

## General Purpose Concrete Cleaner



## General Purpose Concrete Cleaner

On-Crete Australia Pty Ltd

Version No: 1.8

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 22/05/2020

Print Date: 22/05/2020

L.GHS.AUS.EN

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

## Product Identifier

Product name	General Purpose Concrete Cleaner
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	General purpose cleaner concentrate
--------------------------	-------------------------------------

## Details of the supplier of the safety data sheet

Registered company name	On-Crete Australia Pty Ltd	Oncrete New Zealand Pty Ltd
Address	4/489 Scottsdale Drive Queensland 4227 Australia	15A Vega Place, Rosedale Auckland 0632 New Zealand
Telephone	+617 5593 6884	+649 930 8829
Fax	Not Available	Not Available
Website	www.on-crete.com.au	www.oncrete.co.nz
Email	sales@on-crete.com.au	sales@oncrete.co.nz

## Emergency telephone number

Association / Organisation	On-Crete Australia Pty Ltd	Oncrete New Zealand Pty Ltd
Emergency telephone numbers	1300 292 504	0800 120 854
Other emergency telephone numbers	+613 6121 9073	+649 553 8356

## SECTION 2 HAZARDS IDENTIFICATION

## Classification of the substance or mixture

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

## CHEMWATCH HAZARD RATINGS


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

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

Poisons Schedule	S6
Classification [1]	Skin Corrosion/Irritation Category 1B, Acute Aquatic Hazard Category 3, Serious Eye Damage Category 1, Acute Toxicity (Oral) Category 4
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

## General Purpose Concrete Cleaner

## Label elements

Hazard pictogram(s)	
---------------------	---

SIGNAL WORD	<b>DANGER</b>
-------------	---------------

## Hazard statement(s)

H314	Causes severe skin burns and eye damage.
H402	Harmful to aquatic life.
H302	Harmful if swallowed.

## Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/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.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER or doctor/physician.
P321	Specific treatment (see advice on this label).
P363	Wash contaminated clothing before reuse.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

## Precautionary statement(s) Storage

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.
------	--

## SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

## Substances

See section below for composition of Mixtures

## Mixtures

CAS No	%[weight]	Name
111-76-2*	10-30	<u>2-Butoxyethanol</u>
68585-34-2	<10	<u>sodium lauryl ether sulfate</u>
10213-79-3	<10	<u>sodium metasilicate, pentahydrate</u>
68603-42-9	<1	<u>cocamide diethanolamide.</u>
111-42-2	<1	<u>diethanolamine</u>
7631-99-4	<1	<u>sodium nitrate</u>
55965-84-9	<1	<u>isothiazolinones, mixed</u>
7757-82-6	<1	<u>sodium sulfate</u>
6104-59-2	<1	<u>Brilliant Blue R</u>
5989-27-5	<1	<u>d-limonene</u>
5392-40-5	<1	<u>citral</u>

Continued...

## General Purpose Concrete Cleaner

68917-33-9

&lt;1

lemon oil

## 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"> <li>▶ Immediately hold eyelids apart and flush the eye continuously with running water.</li> <li>▶ 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.</li> <li>▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately flush body and clothes with large amounts of water, using safety shower if available.</li> <li>▶ Quickly remove all contaminated clothing, including footwear.</li> <li>▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
Inhalation	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor, without delay.</li> </ul>
Ingestion	<ul style="list-style-type: none"> <li>▶ For advice, contact a Poisons Information Centre or a doctor at once.</li> <li>▶ Urgent hospital treatment is likely to be needed.</li> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Transport to hospital or doctor without delay.</li> </ul>

## Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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

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

Alkalis continue to cause damage after exposure.

## INGESTION:

- ▶ Milk and water are the preferred diluents

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

- ▶ Neutralising agents should never be given since exothermic heat reaction may compound injury.

\* Catharsis and emesis are absolutely contra-indicated.

\* Activated charcoal does not absorb alkali.

\* Gastric lavage should not be used.

Supportive care involves the following:

- ▶ Withhold oral feedings initially.
- ▶ If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- ▶ Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- ▶ Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

## SKIN AND EYE:

- ▶ Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

## SECTION 5 FIREFIGHTING MEASURES

## Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.

Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.

Continued...

## General Purpose Concrete Cleaner

In such an event consider:

- foam.
- dry chemical powder.
- carbon dioxide.

## Special hazards arising from the substrate or mixture

<b>Fire Incompatibility</b>	None known.
-----------------------------	-------------

## Advice for firefighters

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▸ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▸ Wear breathing apparatus plus protective gloves in the event of a fire.</li> <li>▸ Prevent, by any means available, spillage from entering drains or water courses.</li> <li>▸ Use fire fighting procedures suitable for surrounding area.</li> <li>▸ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▸ Cool fire exposed containers with water spray from a protected location.</li> <li>▸ If safe to do so, remove containers from path of fire.</li> <li>▸ Equipment should be thoroughly decontaminated after use.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▸ The material is not readily combustible under normal conditions.</li> <li>▸ However, it will break down under fire conditions and the organic component may burn.</li> <li>▸ Not considered to be a significant fire risk.</li> <li>▸ Heat may cause expansion or decomposition with violent rupture of containers.</li> <li>▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).</li> <li>▸ May emit acrid smoke.</li> </ul> <p>Decomposes on heating and produces toxic fumes of:</p> <ul style="list-style-type: none"> <li>▸ carbon dioxide (CO<sub>2</sub>)</li> <li>▸ other pyrolysis products typical of burning organic material.</li> </ul> <p>May emit poisonous fumes.</p> <p>May emit corrosive fumes.</p>
<b>HAZCHEM</b>	Not Applicable

## 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"><li>▶ Clean up all spills immediately.</li><li>▶ Avoid breathing vapours and contact with skin and eyes.</li><li>▶ Control personal contact with the substance, by using protective equipment.</li><li>▶ Contain and absorb spill with sand, earth, inert material or vermiculite.</li><li>▶ Wipe up.</li><li>▶ Place in a suitable, labelled container for waste disposal.</li></ul>																																																							
Major Spills	<p>Chemical Class: bases</p> <p>For release onto land: recommended sorbents listed in order of priority.</p> <table><thead><tr><th>SORBENT TYPE</th><th>RANK</th><th>APPLICATION</th><th>COLLECTION</th><th>LIMITATIONS</th></tr></thead><tbody><tr><td colspan="5">LAND SPILL - SMALL</td></tr><tr><td>cross-linked polymer - particulate</td><td>1</td><td>shovel</td><td>shovel</td><td>R,W,SS</td></tr><tr><td>cross-linked polymer - pillow</td><td>1</td><td>throw</td><td>pitchfork</td><td>R, DGC, RT</td></tr><tr><td>sorbent clay - particulate</td><td>2</td><td>shovel</td><td>shovel</td><td>R, I, P</td></tr><tr><td>foamed glass - pillow</td><td>2</td><td>throw</td><td>pitchfork</td><td>R, P, DGC, RT</td></tr><tr><td>expanded minerals - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, I, W, P, DGC</td></tr><tr><td>foamed glass - particulate</td><td>4</td><td>shovel</td><td>shovel</td><td>R, W, P, DGC,</td></tr><tr><td colspan="5">LAND SPILL - MEDIUM</td></tr><tr><td>cross-linked polymer -particulate</td><td>1</td><td>blower</td><td>skiploader</td><td>R,W, SS</td></tr><tr><td>sorbent clay - particulate</td><td>2</td><td>blower</td><td>skiploader</td><td>R, I, P</td></tr></tbody></table>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					cross-linked polymer - particulate	1	shovel	shovel	R,W,SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R, I, P	foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT	expanded minerals - particulate	3	shovel	shovel	R, I, W, P, DGC	foamed glass - particulate	4	shovel	shovel	R, W, P, DGC,	LAND SPILL - MEDIUM					cross-linked polymer -particulate	1	blower	skiploader	R,W, SS	sorbent clay - particulate	2	blower	skiploader	R, I, P
SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS																																																				
LAND SPILL - SMALL																																																								
cross-linked polymer - particulate	1	shovel	shovel	R,W,SS																																																				
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT																																																				
sorbent clay - particulate	2	shovel	shovel	R, I, P																																																				
foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT																																																				
expanded minerals - particulate	3	shovel	shovel	R, I, W, P, DGC																																																				
foamed glass - particulate	4	shovel	shovel	R, W, P, DGC,																																																				
LAND SPILL - MEDIUM																																																								
cross-linked polymer -particulate	1	blower	skiploader	R,W, SS																																																				
sorbent clay - particulate	2	blower	skiploader	R, I, P																																																				

Continued...

## General Purpose Concrete Cleaner

expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC
cross-linked polymer - pillow	3	throw	skiploader	R, DGC, RT
foamed glass - particulate	4	blower	skiploader	R, W, P, DGC
foamed glass - pillow	4	throw	skiploader	R, P, DGC., RT

## Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT: Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

Moderate hazard.

- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Stop leak if safe to do so.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Neutralise/decontaminate residue (see Section 13 for specific agent).
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

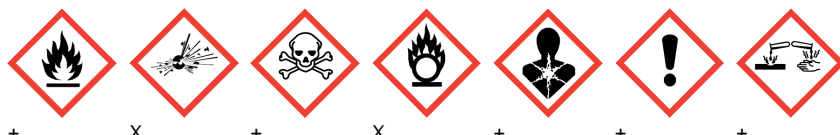
## SECTION 7 HANDLING AND STORAGE

## Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ <b>DO NOT allow material to contact humans, exposed food or food utensils.</b></li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
Other information	

## Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> <li>▶ Polyethylene or polypropylene container.</li> <li>▶ Packing as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
Storage incompatibility	<ul style="list-style-type: none"> <li>▶ Avoid contact with copper, aluminium and their alloys.</li> <li>▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.</li> </ul>



+

X

+

X

+

+

+

X — Must not be stored together

0 — May be stored together with specific precautions

+ — May be stored together

Continued...

