric acid, phosphides, 3-propiolactone, silicides, sulfides, sulfites, sulfuric acid, uranium phosphide, vinyl acetate, vinylidene fluoride
- ▶ attacks most metals forming flammable hydrogen gas, and some plastics, rubbers and coatings
- ▶ reacts with zinc, brass, galvanised iron, aluminium, copper and copper alloys

Storage incompatibility

- ▶ Contact with acids produces toxic fumes
- ▶ Reacts vigorously with alkalis
- ▶ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air.

Dipropylene glycol monomethyl ether:

- ▶ may form unstable peroxides on contact with air
- ▶ reacts violently with strong oxidisers, permanganates, peroxides, ammonium persulfate, bromine dioxide, sulfuric acid, nitric acid, perchloric acid and other strong acids
- ▶ is incompatible with acid halides, aliphatic amines, alkalis, boranes, isocyanates
- ▶ attacks some plastics, rubber and coatings
- ▶ Glycol ethers may form peroxides under certain conditions; the potential for peroxide formation is enhanced when these substances are used in processes such as distillation where they are concentrated or even evaporated to near-dryness or dryness; storage under a nitrogen atmosphere is recommended to minimise the possible formation of highly reactive peroxides
- ▶ Nitrogen blanketing is recommended if transported in containers at temperatures within 15 deg C of the flash-point and at or above the flash-point - large containers may first need to be purged and inerted with nitrogen prior to loading
- ▶ In the presence of strong bases or the salts of strong bases, at elevated temperatures, the potential exists for runaway reactions.
- ▶ Contact with aluminium should be avoided; release of hydrogen gas may result- glycol ethers will corrode scratched aluminium surfaces.
- ▶ May discolour in mild steel/ copper; lined containers, glass or stainless steel is preferred
- ▶ Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water. Investigation of the hazards associated with use of 2-butoxyethanol for alloy electropolishing showed that mixtures with 50-95% of acid at 20 deg C, or 40-90% at 75 C, were explosive and initiatable by sparks. Sparking caused mixtures with 40-50% of acid to become explosive, but 30% solutions appeared safe under static conditions of temperature and concentration.
- ▶ Segregate from alkalis, oxidising agents and chemicals readily decomposed by acids, i.e. cyanides, sulfides, carbonates.
- ▶ Avoid reaction with oxidising agents, bases and strong reducing agents.
- ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	hydrochloric acid	Hydrogen chloride	Not Available	Not Available	5 ppm / 7.5 mg/m ³	Not Available
Australia Exposure Standards	dipropylene glycol monomethyl ether	(2-Methoxymethylethoxy) propanol	50 ppm / 308 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	ammonium fluoride	Fluorides (as F)	2.5 mg/m ³	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
hydrochloric acid	Not Available	Not Available	Not Available
hydrochloric acid	1.8 ppm	22 ppm	100 ppm
lauryl alcohol, ethoxylated	2.9 mg/m ³	31 mg/m ³	200 mg/m ³

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Ingredient	TEEL-1	TEEL-2	TEEL-3
dipropylene glycol monomethyl ether	150 ppm	1700* ppm	9900** ppm
ammonium fluoride	15 mg/m3	160 mg/m3	980 mg/m3
Ingredient	Original IDLH	Revised IDLH	
hydrochloric acid	50 ppm	Not Available	
lauryl alcohol, ethoxylated	Not Available	Not Available	
dipropylene glycol monomethyl ether	600 ppm	Not Available	
ammonium fluoride	Not Available	Not Available	
water	Not Available	Not Available	

Occupational Exposure Banding

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

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>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:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>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.</p> <table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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.</p>	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.)	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4: Large hood or large air mass in motion	4: Small hood-local control only																				
Individual protection measures, such as personal protective equipment																					
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ 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]. 																				
Skin protection	See Hand protection below																				
Hands/feet protection	<ul style="list-style-type: none"> ▶ Elbow length PVC gloves ▶ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. 																				