## General Purpose Concrete Cleaner

## 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	2-Butoxyethanol	2-Butoxyethanol	20 ppm / 96.9 mg/m3	242 mg/m3 / 50 ppm	Not Available	Not Available
Australia Exposure Standards	diethanolamine	Diethanolamine	3 ppm / 13 mg/m3	Not Available	Not Available	Not Available

## EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
2-Butoxyethanol	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
sodium metasilicate, pentahydrate	Sodium metasilicate pentahydrate	6.6 mg/m3	73 mg/m3	440 mg/m3
sodium metasilicate, pentahydrate	Sodium silicate; (Sodium metasilicate)	3.8 mg/m3	42 mg/m3	250 mg/m3
diethanolamine	Diethanolamine	3 mg/m3	28 mg/m3	130 mg/m3
sodium nitrate	Sodium nitrate	4.1 mg/m3	45 mg/m3	270 mg/m3
sodium sulfate	Sodium sulfate, anhydrous	9.8 mg/m3	110 mg/m3	650 mg/m3
d-limonene	Limonene, d-	15 ppm	67 ppm	170 ppm

Ingredient	Original IDLH	Revised IDLH
2-Butoxyethanol	700 ppm	Not Available
sodium lauryl ether sulfate	Not Available	Not Available
sodium metasilicate, pentahydrate	Not Available	Not Available
cocamide diethanolamide.	Not Available	Not Available
diethanolamine	Not Available	Not Available
sodium nitrate	Not Available	Not Available
isothiazolinones, mixed	Not Available	Not Available
sodium sulfate	Not Available	Not Available
Brilliant Blue R	Not Available	Not Available
d-limonene	Not Available	Not Available
citral	Not Available	Not Available
lemon oil	Not Available	Not Available

## OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sodium lauryl ether sulfate	E	≤ 0.01 mg/m³
sodium metasilicate, pentahydrate	E	≤ 0.01 mg/m³
cocamide diethanolamide.	E	≤ 0.1 ppm
sodium nitrate	E	≤ 0.01 mg/m³
isothiazolinones, mixed	E	≤ 0.1 ppm
sodium sulfate	E	≤ 0.01 mg/m³
d-limonene	E	≤ 0.1 ppm
citral	E	≤ 0.1 ppm
lemon oil	E	≤ 0.1 ppm

## Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

## MATERIAL DATA

Fragrance substance with is an established contact allergen in humans.

Continued...

## General Purpose Concrete Cleaner

Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012

for diethanolamine:

Odour Threshold: 2.6 ppm

The TLV-TWA is thought to be protective against the significant risk of eye damage and skin irritation.

Odour Safety Factor (OSF)

OSF=1.7 (DIETHANOLAMINE)





for d-Limonene:

CEL TWA: 30 ppm, 165.6 mg/m<sup>3</sup> (compare WEEL-TWA\*)

(CEL = Chemwatch Exposure Limit)

A Workplace Environmental Exposure Level\* has been established by AIHA (American Industrial Hygiene Association) who have produced the following rationale: d-Limonene is not acutely toxic. In its pure form it is not a sensitiser but is irritating to the skin. Although there is clear evidence of carcinogenicity in male rats, the effect has been attributed to an alpha-2u-globin (a2u-G) renal toxicity which is both species and gender specific. Humans do not synthesise a2u-G, and metabolism studies indicate that 75% to 95% of d-limonene is excreted in 2-3 days with different metabolites identified between humans and rats. In a 2-year study, liver effects were noted in male mice at 500 mg/kg and reduced survival was noted in female rats at 600 mg/kg. The no observable effect levels (NOELs) were 250 and 300 mg/kg, respectively. A WEEL of 30 ppm is recommended to protect against these effects.

## Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.	
	The basic types of engineering controls are:	
	Process controls which involve changing the way a job activity or process is done to reduce the risk.	
	Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.	
	Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.	
	An approved self contained breathing apparatus (SCBA) may be required in some situations.	
	Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.	
	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
Personal protection	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
	3: Intermittent, low production.	3: High production, heavy use
	4: Large hood or large air mass in motion	4: Small hood-local control only
	Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.	
	   	

## General Purpose Concrete Cleaner

<b>Eye and face protection</b>	<ul style="list-style-type: none"> <li>▸ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.</li> <li>▸ Chemical goggles whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted.</li> <li>▸ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.</li> <li>▸ Alternatively a gas mask may replace splash goggles and face shields.</li> <li>▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> </ul>
<b>Skin protection</b>	See Hand protection below
<b>Hands/feet protection</b>	<ul style="list-style-type: none"> <li>▸ Elbow length PVC gloves</li> <li>▸ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▸ 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.</li> <li>▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<ul style="list-style-type: none"> <li>▸ Overalls.</li> <li>▸ P.V.C. apron.</li> <li>▸ Barrier cream.</li> <li>▸ Skin cleansing cream.</li> <li>▸ Eye wash unit.</li> </ul>

## 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:

General Purpose Concrete Cleaner

Material	CPI
VITON	A
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
PVA	C
PVC	C
TEFLON	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 AK-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

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

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

^ - Full-face

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

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

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Continued...



## General Purpose Concrete Cleaner

## Information on basic physical and chemical properties

<b>Appearance</b>	Not Available		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	1.01
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Applicable
<b>pH (as supplied)</b>	13	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Available
<b>Flash point (°C)</b>	Not Applicable	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Applicable	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Applicable	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Miscible	<b>pH as a solution (1%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available

## SECTION 10 STABILITY AND REACTIVITY

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>▶ Unstable in the presence of incompatible materials.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 TOXICOLOGICAL INFORMATION

## Information on toxicological effects

<b>Inhaled</b>	<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. 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>Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales.</p> <p>Not normally a hazard due to non-volatile nature of product</p>
<b>Ingestion</b>	<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>Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may</p>

Continued...

## General Purpose Concrete Cleaner

	result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation.
<b>Skin Contact</b>	<p>The material can produce severe chemical burns following direct contact with the skin.</p> <p>Skin contact is not thought to produce harmful health effects (as classified under EC Directives using animal models). Systemic harm, however, has been identified following exposure of animals by at least one other route and the material may still produce health damage following entry through wounds, lesions or abrasions. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.</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>
<b>Eye</b>	<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>Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight.</p>
<b>Chronic</b>	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p>

General Purpose Concrete Cleaner	TOXICITY	IRRITATION
	Not Available	Not Available
2-Butoxyethanol	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Inhalation (rat) LC50: 449.48655 mg/l/4H <sup>[2]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
sodium lauryl ether sulfate	TOXICITY	IRRITATION
	Oral (rat) LD50: 1600 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 25 mg/24 hr moderate
sodium metasilicate, pentahydrate	TOXICITY	IRRITATION
	Oral (rat) LD50: 847 mg/kg <sup>[2]</sup>	Skin (human): 250 mg/24h SEVERE
		Skin (rabbit): 250 mg/24h SEVERE
cocamide diethanolamide.	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Not Available
diethanolamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 8342.88 mg/kg <sup>[2]</sup>	Eye (rabbit): 5500 mg - SEVERE
	Oral (rat) LD50: 677.04 mg/kg <sup>[2]</sup>	Eye (rabbit): 0.75 mg/24 hr SEVERE
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 50 mg (open)-mild
		Skin (rabbit): 500 mg/24 hr-mild
diethanolamine	TOXICITY	IRRITATION
		Skin: adverse effect observed (irritating) <sup>[1]</sup>

## General Purpose Concrete Cleaner

sodium nitrate	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg <sup>[1]</sup>	Not Available
	Oral (rat) LD50: 1267 mg/kg <sup>[2]</sup>	
isothiazolinones, mixed	TOXICITY	IRRITATION
	dermal (rat) LD50: >1008 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irreversible damage) <sup>[1]</sup>
	Oral (rat) LD50: 53 mg/kg <sup>[2]</sup>	Skin: adverse effect observed (corrosive) <sup>[1]</sup>
sodium sulfate	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
Brilliant Blue R	TOXICITY	IRRITATION
	Not Available	Not Available
d-limonene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Skin (rabbit): 500mg/24h moderate
citral		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2250 mg/kg <sup>[2]</sup>	Skin (guinea pig): 1%/48h - mod
	Oral (rat) LD50: 3450 mg/kg <sup>[2]</sup>	Skin (guinea pig): 100mg/24h SEVERE
		Skin (human): 40 mg/24h - mild
		Skin (man): 16 mg/48h - SEVERE
		Skin (pig): 50 mg/24h - SEVERE
lemon oil	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg <sup>[2]</sup>	Skin (rabbit): 500 mg/24h mod
	Oral (rat) LD50: 2840 mg/kg <sup>[2]</sup>	
<b>Legend:</b> 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		

## SODIUM LAURYL ETHER SULFATE

\* [CESIO]

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-pentaoxaheptacosan-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

## General Purpose Concrete Cleaner

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>

Alkyl ether sulfates (alcohol or alkyl ethoxysulfates) (AES) (syn: AAASD ,alkyl alcohol alkoxyate sulfates, SLES) are generally classified according to Comité Européen des Agents de Surface et leurs Intermédiaires Organiques (CESIO) as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R36 (Irritating to eyes). An exception has been made for AES (2-3EO) in a concentration of 70-75% where R36 is substituted with R41 (Risk of serious damage to eyes).

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

In assessing this family the Cosmetic Ingredient Review (CIR) Expert Panel recognized that most of the acute oral toxicity, dermal irritation and sensitization, subchronic and chronic oral toxicity, reproductive and developmental toxicity, carcinogenicity, and photosensitization studies have been conducted on ammonium laureth sulfate and sodium laureth sulfate. Sodium and ammonium laureth sulfate have not evoked adverse responses in any toxicological testing, including acute oral toxicity, sub-chronic and chronic oral toxicity, reproductive and develop-mental toxicity, carcinogenicity, and photosensitization studies. These data, however, are considered a sufficient basis for concluding that the other ingredients are safe in the practices of use and concentration described in the safety assessment because of the fundamental chemical similarities between them and because they all are chemically similar salts/salts are expected to be dissociated in any product formulation independent of whether the salt is sodium, ammonium, magnesium, or zinc) of sulfated ethoxylated alcohols, and they all function as surfactants in cosmetic formulations. Based on these considerations, safety test data on one ingredient may be extrapolated to all of them. The panel noted that sodium laureth sulfate and ammonium laureth sulfate can produce eye and/or skin irritation in experimental animals and in some human test subjects; irritation may occur in some users of cosmetic formulations containing these ingredients. The irritant effects, however, are similar to those produced by other detergents, and the severity of the irritation appears to increase directly with concentration

**Acute toxicity:** AES are of low acute toxicity. Neat AES are irritant to skin and eyes. The irritation potential of AES containing solutions depends on concentration. Local dermal effects due to direct or indirect skin contact with AES containing solutions in hand-washed laundry or hand dishwashing are not of concern because AES is not a contact sensitizer and AES is not expected to be irritating to the skin at in-use concentrations. The available repeated dose toxicity data demonstrate the low toxicity of AES. Also, they are not considered to be mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. The consumer aggregate exposure from direct and indirect skin contact as well as from the oral route via dishware residues results in an estimated total body burden of 29 ug /kg bw/day.

AES are easily absorbed in the intestine in rats and humans after oral administration. Radiolabelled C11 AE3S and C12 AE3S were extensively metabolized in rats and most of the  $^{14}\text{C}$ -activity was eliminated via the urine and expired air independently of the route of administration (oral, intraperitoneal or intravenous). The main urinary metabolite from C11 AE3S is propionic acid-3-(3EO)-sulfate. For C12 and C16 AE3S, the main metabolite is acetic acid-2-(3EO)-sulfate. The alkyl chain appears to be oxidised to  $\text{CO}_2$  which is expired. The EO-chain seems to be resistant to metabolism.

AES are better tolerated on the skin than, e.g., alkyl sulfates and it is generally agreed that the irritancy of AES is lower than that of other anionic surfactants. Alkyl chain lengths of 12 carbon atoms are considered to be more irritating to the skin compared to other chain lengths. The skin irritating properties of AES normally decrease with increasing level of ethoxylation. Undiluted AES should in general be considered strongly irritating. Even at concentrations of 10% moderate to strong effects can be expected. However, only mild to slight irritation was observed when a non-specified AES was applied at 1% to the skin.

**Subchronic toxicity:** A 90-day subchronic feeding study in rats with 1% of AE3S or AE6S with alkyl chain lengths of C12-14 showed only an increased liver/body weight ratio. In a chronic oral study with a duration of 2 years, doses of C12-AE3S of 0.005 - 0.05% in the diet or drinking water had no effects on rats. The concentration of 0.5% sometimes resulted in increased kidney or liver weight.

Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene)  $n=3$ . One study indicated that C16-18 POE  $n=18$  had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE  $n=2$  (CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE  $n=3$  (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction.

**Reproductive and developmental toxicity:** No evidence of reproductive and teratogenic effects was seen in a two-generation study in rats fed with a mixture (55:45) of AES and linear alkylbenzene sulfonates. Dietary levels of 0.1, 0.5, and 1% were administered to the rats either continuously or during the period of major organogenesis during six pregnancies. No changes in reproductive or embryogenic parameters were observed.

Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives.

**Carcinogenicity:** Chronic dietary studies conducted with rats showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).

**NOTE:** Some products containing AES/ SLES have been found to also contain traces (up to 279 ppm) of 1,4-dioxane; this is formed as a by-product during the ethoxylation step of its synthesis. The U.S. Food and Drug Administration recommends that these levels be monitored. The U.S. Environmental Protection Agency classifies 1,4-dioxane to be a probable human carcinogen

## General Purpose Concrete Cleaner

(not observed in epidemiological studies of workers using the compound, but resulting in more cancer cases in controlled animal studies), and a known irritant with a no-observed-adverse-effects level of 400 milligrams per cubic meter at concentrations significantly higher than those found in commercial products. Under Proposition 65, 1,4-dioxane is classified in the U.S. state of California to cause cancer. The FDA encourages manufacturers to remove 1,4-dioxane, though it is not required by federal law.

**Sensitizing potential:** 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-pentaosaheptacosan-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 autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing

#### Toxicokinetics:

Following oral exposure, AES is readily absorbed in the gastrointestinal tract in human and rat and excreted principally via the urine or faeces depending on the length of the ethoxylate chain but independently of the route of administration. Once absorbed, AES is extensively metabolized by beta- or omega oxidation. The alkyl chain appears to be oxidized to CO<sub>2</sub> which is expired. The EO-chain seems to be resistant to metabolism. Regarding the different anions, it is expected that the salts will be converted to the acid form in the stomach. This means that for all types of parent chemical the same compound structure eventually enters the small intestine. Hence, the situation will be similar for compounds originating from different salts and therefore no differences in uptake are anticipated.

The length of the ethoxylate portion in an AES molecule seems to have an important impact on the biokinetics of AES in humans and in the rat. Alcohol ethoxysulfates with longer ethoxylate chains (>7-9 EO units) are excreted at a higher proportion in the faeces. This is however not of interest for the AES within this category as their ethoxylation grade is 1 to 2.5.

#### Dermal absorption

There are two reliable and relevant studies available assessing the dermal absorption rate of AES. The study with AES (C12 -14; 2 EO) Na (CAS 68891-38-3) was performed according to OECD guideline 428 with human skin of the abdomen region (3 donors, n=2). The test substance was applied at a concentration of 10% for 24 h

The mean amount removed from the skin surface (skin wash) ranged from 87.16% to 94.56% of the dose applied. The amounts in the receptor could not be quantified, since it was below the analytical limit of quantification (LOQ). The mean recovery in the two first tape strips was 1.48% during all performed experiments. In the further 18 tape strips a mean recovery of 2.86% was documented. The recovery values for the cryocuts have accounted 0.56% in mean.

The mean absorbed dose, sum of the amounts found in the viable epidermis, dermis and receptor medium was 0.56%. The mean recovery values have varied from 90.90% to 100.21%, which complies with the acceptance criteria of 100 ± 15%.

There is also an in vivo study according to OECD guideline 427 for AES (C12 -14; 2 EO) Na (CAS 68891-38-3) available (Aulmann, 1996). Wistar rats were exposed to 1% aqueous solutions of the test item for 15 min and 48 h under semi-occlusive conditions. The mean amount of AES (C12-14; 2 EO) Na (CAS 68891-38-3) removed from the skin surface after the 15 min exposure period (via washing) ranged from 92.8% to 97.2% of the dose and from 91.6% to 98.4% after 48 h when the skin was not washed until sacrifice. The amounts in faeces and skin could not always be quantified, since it was below the analytical limit of quantification (LOQ).

The mean absorbed dose, sum of the amounts found in urine, faeces and skin in the experiment with washing was about 0.1% and 0.9% without washing.

The mean recovery values varied from 98.6% to 103%.

Taking the results of both studies together the dermal absorption is very low. The in vitro study with human skin indicated the dermal absorption to be 0.56% within 24 h and the in vivo study indicated the dermal absorption to be 0.9% within 48 h. The mean recovery rates on the skin are greater than 87%. These data demonstrate that the test substance remains on the skin surface. Thus, the value of 0.9% dermal absorption is taken for the dermal absorption.

#### References:

Danish EPA - Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products (2001). Environmental Project No. 615, pp. 24-28

HERA (2003). Human & Environmental Risk Assessment on ingredients of European household cleaning products Alcohol Ethoxysulphates, Human Health Risk Assessment Draft, 2003. <http://www.heraproject.com>.

Final Report of the Amended Safety Assessment of Sodium Laureth Sulfate and Related Salts of Sulfated Ethoxylated Alcohols: (International Journal of Toxicology 29 (Supplement 3) 151S-161S: 2010  
<http://journals.sagepub.com/doi/pdf/10.1177/1091581810373151>

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

#### SODIUM METASILICATE, PENTAHYDRATE

The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.



## General Purpose Concrete Cleaner

sodium metasilicate anhydrous:

\*Stephan SDS Ninol 49-CE

Fatty acid amides (FAA) are ubiquitous in household and commercial environments. The most common of these are based on coconut oil fatty acids alkanolamides. These are the most widely studied in terms of human exposure.

Fatty acid diethanolamides (C8-C18) are classified by Comité Européen des Agents de Surface et de leurs Intermediaires Organiques (CESIO) as Irritating (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes). Fatty acid monoethanolamides are classified as Irritant (Xi) with the risk phrases R41

Several studies of the sensitization potential of cocoamide diethanolamide (DEA) indicate that this FAA induces occupational allergic contact dermatitis and a number of reports on skin allergy patch testing of cocoamide DEA have been published. These tests indicate that allergy to cocoamide DEA is becoming more common.

Alkanolamides are manufactured by condensation of diethanolamine and the methylester of long chain fatty acids. Several alkanolamides (especially secondary alkanolamides) are susceptible to nitrosamine formation which constitutes a potential health problem. Nitrosamine contamination is possible either from pre-existing contamination of the diethanolamine used to manufacture cocoamide DEA, or from nitrosamine formation by nitrosating agents in formulations containing cocoamide DEA. According to the Cosmetic Directive (2000) cocoamide DEA must not be used in products with nitrosating agents because of the risk of formation of N-nitrosamines. The maximum content allowed in cosmetics is 5% fatty acid dialkanolamides, and the maximum content of N-nitrosodialkanolamines is 50 mg/kg. The preservative 2-bromo-2-nitropropane-1,3-diol is a known nitrosating agent for secondary and tertiary amines or amides. Model assays have indicated that 2-bromo-2-nitropropane-1,3-diol may lead to the N-nitrosation of diethanolamine forming the carcinogenic compound, N-nitrosodiethanolamine which is a potent liver carcinogen in rats (IARC 1978).

Several FAAs have been tested in short-term genotoxicity assays. No indication of any potential to cause genetic damage was seen. Lauramide DEA was tested in mutagenicity assays and did not show mutagenic activity in *Salmonella typhimurium* strains or in hamster embryo cells. Cocoamide DEA was not mutagenic in strains of *Salmonella typhimurium* when tested with or without metabolic activation

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Miljøministeriet (Danish Environmental Protection Agency)

# COCAMIDE DIETHANOLAMIDE.

For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides)

The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented. The Fatty nitrogen-derived amides (FND amides) comprise four categories:

Subcategory I: Substituted Amides

Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components)

Subcategory III: Imidazole Derivatives

Subcategory IV: FND Amphoterics

Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies.

Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II.

Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories.

Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonella reverse mutation assay exist for all of the subcategories.

Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II.

In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.

Some typical applications of FND Amides are:

masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antilocking additives for polymers.

The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or

## General Purpose Concrete Cleaner

erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health.

The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals.

Coconut oil diethanolamine condensate is possibly carcinogenic to humans (IARC Group 2B)

In a study of the dermal application in mice, coconut oil diethanolamine condensate increased the incidence of hepatocellular carcinoma and hepatocellular adenoma in males and females, and of hepatoblastoma in males. The incidence of renal tubule adenoma and carcinoma combined was also increased in males. In a study of dermal application in rats, no increase in tumour incidence was observed.

Tumours of the kidney and hepatoblastoma are rare spontaneous neoplasms in experimental animals.

The amide linkage between diethanolamine and of the fatty acid moiety is resistant to metabolic hydrolysis. The carcinogenic effects of the coconut diethanolamine condensate used in the cancer bioassay may be due to the levels of diethanolamine (18.2%) in the solutions tested. Mechanistic data are very weak to evaluate the carcinogenic potential of coconut oil diethanolamine condensate per se.

A test material composed primarily of diethanolamides of coconut oil acids, with unreacted diethanolamine, alkanolamides of unsaturated acids, and amine salts of the acids, was evaluated. The polar nitrosamine, N-nitrosodiethanolamine, was detected at a concentration of 219 ppb

Under test conditions, there was no evidence of carcinogenic activity of the test material in male rats administered 50 or 100 mg/kg bw. There was an equivocal evidence of carcinogenic activity in female rats based on a marginal increase in the incidences of renal tubule neoplasms. However, the absence of an increase in neoplasms in the 100 mg/kg group in the presence of increased hyperplasia makes the association with chemical exposure uncertain.

Coconut oil fatty acid diethanolamine condensates (Cocamide DEA) have not been experimentally determined to be carcinogenic

Metabolic fate:  
Lauramide diethanolamine, a major component of coconut oil diethanolamine condensate, in the presence of rat liver and kidney microsomes, is rapidly converted into 11 and 12 hydroxy derivatives

Toxicity:

Both Oral and Dermal LD50s for rats exceeded 2000 mg/kg for cocamide monoisopropanolamide (CAS RN: 1335203-30-9)

Repeat dose toxicity:

The 28d NOAEL of coconut fatty acid ethanolamide (Comperlan 100) to rat is considered to be 1500 mg/kg bw/d.