	<ul style="list-style-type: none"> ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>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.</p> <p>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.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · 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. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>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.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

NV Hot Stripper

Material	CPI
NEOPRENE	A
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
VITON	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

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

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

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

^ - Full-face

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

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

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Ansell Glove Selection**Glove** — In order of recommendation

AlphaTec® Solvex® 37-185

AlphaTec® 38-612

AlphaTec® 58-008

AlphaTec® 58-530B

AlphaTec® 58-530W

AlphaTec® 58-735

AlphaTec® 79-700

AlphaTec® Solvex® 37-675

DermaShield™ 73-711

MICROFLEX® 63-864

*The suggested gloves for use should be confirmed with the glove supplier.***SECTION 9 Physical and chemical properties****Information on basic physical and chemical properties**

Appearance	Clear liquid.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Not Available	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	197.96

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Contact with alkaline material liberates heat ▶ 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	<p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation.</p> <p>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.</p> <p>Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Acidic corrosives produce respiratory tract irritation with coughing, choking and mucous membrane damage. Symptoms of exposure may include dizziness, headache, nausea and weakness. In more severe exposures, pulmonary oedema may be evident either immediately or after a latent period of 5-72 hours. Symptoms of pulmonary oedema include a tightness in the chest, dyspnoea, frothy sputum and cyanosis. Examination may reveal hypotension, a weak and rapid pulse and moist rates. Death, due to anoxia, may occur several hours after onset of the pulmonary oedema.</p>
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Continued...

	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Hydrogen chloride (HCl) vapour or fumes present a hazard from a single acute exposure. Exposures of 1300 to 2000 ppm have been lethal to humans in a few minutes.</p> <p>Inhalation of HCl may cause choking, coughing, burning sensation and may cause ulceration of the nose, throat and larynx. Fluid on the lungs followed by generalised lung damage may follow.</p> <p>Breathing of HCl vapour may aggravate asthma and inflammatory or fibrotic pulmonary disease.</p> <p>High concentrations cause necrosis of the tracheal and bronchial epithelium, pulmonary oedema, atelectasis and emphysema and damage to the pulmonary blood vessels and liver.</p> <p>In fog-laden atmospheres rats exposed to dipropylene glycol monomethyl ether DPME, for 7 hours, exhibited a mild narcosis from which they rapidly recovered. Controlled human exposures to vapour produced CNS impairment at 1000 ppm in one subject</p> <p>The highly irritant properties of ammonia vapour result as the gas dissolves in mucous fluids and forms irritant, even corrosive solutions. Inhalation of the ammonia fumes causes coughing, vomiting, reddening of lips, mouth, nose, throat and conjunctiva while higher concentrations can cause temporary blindness, restlessness, tightness in the chest, pulmonary oedema (lung damage), weak pulse and cyanosis.</p> <p>Inhalation of high concentrations of vapour may cause breathing difficulty, tightness in chest, pulmonary oedema and lung damage. Brief exposure to high concentrations > 5000 ppm may cause death due to asphyxiation (suffocation) or fluid in the lungs.</p> <p>Prolonged or regular minor exposure to the vapour may cause persistent irritation of the eyes, nose and upper respiratory tract. Massive ammonia exposures may produce chronic airway hyperactivity and asthma with associated pulmonary function changes. The average nasal retention of ammonia by human subjects was found to be 83%.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severely toxic effects; these may be fatal.</p>
<p style="text-align: center;">Ingestion</p>	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.</p> <p>Ingestion of acidic corrosives may produce circumoral burns with a distinct discolouration of the mucous membranes of the mouth, throat and oesophagus. Immediate pain and difficulties in swallowing and speaking may also be evident. Oedema of the epiglottis may produce respiratory distress and possibly, asphyxia. Nausea, vomiting, diarrhoea and a pronounced thirst may occur. More severe exposures may produce a vomitus containing fresh or dark blood and large shreds of mucosa. Shock, with marked hypotension, weak and rapid pulse, shallow respiration and clammy skin may be symptomatic of the exposure. Circulatory collapse may, if left untreated, result in renal failure. Severe cases may show gastric and oesophageal perforation with peritonitis, fever and abdominal rigidity. Stricture of the oesophageal, gastric and pyloric sphincter may occur as within several weeks or may be delayed for years. Death may be rapid and often results from asphyxia, circulatory collapse or aspiration of even minute amounts. Delayed deaths may be due to peritonitis, severe nephritis or pneumonia. Coma and convulsions may be terminal.</p> <p>Dipropylene monomethyl ether (DPME) produces marked central nervous system depression in rats. Lethal doses produced respiratory failure within 48 hours.</p>
<p style="text-align: center;">Skin Contact</p>	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.</p> <p>Skin contact with acidic corrosives may result in pain and burns; these may be deep with distinct edges and may heal slowly with the formation of scar tissue.</p> <p>The skin is readily penetrated by the fluoride ion causing liquefaction necrosis of the soft tissues and decalcification and corrosion of bone. Healing is delayed and necrotic changes may continue to occur and spread beneath a layer of tough coagulated skin.</p> <p>Percutaneous absorption of pure liquefied hydrogen fluoride gas produced severe hypocalcaemia, multiple attacks of ventricular fibrillation, and death 9.5 hours after exposure. Skin contact with hydrogen fluoride or solutions containing more than 30 percent hydrogen fluoride produces immediate pain; reactions to more dilute solutions may be delayed for many hours. The accompanying pain is excruciating and persistent due to delayed healing.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p> <p>Continuous contact with DPME of the skin of numerous rabbits for 90 days caused only slight scaliness. Patch tests on human volunteers produced no evidence of primary irritation or sensitisation. Sufficient absorption did occur in rabbits to produce narcosis and high doses proved lethal. Pathology revealed gastric distension, occasional gastric irritation and granular and hydropic changes to kidneys</p>
<p style="text-align: center;">Eye</p>	<p>Direct eye contact with acid corrosives may produce pain, lachrymation, photophobia and burns. Mild burns of the epithelia generally recover rapidly and completely. Severe burns produce long-lasting and possible irreversible damage. The appearance of the burn may not be apparent for several weeks after the initial contact. The cornea may ultimately become deeply vascularised and opaque resulting in blindness.</p> <p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p> <p>Irritation of the eyes may produce a heavy secretion of tears (lachrymation).</p> <p>When one drop of undiluted dipropylene glycol monomethyl ether (DPME) was placed in a rabbits eyes on each of five consecutive days, a mild transitory irritation of the conjunctival membranes occurred. Fluorescein staining revealed no corneal damage. Direct contact of the substance can produce painful irritation (blepharoconjunctivitis, slight keratitis, and an increase in intra-ocular pressure) which, is however rapidly reversible. Persistent eye lesions do not develop</p>
<p style="text-align: center;">Chronic</p>	<p>Repeated or prolonged exposure to acids may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.</p> <p>The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). Asthmatics appear to be at particular risk for pulmonary effects.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for</p>