Groups of 10 male and 10 female rats were orally gavaged with the test substance diluted in olive oil 5 d/wk for 28 d. Clinical signs, bodyweight, hematology, clinical chemistry, urinalysis, gross and microscopic pathology were recorded. Additional groups of 5 male and 5 female rats were kept for a 4 month recovery period.

No treatment-related adverse effects were observed at any of the doses

A study was conducted to evaluate the subchronic toxic effects of amides, C8 -18 and C18 -unsatd., N, N-bis(hydroxyethyl) when administered by dermal route in B6C3F1 mice.

Under the test conditions, the No Observed Adverse Effect Level (NOAEL) of cocamide monoisopropanolamide was considered to be 50 mg/kg body weight.

Genetic toxicity:

Under the test conditions, test substance was considered to be non-clastogenic in cultured human lymphocytes in vitro. However, the test material may have the potential to disturb mitotic processes and to induce numerical chromosome aberrations.

## DIETHANOLAMINE

While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.

- ▶ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis.
- ▶ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient.

Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.

**Inhalation:**

Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs.

Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.

Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.

Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies.

While most polyurethane amine catalysts are not sensitizers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function,

## General Purpose Concrete Cleaner

	<p>breathlessness, chronic bronchitis, and immunologic lung disease.</p> <p>Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.</p> <p><b>Skin Contact:</b></p> <p>Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.</p> <p>Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.</p> <p><b>Eye Contact:</b></p> <p>Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations.</p> <p>Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.)</p> <p>Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.</p> <p>The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases.</p> <p>Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.</p> <p><b>Ingestion:</b></p> <p>The oral toxicity of amine catalysts varies from moderately to very toxic.</p> <p>Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract.</p> <p>Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.</p> <p>Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.</p> <p><b>Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000 Alliance for Polyurethanes Industry</b></p>
<b>SODIUM NITRATE</b>	<p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man.</p> <p>This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p>
<b>SODIUM SULFATE</b>	<p>for sodium sulfate:</p> <p>Sulfate (and sodium) ions are important constituents of the mammalian body and of natural foodstuffs and there is a considerable daily turnover of both ions (several grams/day expressed as sodium sulfate). Near-complete absorption of dietary sulfates may occur at low concentration, depending on the counter-ion, but absorption capacity can be saturated at higher artificial dosages resulting in cathartic effects. Absorption through skin can probably be ignored since sodium sulfate is fully ionised in solution. One source suggests that very high levels of sulfate in urine may occur due to absorption from dust inhalation. At dietary levels, excretion is mainly in the urine. Sulfates are found in all body cells, with highest concentrations in connective tissues, bone and cartilage.</p> <p>Sulfates play a role in several important metabolic pathways, including those involved in detoxification processes.</p> <p>The acute toxicity (LD50) of sodium sulfate has not been reliably established but is probably far in excess of 5000 mg/kg. In an inhalation study with an aerosol, no adverse effects were found at 10 mg/m<sup>3</sup>. Also human data indicate a very low acute toxicity of sodium sulfate. Human clinical experience indicates that very high oral doses of sodium sulfate, 300 mg/kg bw up to 20 grams for an adult, are well tolerated, except from (intentionally) causing severe diarrhoea. WHO/FAO did not set an ADI for sodium sulfate. There is no data on acute dermal toxicity, but this is probably of no concern because of total ionisation in solution.</p> <p>Sodium sulfate is not irritating to the skin and slightly irritating to the eyes. Respiratory irritation has never been reported. Based on wide practical experience with sodium sulfate, in combination with the natural occurrence of sulfate in the body, sensitising effects are highly unlikely.</p> <p>No suitable dermal and inhalation repeated-dose toxicity studies are available. Valid oral repeated dose toxicity studies with 21, 28 and 35 day studies in hens and pigs are available. Toxicity was confined to changes in bodyweight, water and feed intake and diarrhoea. These changes occurred only at very high doses of sodium sulfate. In ruminants, high concentrations of sulfate in food may result in the formation of toxic amounts of sulfites by bacterial reduction the rumen, leading to poly-enkephalomalacia. The available data do not allow the derivation of a NOAEL. Based on available consumer data, a daily dose of around 25 mg/kg/day is well tolerated by humans.</p> <p>There are no data on <i>in vitro</i> and <i>in vivo</i> genotoxicity, apart from a negative Ames test. There is no valid oral carcinogenicity study. Limited data from experimental studies support the notion that a substance that is abundantly present in and essential to the body is unlikely to be carcinogenic.</p> <p>Limited data of poor validity did not provide an indication of toxicity to reproduction.</p> <p>Equivocal Tumorigen by RTECS criteria. Reproductive effector in mice.</p>
<b>D-LIMONENE</b>	<p>The substance is classified by IARC as Group 3:</p> <p><b>NOT</b> classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p> <p>Tumorigenic by RTECS criteria</p>
<b>CITRAL</b>	<p>For monoterpenes:</p> <p>The chemical category designated terpenoid hydrocarbons includes three simple C<sub>10</sub> isomeric monocyclic terpene hydrocarbons (<i>d</i>-limonene, <i>dl</i>-limonene, and terpinolene) two simple C<sub>10</sub> acyclic terpene hydrocarbons (<i>beta</i>-myrcene and dihydromyrcene) and mixtures composed primarily of <i>d</i>-limonene, <i>dl</i>-limonene (dipentene), terpinolene, myrcene, and <i>alpha</i> and <i>beta</i>-pinene</p> <p>Monoterpene hydrocarbons are mainly released by coniferous woodland such as pine trees, cedars, redwood and firs. To a</p>



## General Purpose Concrete Cleaner

lesser extent, they are also produced and released by deciduous plants. They are common components of traditional foods occurring in essentially all fruits and vegetables.

Members of this chemical category are of very low acute toxicity

Studies of terpene hydrocarbons indicate that they are rapidly absorbed, distributed, metabolised and excreted. The principal metabolic pathway involves side chain oxidation to yield monocyclic terpene alcohols and carboxylic acids. These metabolites are mainly conjugated with glucuronic acid and excreted in the urine, or to a lesser extent in the feces. A secondary pathway involves epoxidation of either the exocyclic or endocyclic double bond yielding an epoxide that is subsequently detoxicated via formation of the corresponding diol or conjugation with glutathione. Although some species- and sex-specific differences exist, studies for *d*-limonene and *beta*-myrcene indicate that the monoterpene hydrocarbons in this chemical category will participate in common pathways of absorption, distribution, metabolism and excretion.

**Genotoxicity:** Based on the results of this *in vivo* genotoxicity assay and the numerous *in vitro* genotoxicity assays, it is unlikely that any of these materials would exhibit a significant genotoxic potential *in vivo*.

**Carcinogenicity:** Under the conditions of 2-year gavage studies, conducted by NTP, there was clear evidence of carcinogenic activity of *d*-limonene for male F344/N rats as shown by increased incidences in tubular cell hyperplasia, adenomas, and adenocarcinomas of the kidney. There was no evidence of carcinogenic activity of *d*-limonene for female rats receiving 300 or 600 mg/kg bw/d. It has been demonstrated that renal lesions, which were observed in the NTP study, resulted from the accumulation of aggregates of *alpha*-2 microglobulin (a low molecular-weight protein synthesised in the liver) and limonene-1,2-epoxide in the P2 segment of the renal proximal tubule. While humans produce low molecular weight serum proteins, which are reabsorbed by the kidney, there is no evidence that a similar *alpha*-2 microglobulin is produced.

The kidney changes seen in male rats administered limonene have been well characterized, and are known to be specific to the male rat and of no significance in human risk assessment.

**Reproductive toxicity:** Substances within this chemical category exhibit low reproductive toxicity potential. This is based on the results of three reproductive toxicity assays. using sweet orange peel oil predominantly composed of *d*-limonene and *beta*-myrcene.

**Developmental toxicity:** Given the results of six developmental toxicity assays using limonene, sweet orange oil and *beta*-myrcene, it may be concluded that the substances within this chemical category exhibit low developmental toxicity potential

Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides, so formed, were found to possess sensitising capacity *in vivo* and *in vitro* and to chemically reactive towards a common hexapeptide containing the most common nucleophilic amino acids. Further-more, a SAR study of potentially prohaptenic alkenes demonstrated that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, whereas related alkenes containing isolated double bonds or an acyclic conjugated diene were weak or nonsensitizing compounds. This difference in sensitizing capacity of conjugated dienes as compared to alkenes with isolated double bonds was found to be due to the high reactivity and sensitizing capacity of the allylic epoxides metabolically formed from conjugated dienes.

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

Ann-Therese Karlberg et al: Chem. Res. Toxicol. 2008, 21, pp 53–69

[http://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karlberg\\_2008.pdf](http://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karlberg_2008.pdf)

for citral

Citral is rapidly absorbed from the gastrointestinal tract. Much of an applied dermal dose is lost due to its extreme volatility, but the citral remaining on the skin was fairly well absorbed. Citral is rapidly metabolised and excreted as metabolites. Urine is the major route of elimination.

**Acute toxicity** of this chemical is low in rodents because the oral or dermal LD50 values were more than 1000 mg/kg. This chemical is irritating to skin and not irritating to eyes in rabbits. There is some evidence that this chemical is a human skin sensitizer.

**Repeat dose toxicity:** Several repeated dose oral studies show no adverse effect of citral at less than 1,000 mg/kg/day exposure and some histological changes in the nasal cavity or forestomach, the first exposure sites, probably due to irritation, at more than 1,000 mg/kg/day. Male and female F344/N rats received microencapsulated citral in feed at concentrations of 0, 0.63, 1.25, 2.5, 5 and 10% (resultant doses: 0, 142, 285, 570, 1,140 and 2,280 mg/kg/day) for 14 days. Minimal to mild hyperplasia and/or squamous metaplasia of the respiratory epithelium was observed in nasal cavity without inflammatory response at 1,140 and 2,280 mg/kg/day of both sexes. The NOAEL was established at 570 mg/kg/day. In an OECD preliminary reproduction toxicity screening test [TG 421], citral was administered to Crj:CD (SD) rats by gavage at doses of 0, 40, 200 and 1,000 mg/kg/day in males for 46 days and in females for 39-50 days including before and through mating and gestation periods and until day 3 of lactation. Squamous hyperplasia, ulcer and granulation in lamina propria were observed in the forestomach at 1,000 mg/kg/day of both sexes. Therefore, the NOAEL for repeated dose toxicity was 200 mg/kg/day for both sexes.

**Developmental toxicity:** in the above preliminary reproductive study, no effects were detected in reproductive ability, organ weights or histopathology of the reproductive organs of both sexes, and delivery or maternal behavior. However, body weights of male and female pups were reduced in the 1000 mg/kg group. Therefore, an oral NOAEL for developmental toxicity was 200 mg/kg/day.

In a teratogenicity study, SD pregnant rats were exposed to citral by inhalation for 6 hr/day on gestation days 6-15 at mean concentration of 0, 10 or 34 ppm as vapour, or 68 ppm as an aerosol/vapour mixture. Even in the presence of the maternal effects, no significant teratogenicity was noted at 68 ppm. An inhalation NOAEL of teratogenicity was established at 68 ppm (423 mg/m3).

**Genotoxicity:** Seven bacterial reverse mutation studies indicate negative results with and without metabolic activation. As for non-bacterial *in vitro* study, two chromosomal aberration results in Chinese hamster cells are negative however one positive result in sister chromatid exchange is given in the same cells. Additionally, two *in vivo* micronucleus tests in rodents indicate negative results. Based on the above information, the genotoxic potential of citral can be considered to be negative.

**Carcinogenicity:** A NTP study shows that there was no evidence of carcinogenic activity in male/female rats and male mice but some evidence of malignant lymphoma in female mice (up to 4,000 ppm in feed in rats and up to 2,000 ppm in feed in mice). Dermal application of citral induces prostate hyperplasia with low severity only in some strains of rats. However, the NTP oral carcinogenicity studies in rats and mice found no evidence of lesions (neoplastic or non-neoplastic) in any male reproductive organ, including the prostate. The health significance of the effects seen in the dermal studies in rats is uncertain due to dramatic strain differences and it is noted that the work has primarily been performed in a single laboratory.

## General Purpose Concrete Cleaner

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

For dialdehydes:

Dienaldehydes are by-products of peroxidation of polyunsaturated lipids and commonly found in many foods or food-products.

Both National Cancer Institute (NCI) and NTP have expressed great concern on the potential genotoxicity and carcinogenicity of dialdehydes. 2,4-Decadienal and 2,4-hexadienal are autooxidation products of polyunsaturated fatty acids

Several researchers have implied there could be a link between exposures to lipid peroxidation products in the diet and the development of human cancers. Lipid hydroperoxides have been shown to give rise to low intracellular levels of 2,4-decadienal and other alpha-beta-unsaturated aldehydes that are known to be reactive with DNA. Ingested lipid oxidation products and oxidized fats have been reported to cause increased excretion of mutagens, cellular injury to liver and kidneys, increased cell proliferation in the gastrointestinal tract, and other nonspecific tissue injury and irritation effects resulting from induced oxidative stress.

Treatment related changes following gastric lavage administration for up to 3 months were similar for 2,4-hexadienal and 2,4-decadienal, and in both cases the forestomach and nose were identified as target organs. In two week studies of 2,4-hexadienal and 2,3 decadienal in rats and mice, forestomach lesions included necrosis and ulceration; epithelial hyperplasia was observed in rats and mice in the 2,4-hexadienal studies. In the 3-month studies of 2,4-hexadienal and 2,4-decadienal, forestomach epithelial hyperplasia and degeneration with or without chronic active inflammation occurred in addition to nasal olfactory epithelia atrophy and necrosis.

Carcinogenicity and mutagenicity data from testing of dienals are limited. In the two year carcinogenicity studies, 2,4-hexadienal induced significantly increased incidences of forestomach neoplasms in rats and mice.

NTP Technical Report 2,4-decadienal

Trans, trans-2,4-decadienal (tt-DDE or 2,4-De), a specific type of dialdehyde, is abundant in heated oils and has been associated with lung adenocarcinoma development in women due to their exposure to oil fumes during cooking. Cultured human bronchial epithelial cells (BEAS-2B cells) were exposed to 0.1 or 1.0 uM tt-DDE for 45 days, and oxidative stress, reactive oxygen species (ROS) production, GSH/GSSG ratio, cell proliferation, and expression of TNFalpha and IL-1beta were measured. The results show that tt-DDE induced oxidative stress, an increase in ROS production, and a decrease in GSH/GSSG ratio (glutathione status) in a dose-dependent manner. Treatment of BEAS-2B cells with 1.0 uM tt-DDE for 45 days increased cell proliferation and the expression and release of pro-inflammatory cytokines TNFalpha and IL-1beta. Cotreatment of BEAS-2B cells with antioxidant N-acetylcysteine prevented tt-DDE-induced cell proliferation and release of cytokines. Therefore, these results suggest that tt-DDE-induced changes may be due to increased ROS production and enhanced oxidative stress. Since increased cell proliferation and the release of TNF-alpha and IL-1beta are believed to be involved in tumor promotion, these results suggest that tt-DDE may play a role in cancer promotion. Previous studies on dialdehydes have focused on their genotoxic or carcinogenic effects in the gastrointestinal tract; the present study suggests a potential new role of tt-DDE as a tumor promoter in human lung epithelial cells.

Trans, Trans-2,4-Decadienal, a Product Found in Cooking Oil Fumes, Induces Cell Proliferation and Cytokine Production Due to Reactive Oxygen Species in Human Bronchial Epithelial Cells Louis W. Chang Wai-Sze Lo Pinpin Lin

Toxicological Sciences, Volume 87, Issue 2, 1 October 2005, Pages 337–343, <http://doi.org/10.1093/toxsci/kfi258>

2,4-Decadienal is produced by the oxidation of linoleic acid. 2,4-Decadienal is found as a contaminant in water. It is generated from polyunsaturated fatty acids by the action of plant lipoxygenases and is produced in mammalian tissues in certain physiological and pathophysiological processes such as lipid peroxidation, arachidonic acid oxidation, and reactions with reactive oxygen species

A member or analogue of a group of aliphatic, linear alpha,beta-unsaturated aldehydes and structurally related substances generally regarded as safe (GRAS) flavourings based, in part, on their self-limiting properties as flavouring substances in food; their low level of flavour use; the rapid absorption and metabolism of low in vivo concentrations by well-recognized biochemical pathways; adequate metabolic detoxication at much higher levels of exposure in humans and animals; the wide margins of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from subchronic and chronic studies. While some of the compounds described here have exhibited positive in vitro genotoxicity results, evidence of in vivo genotoxicity and carcinogenicity occurs only under conditions in which animals are repeatedly and directly exposed to high irritating concentrations of the aldehyde. These conditions are not relevant to humans who consume alpha,beta unsaturated aldehydes as flavour ingredients at low concentrations distributed in a food or beverage matrix.

Acetal derivatives are expected to readily hydrolyse in the acidic environments of the stomach, intestinal fluid and the liver to yield common aldehydes and alcohols.

alpha,beta-Unsaturated esters undergo rapid hydrolysis to their component alcohols and carboxylic acids in reactions that are principally catalysed by carboxylesterases. In general alpha,beta-unsaturated esters are more rapidly hydrolysed than their saturated analogues.

Linear alpha,beta-Unsaturated carboxylic acids participate directly in fatty acid metabolism and then are further metabolised to carbon dioxide and water.

Flavor and Extract Manufacturers Association (FEMA)

- Produces maternal effects (oogenesis, ovaries, fallopian tube changes) and effects live-birth index.

## LEMON OIL

The essential oils, oleoresins (solvent-free), and natural extractives (including distillates) derived from citrus fruits are generally recognized as safe (GRAS) for their intended use in foods for human consumption.

Botanicals such as citrus are comprised of hundreds of constituents, some of which have the potential to cause toxic effects; for example, bergapten (aka 5-methoxysporalen or 5-MOP) is a naturally occurring furanocoumarin (psoralen) in bergamot oil that causes phototoxicity. Under the rules governing cosmetic products in the European Union, citrus-derived ingredients must have furocoumarin content below 1 mg/kg in sun-protection and bronzing products.

Acute toxicity:

The dermal LD50 of undiluted either bitter orange or citrus reticulata (tangerine) leaf oil (described as "petitgrain bigarade oil") was reported as greater than 2000 mg/kg in rabbits. The dermal LD50 of undiluted mandarin peel oil (Citrus reticulata) was greater than 5000 mg/kg in rabbits

Dermal irritation:

## General Purpose Concrete Cleaner

Varying degrees of irritation were observed in animals treated with undiluted citrus aurantium amara (bitter orange) flower wax, unreported concentrations of either bitter orange or citrus reticulata (tangerine) leaf oil (described as "petitgrain bigarade oil"), or unreported concentrations of mandarin peel oil. In human subjects, no irritation was observed after topical exposure to citrus aurantium dulcis (orange) peel wax (100%), bergamot oil (up to 15%), either bitter orange or citrus reticulata (tangerine) leaf oil (described as "petitgrain bigarade oil"; up to 8%), lemon oil (up to 20%), or mandarin peel oil (8%).

**Ocular irritation:**

The eye tolerance of citrus aurantium amara (bitter orange) flower wax (> 50%) was tested in vitro using the SIRC cell strain. Tolerance was evaluated by measuring cytotoxicity. Negative controls solutions were physiological serum or sample diluent and the positive control solutions were 0.01% to 0.2% SDS. Negligible cytotoxicity was observed.

**Sensitisation:**

Bitter orange or citrus reticulata (tangerine) leaf oil (described as "petitgrain bigarade oil") and mandarin peel oil were not sensitising in human maximization tests. In studies of 250 dermatitic patients, less than 2.5% had positive reactions to bergamot oil, bitter orange oil, lemon oil, or sweet orange oil tested at 2% in paraffin.

In a retrospective study (2001-2010) of professional food handlers in Denmark, 8.5% (16/188) of the patients had positive reactions to orange peel and 7.9% (15/191) of the patients had positive reactions to lemon peel

**Phototoxicity and Photosensitisation:**

Citrus aurantium dulcis (orange) peel wax (100%) was not photosensitising in a human study. Mixed results were observed in non-human and human phototoxicity and photosensitisation studies of diluted and undiluted bergamot oil, either bitter orange or citrus reticulata (tangerine) leaf oil (described as "petitgrain bigarade oil"), lime oil, lemon oil, lemon fruit and peel juice, grapefruit oil, mandarin oil, tangerine oil, bitter orange oil, bitter orange peel oil, orange peel, orange mesocarp, and orange fruit. Many of the citrus-derived ingredients contain constituents that are photoactive agents, although those noted to be furocoumarin free tended not to induce photosensitisation.

Phototoxicity and photosensitisation were noted in several patients exposed to bergamot oil or limes/lime juice

**Carcinogenicity:**

Tumour-promoting activity was observed in mouse skin exposed to essential oils of orange (sweet), lemon, grapefruit, or lime. Groups of mice received weekly applications of 0.25 ml of the test substances 3 weeks after the application of 9,10-dimethyl-1,2-benzanthracene (DMBA) a tumour initiator. By the fifth week, papillomas were observed in mice exposed to lemon oil, grapefruit oil, and lime oil. Papillomas were observed in the orange oil group by the 12 th week. After 33 weeks, 10/20 mice in the lemon oil and lime oil treatment groups and 13/20 mice in the grapefruit oil and orange oil groups had papillomas.

No malignant skin tumours were observed in the orange oil group: treatment was stopped after 42 weeks. Squamous cell carcinomas of the skin were observed in 2 mice from the lemon oil group and 2 mice of the grapefruit oil group between weeks 36 and 55.