making a satisfactory assessment.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats Which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure.

Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer . beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

Chronic minor exposure to hydrogen chloride (HCl) vapour or fume may cause discolouration or erosion of the teeth, bleeding of the nose and gums; and ulceration of the nasal mucous membranes.

Repeated exposures of animals to concentrations of about 34 ppm HCl produced no immediate toxic effects.

Workers exposed to hydrochloric acid suffered from gastritis and a number of cases of chronic bronchitis have also been reported.

Repeated or prolonged exposure to dilute solutions of HCl may cause dermatitis.

Rats, rabbits, guinea pigs and monkeys exposed to DPME, 7 hr/day, 5 days a week for periods of 6-8 months to saturated atmospheres (300 ppm), exhibited little effect. Narcotic effects were produced in rats. This concentration of vapour is objectionable to human beings.

Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.

NV Hot Stripper	TOXICITY	IRRITATION
	Not Available	Not Available
hydrochloric acid	TOXICITY	IRRITATION
	dermal (mouse) LD50: 1449 mg/kg ^[2]	Eye (rabbit): 5mg/30s - mild
	Oral (Rat) LD50: 900 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin: adverse effect observed (corrosive) ^[1]
lauryl alcohol, ethoxylated	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye (rabbit): 0.75 mg/24h SEVERE
	Oral (Rat) LD50: 1000 mg/kg ^[2]	Eye (rabbit): 100 mg
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg/24h mild
		Skin (rabbit): 75 mg/24h mild
dipropylene glycol monomethyl ether	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 9500 mg/kg ^[2]	Eye (human): 8 mg - mild
	Oral (Rat) LD50: 5135 mg/kg ^[2]	Eye (rabbit): 500 mg/24hr - mild
		Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 238 mg - mild
ammonium fluoride	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Inhalation (Rat) LC50: 1 mg/4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
water	TOXICITY	IRRITATION
	Oral (Rat) LD50: >90000 mg/kg ^[2]	Not Available

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

HYDROCHLORIC ACID

for acid mists, aerosols, vapours

Data from assays for genotoxic activity in vitro suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events in vivo in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH in vivo differ from exposures in vitro in that, in vivo, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than in vitro.

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.

NV Hot Stripper

LAURYL ALCOHOL, ETHOXYLATED

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.

Ann-Therese Karlberg et al; Chem. Res. Toxicol. 2008, 21, 53-69

Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations.

Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used.

Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology

<http://doi.org/10.5487/TR.2015.31.2.105>

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing.