Non-dermal tumors during the treatment period were observed in 1 mouse of the orange oil group (a haemangioma of the subcutaneous tissue starting at week 7) and in 1 mouse of the grapefruit oil group (a spindle cell sarcoma of the subcutaneous tissues). No tumours of the internal organs were observed. The survival of all the mice in this experiment was poor due to a very high incidence of renal disease.

Cross-reactivity is also expected between ester derivatives and their parent alcohols, as the esters will be hydrolysed by esterases in the skin. Esters of important contact allergens that can be activated by hydrolysis in the skin are isoeugenol acetate, eugenyl acetate and geranyl acetate all of which are known to be used as fragrance ingredients.

**For bicyclic terpenes:**

**Acute toxicity:** The literature abounds with clinical reports of accidental and intentional acute poisoning with pinene-based turpentine.

Rat oral LD50 values are available for *alpha*-pinene, *beta*-pinene, camphene and turpentine oil and indicate these materials to be very low in oral acute toxicity with LD50 values in the range from 3388 mg/kg to greater than 5000 mg/kg. Rabbit dermal LD50 values similarly indicate very low toxicities with values greater than the limit doses of 2000 or 5000 mg/kg.

Acute inhalation toxicity has been measure in different animal species. The acute LC50 was reported to be 13,500 mg/m3 in rats, 13,500 mg/m3 in guinea pigs, and 9000 mg/m3 in mice . The acute inhalation LC50 of commercial grade turpentine in Wistar rats is reported to be in the range of 12,000-20,000 mg/m3 for 1 to 6 hour exposures and the LC50 for a 2-hour exposure in Swiss-Webster mice is 29,000 mg/m3 . Based on these results the acute oral, dermal, and inhalation toxicities of bicyclic terpene hydrocarbons is concluded to be low.

**Repeat dose toxicity:** A 28-day repeat dose study has been performed with camphene according to an OECD Guideline 407 in both sexes of Wistar rats. Animals of both sexes at the 1000 mg/kg bw/day dose exhibited vacuolization of hepatocytes and increase liver weights. Male rats also exhibited *alpha*-2-microglobulin-type nephrotoxicity at all dose levels.

Subsequent investigations have shown that the *alpha*-2-microglobulin nephropathy found in the F344/N male rat does not develop in mammals that do not express the hepatic form of *alpha*-2-microglobulin (e.g. other strains of rats, mice, dogs, humans). Therefore, the nephrotoxicity observed in the camphene study in male F344 rats is not relevant to the human health risk assessment. Based on liver toxicity, the NOAEL for this study is concluded to be 250 mg/kg bw/day

**Reproductive toxicity:** In the a-animal species study, no reproductive effects were observed when dose levels of up to 260 to 600 mg/kg bw of an essential oil predominantly composed of bicyclic terpene hydrocarbons (*alpha*-pinene, *beta*-pinene, and sabinene) was administered daily to mice, rats, or hamsters during gestation. When this data is combined with the fact that no adverse effects were observed to the reproductive organs in a 28-day study with camphene at dose levels up to 250 mg/kg bw/day, it is concluded that bicyclic terpene hydrocarbons including *alpha*-pinene and *beta*-pinene are not reproductive toxicants. Two ninety day inhalation studies have been performed for alpha-pinene in which a full complement of male and female sex organs and tissues were subjected to histopathological examination. Both studies reported no microscopic changes that could be associated with exposure to the test substance. Taking into account the lack of any effects to females in a earlier teratology study, the absence of any maternal or developmental effects in a reproductive/developmental study of a pinene-based oil and for a structurally related monoterpene hydrocarbon, myrcene, it can be concluded that the members of this category show no significant reproductive or developmental toxicity

**Developmental toxicity:** Based on the NOAELs for maternal and developmental toxicity in studies with camphene (250 and 1000 mg/kg bw/day) and a terpene hydrocarbon mixture containing *alpha*- and *beta*-pinene and camphene (688 mg/kg bw/day) , and the lack of any signs of maternal or developmental toxicity in a mice, rats, or hamsters given 260 to 600 mg/kg bw/day of a mixture composed primarily (>80%) of *alpha*- and *beta*-pinene and

## General Purpose Concrete Cleaner

	<p>sabinene, it is concluded that bicyclic terpene hydrocarbons are not maternal or developmental toxicants.</p> <p><b>Genotoxicity:</b></p> <p><b>In vitro:</b> <i>In vitro</i> genotoxicity assays available for <i>alpha</i>-pinene, <i>beta</i>-pinene and camphene demonstrate that these substances have a little, if any, genotoxic potential. In standard Ames assays of <i>alpha</i>-pinene, <i>beta</i>-pinene and camphene, <i>Salmonella typhimurium</i> strains TA97, TA98, TA100, TA1535, TA1537, and TA1538 provided no evidence of mutagenicity at any dose tested.</p> <p><b>In vivo:</b> Based on the lack of any evidence of genotoxicity in numerous <i>in vitro</i> assays with and without metabolic activation, it is unlikely that any of these bicyclic terpenes would exhibit a significant genotoxic potential <i>in vivo</i>.</p> <p>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.</p>
General Purpose Concrete Cleaner & SODIUM METASILICATE, PENTAHYDRATE & COCAMIDE DIETHANOLAMIDE. & DIETHANOLAMINE & SODIUM NITRATE & ISOTHIAZOLINONES, MIXED & SODIUM SULFATE & CITRAL & LEMON OIL	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>
General Purpose Concrete Cleaner & COCAMIDE DIETHANOLAMIDE. & ISOTHIAZOLINONES, MIXED & D-LIMONENE & CITRAL & LEMON OIL	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
General Purpose Concrete Cleaner & D-LIMONENE & CITRAL & LEMON OIL	<p>Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.</p> <p>Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.</p> <p>Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.</p> <p>Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.</p> <p>Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.</p> <p>Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease. Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.</p> <p><b>Hands:</b> Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe)</p>



## General Purpose Concrete Cleaner

chronic hand eczema may not be clear.

**Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

**Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.

**Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

**Pigmentary anomalies:** The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

**Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

**General/respiratory:** Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems.

In the case of prehapten, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers.

#### Prehapten

Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen.

Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxy radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context.

It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clinical studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.

#### Prohapten

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohapten.

In the case of prohapten, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask

## General Purpose Concrete Cleaner

	<p>hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitizers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.</p> <p><b>QSAR prediction:</b> The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.</p>
<b>SODIUM LAURYL ETHER SULFATE &amp; ISOTHIAZOLINONES, MIXED &amp; BRILLIANT BLUE R &amp; CITRAL &amp; LEMON OIL</b>	No significant acute toxicological data identified in literature search.
<b>SODIUM METASILICATE, PENTAHYDRATE &amp; ISOTHIAZOLINONES, MIXED</b>	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
<b>SODIUM METASILICATE, PENTAHYDRATE &amp; COCAMIDE DIETHANOLAMIDE. &amp; DIETHANOLAMINE &amp; ISOTHIAZOLINONES, MIXED</b>	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.
<b>COCAMIDE DIETHANOLAMIDE. &amp; LEMON OIL</b>	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
<b>COCAMIDE DIETHANOLAMIDE. &amp; DIETHANOLAMINE</b>	<p>for diethanolamine (DEA):</p> <p>In animal studies, DEA has low acute toxicity via the oral and dermal routes with moderate skin irritation and severe eye irritation. In subchronic toxicity testing conducted via the oral route in rats and mice, the main effects observed were increased organ weights and histopathology of the kidney and/or liver, with the majority of other tissue effects noted only at relatively high dosages. In subchronic studies conducted via the dermal route, skin irritation was noted as well as systemic effects similar to those observed in the oral studies. DEA has not been shown to be mutagenic or carcinogenic in rats; however, there is evidence of its carcinogenicity in mice.</p> <p><b>Subchronic toxicity:</b> The subchronic toxicity of DEA has been studied in F344 rats and B6C3F1 mice by exposure through drinking water or dermal administration, in 2 week and 13 week studies.</p> <p>Target organs for toxicity included blood, kidney, brain and spinal cord, seminiferous tubules and dermal application site in rats and liver, kidney, heart, salivary gland and dermal application site in mice. Effects on seminiferous tubules were accompanied by reductions in sperm count and reduced sperm motility. Hematological evaluations indicated normochromic, microcytic anemia in the dermal study in male rats (NOEL = 32 mg/kg) and females (LOEL = 32 mg/kg). Anemia was also observed in rats in the drinking water study with a LOEL of 14 mg/kg/d in females and a LOEL of 48 mg/kg/d in males for altered hematological parameters. These findings were similar to those observed in the 2 week studies, but the magnitude of the changes was greater in the 13 week studies. Hematological parameters were normal in controls. No associated histopathological changes were noted in femoral bone marrow. Haematological parameters were not evaluated in mice.</p> <p><b>Developmental toxicity:</b> In a developmental toxicity study conducted via the oral route, effects of concern were observed only in the presence of maternal toxicity. In a developmental toxicity study conducted via the dermal route using two species of mammals, developmental toxicity was observed only in one species and only at doses causing significant maternal toxicity. Metabolically, DEA is excreted largely unchanged in the urine.</p> <p><b>Carcinogenicity:</b> A two-year dermal cancer study bioassay results on DEA and three fatty acid condensates of DEA indicated that liver tumours occurred in male and female mice exposed to DEA and two of the condensates. In addition kidney tumours occurred in male mice exposed to DEA and one of the condensates. Compelling evidence suggested that the toxicity observed in mice and rats treated with the DEA condensates was associated with free DEA and not with other components of the condensates. A weight of evidence analysis of data relevant to the assessment of the liver and kidney tumours in mice resulted in the conclusion that these tumours are not relevant to humans under the expected conditions of exposure and that liver and kidney toxicity should be evaluated on a threshold basis. This conclusion is based on the following:</p> <ul style="list-style-type: none"> <li>▸ DEA is not genotoxic</li> <li>▸ tumour development occurred at doses also associated with chronic hyperplasia</li> </ul>

## General Purpose Concrete Cleaner

- ▶ there was no dose-related increase in malignancy, multiplicity of tumours or decrease in latency period
- ▶ tumours occurred late in life
- ▶ tumour response was species-specific (only mice were affected, not rats)
- ▶ tumour response was sex-specific (only male mice were affected, not females)
- ▶ tumour development was site-specific, with only liver and kidney affected, both sites of DEA accumulation;
- ▶ there was no tumour response in skin, despite evidence of chronic dermal toxicity
- ▶ there is a plausible mechanism, supported by various data, to explain the renal toxicity of DEA
- ▶ data support threshold mechanisms of renal carcinogenesis for a number of non-genotoxic chemicals
- ▶ the exposure regime used in the mouse study (*i.e.*, lifetime continuous exposure to DEA in ethanol vehicle at doses causing chronic dermal toxicity) is not relevant to human exposure (exposure through cosmetic vehicles with daily removal, under non-irritating conditions).