Overall, alcohol alkoxyates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length $C \geq 6$ may also contain shorter alkyl chains $C < 6$. It is not practical to quantify the proportion of shorter $C < 6$ chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD₅₀ values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an

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adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm²/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm²/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME.

Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or

haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats. In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

Mutagenicity: Mutagenicity studies have been conducted for several category members. All *in vitro* and *in vivo* studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

DIPROPYLENE GLYCOL MONOMETHYL ETHER

for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

Continued...

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This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating. None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

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.

HYDROCHLORIC ACID & LAURYL ALCOHOL, ETHOXYLATED & DIPROPYLENE GLYCOL MONOMETHYL ETHER

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

HYDROCHLORIC ACID & WATER

No significant acute toxicological data identified in literature search.

HYDROCHLORIC ACID & DIPROPYLENE GLYCOL MONOMETHYL ETHER

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

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

SECTION 12 Ecological information

Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
NV Hot Stripper	Not Available	Not Available	Not Available	Not Available	Not Available
hydrochloric acid	EC50(ECx)	9.33h	Fish	0.51mg/L	4
	LC50	96h	Fish	334.734mg/L	4

	Endpoint	Test Duration (hr)	Species	Value	Source
	lauryl alcohol, ethoxylated	NOEC(ECx)	672h	Fish	0.139mg/l
EC50		72h	Algae or other aquatic plants	1.225mg/l	2
EC50		48h	Crustacea	1.2mg/L	5
LC50		96h	Fish	1.5mg/L	4
dipropylene glycol monomethyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>969mg/l	2
	EC50	96h	Algae or other aquatic plants	>969mg/l	2
	LC50	96h	Fish	>1000mg/l	2
	NOEC(ECx)	528h	Crustacea	>=0.5mg/l	2
ammonium fluoride	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	364mg/l	Not Available
	EC50	48h	Crustacea	2.94mg/l	2
	NOEC(ECx)	672h	Fish	0.17mg/l	2
	EC50	96h	Algae or other aquatic plants	43mg/l	2
water	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

Very toxic to aquatic organisms.

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

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

For Propylene Glycol Ethers: log Kow's range from 0.309 for TPM to 1.523 for DPnB. Calculated BCFs range from 1.47 for DPnB to 3.16 for DPMA and TPM, indicating low bioaccumulation. Henry's Law Constants are low for all category members, ranging from 5.7×10^{-9} atm-m³/mole for TPM to 2.7×10^{-9} atm-m³/mole for PnB.

Environmental Fate: Most are liquids at room temperature and all are water-soluble.

Atmospheric Fate: In air, the half-life due to direct reactions with photochemically generated hydroxyl radicals, range from 2.0 hours for TPM to 4.6 hours for PnB.

Aquatic/Terrestrial Fate: Most propylene glycol ethers are likely to partition roughly equally into the soil and water compartments in the environment with small to negligible amounts remaining in other environmental compartments (air, sediment, and aquatic biota). In water, most members of this family are "readily biodegradable" under aerobic conditions. In soil, biodegradation is rapid for PM and PMA.

Ecotoxicity: Propylene glycol ethers are unlikely to persist in the environment. Acute aquatic toxicity testing indicates low toxicity for both ethers and acetates.

Ecotoxicity:

The tolerance of water organisms towards pH margin and variation is diverse. Recommended pH values for test species listed in OECD guidelines are between 6.0 and almost 9. Acute testing with fish showed 96h-LC50 at about pH 3.5

Although small amounts of fluorides are conceded to have beneficial effects, two forms of chronic toxic effect, dental fluorosis and skeletal fluorosis may be caused by excessive intake over long periods. Fluorides are absorbed by humans following inhalation of workplace and ambient air that has been contaminated, ingestion of drinking water and foods and dermal contact.

Fluoride accumulates, food-dependently in skeletal tissues of both aquatic and terrestrial vertebrates and invertebrates. Bioaccumulation occurs in marine organisms and, to a lesser extent, fresh water organisms. Reported BCF-values for marine organisms range up to approximately 150 and 60 for fish and crustacea, respectively. The most important exposure route for plants is uptake from the atmosphere. Concentrations in plants in the vicinity of a HF production plant range up to approximately 200 mg/kg, with mean levels between 20 and 50 mg/kg dry weight. Generally, lowest fluoride levels are found in herbivores and (somewhat) higher levels in predators.

Fluorides have been shown to accumulate in animals that consume fluoride-containing foliage. However, accumulation is primarily in skeletal tissue and therefore, it is unlikely that fluoride will biomagnify up the food chain.