In considering the aggregate data on a DEA basis from the four studies using DEA and related condensates, the NOEL for kidney toxicity was 19 mg/kg/d, which resulted from a dose of 100 mg/kg/d of cocamide DEA containing 19% free DEA.

**Anaemia:** Rats exposed to DEA condensates developed anaemia. This was considered to be of to be relevant for humans since anaemia in rodents and humans share common etiologies. The proposed mechanism by which DEA could cause anemia involves disruption of phospholipid metabolism leading to membrane perturbation and functional change to erythrocytes. Some doubt about the relevance of the findings arises because ethanol was used as the vehicle in the dermal studies, and ethanol is known to cause anaemia in rodents through a mechanism involving membrane disruption. The possibility of a synergistic or additive role for DEA and ethanol in combination cannot be ruled out.

In considering the aggregate data on a DEA basis from the four 13-week dermal studies using DEA and related condensates, the NOEL for microcytic anemia was 9.5 mg/kg/d, which resulted from a dose of 50 mg/kg/d of cocamide DEA containing 19% free DEA.

The NOELs for mice and rats derived in this hazard assessment were as follows:

Anaemia in rats: 9.5 mg/kg/d (based on microcytic anemia)

Organ toxicity in mice: 2.2 mg/kg/d (based on liver toxicity)

In extrapolating among species for the purposes of risk assessment, the prime consideration with respect to dermally applied DEA was differential dermal absorption. Evidence indicates that dermal penetration of

DEA is greatest in mice and lower in rats and humans. Interspecies extrapolation was accomplished in this assessment by converting applied doses to bioavailable doses (*i.e.*, internal doses) using dermal bioavailability determined in studies with rats and mice *in vivo*, so as to be able to compare these with internal doses expected to be experienced by humans through use of personal care products.

Based on measured bioavailability in mice and rats, the bioavailable NOELs corresponding to the foregoing were:

Anaemia in rats: 0.8 mg/kg/d (based on microcytic anemia)

Organ toxicity in mice: 0.55 mg/kg/d (based on liver toxicity)

**Kidney toxicity:** Effects on the kidney were observed in rats treated with DEA in drinking water or by dermal exposure after as little as 2 weeks of exposure. Effects included renal tubule hyperplasia, renal tubular epithelial necrosis, renal tubule mineralization and increased relative organ weight. Similar changes were observed after 13 weeks of exposure of rats to DEA in drinking water and by dermal administration. The NOEL in male rats was 250 mg/kg/d in the dermal study, while in female rats renal tubule mineralisation was observed at the lowest dose of 32 mg/kg/d. After 2 years of dermal exposure there were no histopathological changes in the kidneys of male rats given doses of up to 64 mg/kg/d. In females, there were no significant increases in the incidences of renal tubule epithelial necrosis, hyperplasia or mineralisation as was observed after 13 weeks of exposure, however, there was an increase in the severity and incidence of nephropathy. This was the result of a treatment-related exacerbation of a previously existing lesion, since the incidence in controls was 80%, increasing to 94-96% in treated groups. There was no significant increase in the incidence of kidney tumours in rats treated with DEA or any of the condensates in 2-year dermal studies.

**Liver toxicity:** Effects on liver, including increases in relative organ weight and histopathological changes were observed in male and female mice in the 2 week drinking water study with DEA. Increases in liver weight were observed in the two week dermal study, but were not associated with histopathological changes. After 13 weeks of exposure, relative liver weights were increased compared to controls in male and female rats, with no associated histopathology. There is some doubt about whether these changes in liver weights were of toxicological significance, since there was no associated histopathology, the dose-response was not consistent and there were no effects on liver in the 2 year study in rats.

In the study with coconut diethanolamide (CDEA) (100 and 200 mg/kg/d) in which 19% of the applied dose was DEA, there were no liver effects in rats after 13 weeks or 2 years of dermal exposure. No liver toxicity in rats was observed in the 2 year dermal studies of lauramide or oleamide DEA

**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

## D-LIMONENE &amp; LEMON OIL

d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine.

Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes.

Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs.

Renal tumours induced by limonene in male rats is thought to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify a NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.

Monomethyltin chloride, thioglycolate esters, and tall oil ester reaction product:

Monomethyltin trichloride (MMTC, CAS RN: 993-16-8), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA), CAS RN: 57583-34-3), monomethyltin tris[isooctylmercaptoacetate (MMT (IOTG), CAS RN: 54849-38-6) and methyltin

## General Purpose Concrete Cleaner

reverse ester tallate reaction product (TERP, CAS RNs: 201687-58-3, 201687-57-2, 68442-12-6, 151436-98-5) are considered one category of compounds for mammalian studies via the oral route. The justification for this category is based on structural similarities and the demonstrated rapid conversion of all of the esters to the MMTC when placed in simulated mammalian gastric contents [0.07M HCl] under physiological conditions. For the MMT(EHTG) >90% conversion to MMTC occurred within 0.5 hours. For TERP, 68% of the monomethyltin portion of the compound was converted to MMTC within 1 hour. Thus, MMTC is the appropriate surrogate for mammalian toxicology studies via the oral route.

TERP is a reaction product of MMTC and dimethyltin dichloride (DMTC), Na<sub>2</sub>S, and tall oil fatty acid [a mixture of carboxylic acids, predominantly C-18]. The reaction product is a mixture of carboxylic esters and includes short oligomers of mono/dimethyltins bridged by sulfide groups. Although the tall oil component of TERP is not structurally similar to EHTG, TERP's conversion to MMTC justifies its inclusion. While the contribution of the various ligands to the overall toxicity may vary, the contribution is expected to be small relative to that of the MMTC. Further, the EHTG ligand from MMT(EHTG) is likely to be more toxic than the oleic or linoleic acid from TERP so inclusion of TERP in the category is a rather conservative approach. The other possible degradate of tall oil and EHTG is 2-mercaptoethanol (2-ME), and it is common to both ligands.

Data for MMT(EHTG) and MMT(IOTG) are used interchangeably because they are isomers differing only slightly in the structure of the C-8 alcohol of the mercaptoester ligand. In addition, the breakdown products of MMT(EHTG) and MMT(IOTG) are the thioglycolate esters (EHTG and IOTG), which have the common degradates, thioglycolic acid and C-8 alcohols (either 2-ethylhexanol or isooctanol). EHTG and IOTG also have similar physicochemical and toxicological properties.

The chemistry of the alkyl organotin has been well studied. For organotins, like MMT(EHTG), the alkyl groups are strongly bound to tin and remain bound to tin under most reaction conditions. However, other ligands, such as carboxylates or sulfur based ligands (EHTG), are more labile and are readily replaced under mild reaction conditions. To assess the reactivity of MMT(EHTG) under physiological conditions simulating the mammalian stomach, an in-vitro hydrolysis test was performed. This in vitro test provides chemical information that strongly suggests both the probable in vivo metabolic pathway and the toxicokinetics of the MMT(EHTG) substance. This result verifies that under physiological conditions MMT(EHTG) is rapidly and essentially completely converted to the corresponding monomethyltin chloride, MMTC.

#### Acute toxicity:

The majority of toxicology studies were conducted with commercial mixtures having high monoalkyltin to dialkyltin ratios. Gastric hydrolysis studies were conducted with TERP and MMT(EHTG) in which simulated gastric fluid [0.07M HCl under physiological conditions] converted these substances to methyltin chloride and the respective organic acids. Based on data for DMTC and DMT esters the dermal penetration of MMTC and its esters is expected to be low.

#### Oral:

Acute oral LD<sub>50</sub> values for MMTC, MMT(EHTG), MMT(IOTG), and TERP indicated low to moderate toxicity; the most reliable data place the LD<sub>50</sub>s in the range of 1000 mg/kg.

The acute oral LD<sub>50</sub> of MMT(2-EHMA) was 880 mg/kg in rats. Clinical observations included depression, comatose, piloerection, eye squinting, hunched posture, laboured breathing, ataxia, faecal/urine stains, and masticatory movement. No gross pathological changes were reported in surviving animals.

#### Dermal

Acute dermal LD<sub>50</sub> values were =1000 mg/kg bw, and inhalation LC<sub>50</sub> was >200 mg/L. MMTC was corrosive to skin and assumed corrosive to eyes.

The acute dermal LD<sub>50</sub> of MMT(2-EHMA) in rabbits was 1000 (460 to 2020) mg/kg for females and 2150 (1000 to 4620) mg/kg for males. There were no deaths at 215 and 464 mg/kg, 0/2 males and 1/2 females died at 1000 mg/kg and 1/2 males and 2/2 females died at 2150 mg/kg. All animals died at 4640 and 10 000 mg/kg. A variety of clinical abnormalities were observed and disappeared in surviving animals by the end of the exposure period. Clinical signs included death, uncoordinated movements, shaking, and hypersensitivity to external stimuli.

Gross necropsy results for animals that died during the study included irritated intestines; blanched stomach; reddened lungs; pale or congested kidneys; and oral, ocular and/or nasal discharges

#### Inhalation:

The acute inhalation LC<sub>50</sub> of MMT(2-EHMA) was 240 mg/L.

The study reported an acute inhalation LC<sub>50</sub> of 240 (212 to 271) mg/L in a 1-hr aerosol exposure to male and female rats. The mortality rate was 2/10, 6/10, 9/10 and 10/10 animals at dose levels of 200, 250, 300 and 250 mg/L/hr, respectively. Gross findings included blood in lungs, dark spleen, pale kidneys, fluid in the chest cavity, and heart failure. The slope of the dose-response curve was 1.22 (1.04 to 1.43).

#### Irritation:

MMT(IOTG)/(EHTG) are irritating to skin, but not to eyes.

#### Sensitisation:

No data on sensitization are available on MMT(EHTG)/(IOTG), but the hydrolysis products EHTG or IOTG are sensitizers. No primary irritation data were available for TERP, but it was a sensitizer in the mouse Local Lymph Node Assay.

Topical application with 5, 25 and 50 % v/v MMT(2-EHMA) elicited a stimulation index (SI) of 2.13, 7.25 and 9.05, respectively in a local lymph node assay (OECD 429), thus the material is a sensitizer.

#### Repeat dose toxicity:

There are no repeated-dose studies for the category members via the dermal or inhalation routes.

In a 90-day repeated dose oral study of MMTC, treatment-related changes were limited to the high dose group (750 ppm in diet; 50 mg/kg bw/d with some gender-related variation). Organ weight changes (adrenal, kidney, thymus, spleen, brain, epididymides), haematology, clinical chemistry, and urinalysis changes were noted, but histopathology only confirmed effects in the thymus and brain. The critical toxic effects were neurotoxicity and thymic atrophy. Both sexes had decreased cortex/medulla ratios in the thymus. In the brain there was loss of perikarya of neuronal cells in the pyramidal layer of the Hippocampus CA1/2 in both sexes, and in males there was loss of perikarya in the piriform cortex. The NOAEL was 150 ppm (10 mg/kg bw/d). Another 90-day dietary study using MMTC showed increased relative kidney weights and slight to moderate epithelial hyperplasia of the bladder in females at the lowest dose (NOAEL <20 ppm in diet [ $<1.3.6$  mg/kg bw/d]) and additional effects including increased relative thymus weights in females and urinalysis results in both sexes at higher doses.

A 90-day dietary study with dose levels of 30, 100, 300, and 1000 ppm TERP in the diet resulted in slightly decreased food intake, body and organ weight changes, and decreased specific gravity of the urine at the highest dose. The NOAEL was 300 ppm in diet (equivalent to 15 mg/kg bw/d). A 28-day gavage study using TERP showed changes in clinical chemistry and slight



## General Purpose Concrete Cleaner

differences in haematology at 150 mg/kg bw/d and higher. The NOAEL was 50 mg/kg bw/d.

The effects of MMT(IOTG) were evaluated in a 90-day dietary study using doses of 100, 500, and 1500 ppm (decreased from 2500 ppm) in the diet. Based on clinical chemistry effects at 500 ppm and other effects at higher doses, the NOAEL was 100 ppm in diet (approximately 6-21 mg/kg bw/d).

**Neurotoxicity:**

In a guideline 90-day subchronic dietary study conducted in Wistar rats, effects occurred at the high dose of 750 ppm MMT(2-EHMA), (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), which consisted of changes in neurobehavioral parameters and associated brain histopathology. The NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females).

**Immunotoxicity:**

Immune function was assessed in male Sprague-Dawley rats exposed to the mixture of organotin compounds used in PVC pipe production. Adult male rats were given drinking water for 28 d containing a mixture of dibutyltin dichloride (DBTC), dimethyltin dichloride (DMTC), monobutyltin trichloride (MBT), and monomethyltin trichloride (MMT) in a 2:2:1:1 ratio, respectively, at 3 different concentrations (5:5:2.5:2.5, 10:10:5:5, or 20:20:10:10 mg organotin/L). Rats were also exposed to MMT alone (20 or 40 mg MMT/L) or plain water as a control. Delayed-type hypersensitivity, antibody synthesis, and natural killer cell cytotoxicity were evaluated in separate endpoint groups immediately after exposure ended.

The evaluated immune functions were not affected by the mixture or by MMT alone. The data suggest that immunotoxicity is unlikely to result from the concentration of organotin compounds present in drinking water delivered via PVC pipes, as the concentrations used were several orders of magnitude higher than those expected to leach from PVC pipes.

**Genotoxicity:**

In a guideline 90-day subchronic dietary study in rats, with MMT(2-EHMA), based on the changes in neurobehavioral parameters and associated brain histopathology that occurred at the high dose of 750 ppm (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), as well as changes in haematology, clinical chemistry, urinalysis, organ weights, and pathology of the thymus at the same dose, the NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females).

The monomethyltin compounds as a class are not mutagenic in the Ames test. TERP was positive in a human lymphocyte assay. MMTC was equivocal for induction of micronucleated polychromatic erythrocytes (MPEs) in an in vivo rat micronucleus test (OECD 474). In this study a statistically significant increase in MPE was observed only at 24 h and not at 48 h after treatment and there was no dose-response. Based on these observations the overall conclusion is that MMTC does not have genotoxic potential.

From the results obtained in a micronucleus test with MMT(2-EHMA), it was demonstrated that the substance was weakly genotoxic to bone marrow cells of rats and that the substance has the potential to induce damage to the mitotic spindle apparatus of the bone marrow target cells.

**Carcinogenicity:**

In a limited carcinogenicity study, MMT(EHTG) produced no compound-related macroscopic or microscopic changes in rats fed 100 ppm in the diet for two years.

**Toxicity to reproduction:**

In the reproductive satellite portion of the 90-day study using MMTC (with dose levels of 30, 150, and 750 ppm in the diet), post-implantation loss, decreased litter size and increased neonatal mortality occurred at 750 ppm (26-46 mg/kg bw/d for females). Maternal gestational body weights were transiently suppressed and other maternal toxicity was inferred from the repeated dose results at this dose. There were no malformations observed at any dose. The NOAEL for maternal toxicity, and reproductive, and foetotoxic effects was 150 ppm in the diet (6-12 mg/kg bw/d).

SIDS Initial Assessment Profile (SIAM 23 2006)

ECHA Registration Dossier for MMT(2-EHMA) (ethylhexyl 10-ethyl-4-[[2-[(2-ethylhexyl)oxy]-2-oxoethyl]thio]-4-methyl-7-oxo-8-oxa-3,5-dithia-4-stannatetradecanoate)

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

General Purpose Concrete Cleaner	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
2-Butoxyethanol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-700mg/L	2
	EC50	48	Crustacea	ca. 1-800mg/L	2

Continued...

## General Purpose Concrete Cleaner

	EC50	72	Algae or other aquatic plants	1-840mg/L	2
	NOEC	24	Crustacea	>1-mg/L	2
sodium lauryl ether sulfate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	NOEC	48	Fish	0.26mg/L	5
sodium metasilicate, pentahydrate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	2-320mg/L	2
	EC50	48	Crustacea	1-700mg/L	2
	EC50	72	Algae or other aquatic plants	207mg/L	2
cocamide diethanolamide.	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	2.4mg/L	2
	EC50	48	Crustacea	ca.3.2mg/L	2
	NOEC	504	Crustacea	0.07mg/L	2
diethanolamine	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-480mg/L	2
	EC50	48	Crustacea	=28.8mg/L	1
	EC50	96	Algae or other aquatic plants	=2.1-2.3mg/L	1
	EC10	72	Algae or other aquatic plants	0.7mg/L	2
	NOEC	72	Algae or other aquatic plants	0.6mg/L	2
sodium nitrate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-559mg/L	2
	EC50	48	Crustacea	3-581mg/L	2
	EC50	96	Algae or other aquatic plants	1181.887mg/L	3
	NOEC	2880	Fish	1.6mg/L	4
isothiazolinones, mixed	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.129mg/L	2
	EC50	48	Crustacea	0.007mg/L	2
	EC50	72	Algae or other aquatic plants	0.0063mg/L	2
	NOEC	48	Algae or other aquatic plants	0.00049mg/L	2
sodium sulfate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	48	Crustacea	2-564mg/L	2
	EC50	96	Algae or other aquatic plants	1900mg/L	4
	NOEC	168	Fish	<220mg/L	4
Brilliant Blue R	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
d-limonene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.199mg/L	3
	EC50	48	Crustacea	0.307mg/L	2
	EC50	96	Algae or other aquatic plants	0.212mg/L	3
	NOEC	504	Crustacea	0.05mg/L	2
citral	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	4.472mg/L	3
	EC50	48	Crustacea	6.8mg/L	2
	EC50	96	Algae or other aquatic plants	3.938mg/L	3
	EC10	96	Algae or other aquatic plants	=1.9mg/L	1
	NOEC	96	Fish	4.6mg/L	1

Continued...

## General Purpose Concrete Cleaner

lemon oil	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	5.65mg/L	2
	EC50	48	Crustacea	1.1mg/L	2
	EC50	72	Algae or other aquatic plants	8mg/L	2
	NOEC	48	Crustacea	3.8mg/L	2
<b>Legend:</b>	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 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				

Harmful to aquatic organisms.

**DO NOT** discharge into sewer or waterways.

## Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
2-Butoxyethanol	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
diethanolamine	LOW (Half-life = 14 days)	LOW (Half-life = 0.3 days)
sodium nitrate	LOW	LOW
sodium sulfate	HIGH	HIGH
d-limonene	HIGH	HIGH
citral	LOW	LOW

## Bioaccumulative potential

Ingredient	Bioaccumulation
2-Butoxyethanol	LOW (BCF = 2.51)
diethanolamine	LOW (BCF = 1)
sodium nitrate	LOW (LogKOW = 0.209)
sodium sulfate	LOW (LogKOW = -2.2002)
d-limonene	HIGH (LogKOW = 4.8275)
citral	LOW (LogKOW = 3.4453)

## Mobility in soil

Ingredient	Mobility
2-Butoxyethanol	HIGH (KOC = 1)
diethanolamine	HIGH (KOC = 1)
sodium nitrate	LOW (KOC = 14.3)
sodium sulfate	LOW (KOC = 6.124)
d-limonene	LOW (KOC = 1324)
citral	LOW (KOC = 147.7)

## SECTION 13 DISPOSAL CONSIDERATIONS

## Waste treatment methods

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>Containers may still present a chemical hazard/ danger when empty.</li> <li>Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>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.</li> <li>Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> <li>Recycle wherever possible.</li> <li>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.</li> <li>Treat and neutralise at an approved treatment plant.</li> <li>Treatment should involve: Neutralisation with suitable dilute acid followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible</li> </ul>
-------------------------------------	---

Continued...

## General Purpose Concrete Cleaner

material).

▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

## SECTION 14 TRANSPORT INFORMATION

## Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

Not Applicable

## SECTION 15 REGULATORY INFORMATION

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

## 2-BUTOXYETHANOL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

## SODIUM LAURYL ETHER SULFATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

## SODIUM METASILICATE, PENTAHYDRATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

## COCAMIDE DIETHANOLAMIDE. IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B : Possibly carcinogenic to humans

## DIETHANOLAMINE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

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

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B : Possibly carcinogenic to humans

## SODIUM NITRATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

## ISOTHIAZOLINONES, MIXED IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

## SODIUM SULFATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

## BRILLIANT BLUE R IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

## D-LIMONENE IS FOUND ON THE FOLLOWING REGULATORY LISTS

## General Purpose Concrete Cleaner

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

#### CITRAL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

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

#### LEMON OIL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

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

### National Inventory Status

National Inventory	Status
Australia - AICS	No (isothiazolinones, mixed)
Canada - DSL	Yes
Canada - NDSL	No (2-Butoxyethanol; sodium lauryl ether sulfate; sodium metasilicate, pentahydrate; cocamide diethanolamide.; diethanolamine; sodium nitrate; isothiazolinones, mixed; sodium sulfate; Brilliant Blue R; d-limonene; citral; lemon oil)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (isothiazolinones, mixed; lemon oil)
Japan - ENCS	No (isothiazolinones, mixed; lemon oil)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (isothiazolinones, mixed)
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium lauryl ether sulfate; isothiazolinones, mixed; Brilliant Blue R; lemon oil)
Vietnam - NCI	Yes
Russia - ARIPS	No (Brilliant Blue R; lemon oil)
<b>Legend:</b>	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)</p>

### SECTION 16 OTHER INFORMATION

<b>Revision Date</b>	22/05/2020
<b>Initial Date</b>	22/05/2020

#### Other information

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

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

#### Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average  
 PC—STEL: Permissible Concentration-Short Term Exposure Limit  
 IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit.  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 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

Continued...

## General Purpose Concrete Cleaner

BCF: BioConcentration Factors

BEI: Biological Exposure Index

Powered by AuthorITe, from Chemwatch.