Both hydrogen fluoride and particulate fluorides will be transported in the atmosphere and deposited on land or water by wet and dry deposition. Non-volatile inorganic fluoride particulates are removed from the atmosphere via condensation or nucleation processes. Fluorides adsorbed on particulate matter in the atmosphere are generally stable and are not readily hydrolysed, although they may be degraded by radiation if they persist in the atmosphere. Fluorine and the silicon fluorides (fluosilicates, silicofluorides) are hydrolysed in the atmosphere to form hydrogen fluoride. Hydrogen fluoride may combine with water vapour to produce an aerosol or fog of aqueous hydrofluoric acid. Based upon available data, inorganic fluoride compounds, with the exception of sulfur hexafluoride, are not expected to remain in the troposphere for long periods or to migrate to the stratosphere. Estimates of the residence time of sulfur hexafluoride in the atmosphere range from 500 to several thousand years. Fluoride in aerosols can be transported over large distances by wind or as a result of atmospheric turbulence. The distance travelled is determined by the deposition velocity of both the gaseous hydrogen fluoride and the fluorides in particulate form. Atmospheric fluorides may be transported to soils and surface waters through both wet and dry deposition processes.

Fluorides undergo transformations in soil and water, forming complexes and binding strongly to soil and sediment.

In water, the transport and transformation of inorganic fluorides are influenced by pH, water hardness and the presence of ion-exchange materials such as clays. In natural water, fluoride forms strong complexes with aluminum in water, and fluorine chemistry in water is largely regulated by aluminum concentration and pH. Below pH 5, fluoride is almost entirely complexed with aluminum and consequently, the concentration of free F⁻ is low. As the pH increases, Al-OH complexes dominate over Al-F complexes and the free F⁻ levels increase. Fluoride forms stable complexes with calcium and magnesium, which are present in sea water. Calcium carbonate precipitation dominates the removal of dissolved fluoride from sea water. The residence time for fluoride in ocean sediment is calculated to be 2-3 million years. Fluorosilicic acid and hydrofluoric acid in high aquatic concentrations such as may be found in industrial waste ponds may volatilise, releasing silicon tetrafluoride and hydrogen fluoride into the atmosphere.

Solubilisation of inorganic fluorides from minerals may also be enhanced by the presence of ion-exchange materials (e.g., bentonite clays and humic acid). Once dissolved, inorganic fluorides remain in solution under conditions of low pH and hardness and in the presence of ion-exchange material. Soluble inorganic fluorides may also form aerosols at the air/water interface or vaporise into the atmosphere whereas undissolved species generally undergo sedimentation.

Factors that influence the mobility of inorganic fluorides in soil are pH and the formation of aluminium and calcium complexes. In more acidic soils, concentrations of inorganic fluoride were considerably higher in the deeper horizons. The low affinity of fluorides for organic material results in leaching from the more acidic surface horizon and increased retention by clay minerals and silts in the more alkaline, deeper horizons. The maximum adsorption of fluoride to soil was reported to occur at pH 5.5. In acidic soils with pH below 6, most of the fluoride is in complexes with either aluminium or iron. Fluoride in alkaline soils at pH 6.5 and above is almost completely fixed in soils as calcium fluoride, if sufficient calcium carbonate is available. Fluoride is extremely immobile in soil, as determined by lysimeter experiments.

Populations living in areas with high fluoride levels in groundwater may be exposed to higher levels of fluorides in their drinking water or in beverages prepared with the water. Among these populations, outdoor laborers, people living in hot climates, and people with polydipsia will generally have the greatest daily intake of fluorides because they consume greater amounts of water.

Foods characteristically high in fluoride content are certain types of fish and seafood (1.9-28.5 mg/kg), especially those types in which the bones are consumed, bone products such as bone meal and gelatin, and tea, which contains approximately 0.52 mg fluoride/cup

Fluoride is mainly absorbed by the body in the form of hydrogen fluoride, which has a pKa of 3.45. That is, when ionic fluoride enters the acidic environment of the stomach lumen, it is largely converted into hydrogen fluoride. Most of the fluoride that is not absorbed from the stomach will be rapidly absorbed from the small intestine.

For Glycol Ethers:

Environmental Fate: Several glycol ethers have been shown to biodegrade however; biodegradation slows as molecular weight increases. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes.

Atmospheric Fate: Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photo-degradation (atmospheric half lives = 2.4-2.5 hr).

Aquatic Fate: In water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity: Tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers. Glycols exert a high oxygen demand for decomposition and once released to the environment death of aquatic organisms occurs if dissolved oxygen is depleted.

For Ammonia:

Atmospheric Fate: Ammonia reacts rapidly with available acids (mainly sulfuric, nitric, and sometimes hydrochloric acid) to form the corresponding salts. Ammonia is persistent in the air.

Aquatic Fate: Biodegrades rapidly to nitrate, producing a high oxygen demand. Non-persistent in water (half-life 2 days).

Ecotoxicity: Moderately toxic to fish under normal temperature and pH conditions and harmful to aquatic life at low concentrations. Does not concentrate in food chain.

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
hydrochloric acid	LOW	LOW
lauryl alcohol, ethoxylated	LOW	LOW
dipropylene glycol monomethyl ether	HIGH	HIGH
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
hydrochloric acid	LOW (LogKOW = 0.5392)
lauryl alcohol, ethoxylated	LOW (LogKOW = 3.6722)
dipropylene glycol monomethyl ether	LOW (BCF = 100)

Mobility in soil

Ingredient	Mobility
hydrochloric acid	LOW (Log KOC = 14.3)
lauryl alcohol, ethoxylated	LOW (Log KOC = 10)
dipropylene glycol monomethyl ether	LOW (Log KOC = 10)

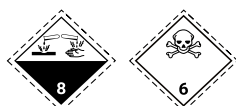
SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>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.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>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.</p> <ul style="list-style-type: none"> ▶ 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 soda-ash or soda-lime followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▶ Decontaminate empty containers with 5% aqueous sodium hydroxide or soda ash, followed by water. Observe all label safeguards until containers are cleaned and destroyed.
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SECTION 14 Transport information

Labels Required



Marine Pollutant	NO
HAZCHEM	2X

Land transport (ADG)

14.1. UN number or ID number	2922	
14.2. UN proper shipping name	CORROSIVE LIQUID, TOXIC, N.O.S. (contains ammonium fluoride and hydrochloric acid)	
14.3. Transport hazard class(es)	Class	8
	Subsidiary Hazard	6.1
14.4. Packing group	II	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	274
	Limited quantity	1 L

Air transport (ICAO-IATA / DGR)

14.1. UN number	2922	
14.2. UN proper shipping name	Corrosive liquid, toxic, n.o.s. * (contains ammonium fluoride and hydrochloric acid)	
14.3. Transport hazard class(es)	ICAO/IATA Class	8
	ICAO / IATA Subsidiary Hazard	6.1
	ERG Code	8P
14.4. Packing group	II	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	A3 A4 A803
	Cargo Only Packing Instructions	855
	Cargo Only Maximum Qty / Pack	30 L
	Passenger and Cargo Packing Instructions	851
	Passenger and Cargo Maximum Qty / Pack	1 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y840
	Passenger and Cargo Limited Maximum Qty / Pack	0.5 L

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	2922	
14.2. UN proper shipping name	CORROSIVE LIQUID, TOXIC, N.O.S. (contains ammonium fluoride and hydrochloric acid)	
14.3. Transport hazard class(es)	IMDG Class	8
	IMDG Subsidiary Hazard	6.1
14.4. Packing group	II	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	F-A, S-B
	Special provisions	274
	Limited Quantities	1 L

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

Not Applicable

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

Product name	Group
hydrochloric acid	Not Available
lauryl alcohol, ethoxylated	Not Available
dipropylene glycol monomethyl ether	Not Available
ammonium fluoride	Not Available
water	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
hydrochloric acid	Not Available
lauryl alcohol, ethoxylated	Not Available

NV Hot Stripper

Product name	Ship Type
dipropylene glycol monomethyl ether	Not Available
ammonium fluoride	Not Available
water	Not Available

SECTION 15 Regulatory information

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

hydrochloric acid is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
 Australian Inventory of Industrial Chemicals (AIIC)
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

lauryl alcohol, ethoxylated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Industrial Chemicals Introduction Scheme Comparable Chemicals Table
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
 Australian Inventory of Industrial Chemicals (AIIC)

dipropylene glycol monomethyl ether is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

ammonium fluoride is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australian Inventory of Industrial Chemicals (AIIC)
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (hydrochloric acid; lauryl alcohol, ethoxylated; dipropylene glycol monomethyl ether; ammonium fluoride; water)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	12/10/2021
Initial Date	12/10/2021

Other information

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

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

Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer

Continued...

- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit,
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration

- ▶ AIIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European INventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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