



# HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

JTC Import Export Pty Ltd

Chemwatch: 5390-42

Version No: 2.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

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

### Product Identifier

Product name	HomeBright Foaming Super Oxygen Toilet Bowl Cleaner
Synonyms	Product Code: 43056
Proper shipping name	AEROSOLS
Other means of identification	Not Available

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

Relevant identified uses	Toilet/urinal care product- Aerosol.
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### Details of the supplier of the safety data sheet

Registered company name	JTC Import Export Pty Ltd
Address	98 South Park Drive Dandenong South VIC 3175 Australia
Telephone	+61 3 9532 5100
Fax	+61 3 9532 6102
Website	<a href="http://www.jtcimportexport.com.au">http://www.jtcimportexport.com.au</a>
Email	<a href="mailto:sales@jtcimportexport.com.au">sales@jtcimportexport.com.au</a>

### Emergency telephone number

Association / Organisation	JTC Import Export Pty Ltd
Emergency telephone numbers	+61 3 9532 5100 (Mon-Thurs 8.30am to 5.30pm; Friday 8.30am to 3pm)
Other emergency telephone numbers	Not Available

## SECTION 2 HAZARDS IDENTIFICATION

### Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Germ cell mutagenicity Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Acute Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

### Label elements

Hazard pictogram(s)	
SIGNAL WORD	DANGER

### Hazard statement(s)

H315	Causes skin irritation.
H318	Causes serious eye damage.
H341	Suspected of causing genetic defects.
H335	May cause respiratory irritation.
H402	Harmful to aquatic life.
AUH044	Risk of explosion if heated under confinement.

### Precautionary statement(s) Prevention

Continued...

## HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.

## Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P310	Immediately call a POISON CENTER or doctor/physician.
P321	Specific treatment (see advice on this label).
P362	Take off contaminated clothing and wash before reuse.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.

## Precautionary statement(s) Storage

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

## Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation
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## SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

## Substances

See section below for composition of Mixtures

## Mixtures

CAS No	%[weight]	Name
15630-89-4	1-5	<u>sodium percarbonate</u>
111-76-2	1-5	<u>ethylene glycol monobutyl ether</u>
9002-92-0	0.1-1	<u>lauryl alcohol, ethoxylated</u>
1336-21-6	0.1-1	<u>ammonium hydroxide</u>
7632-00-0	0.1-1	<u>sodium nitrite</u>
68476-85-7.	2-10	<u>hydrocarbon propellant</u>

## SECTION 4 FIRST AID MEASURES

## Description of first aid measures

Eye Contact	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh 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>▶ 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 solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Remove any adhering solids with industrial skin cleansing cream.</li> <li>▶ <b>DO NOT use solvents.</b></li> <li>▶ Seek medical attention in the event of irritation.</li> </ul>
Inhalation	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> <li>▶ Remove to fresh air.</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>▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, 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.</li> </ul>
Ingestion	<ul style="list-style-type: none"> <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>▶ Seek medical advice.</li> </ul>

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

Treat symptomatically.

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## HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

## SECTION 5 FIREFIGHTING MEASURES

## Extinguishing media

## SMALL FIRE:

- ▶ Water spray, dry chemical or CO2

## LARGE FIRE:

- ▶ Water spray or fog.

## Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent 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>
Fire/Explosion Hazard	<ul style="list-style-type: none"> <li>▶ Non combustible.</li> <li>▶ Not considered to be a significant fire risk.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ Aerosol cans may explode on exposure to naked flames.</li> <li>▶ Rupturing containers may rocket and scatter burning materials.</li> <li>▶ Hazards may not be restricted to pressure effects.</li> <li>▶ May emit acrid, poisonous or corrosive fumes.</li> <li>▶ Decomposes on heating and may emit toxic fumes of carbon monoxide (CO).</li> </ul> <p>Decomposition may produce toxic fumes of: Combustion products include: carbon dioxide (CO2) other pyrolysis products typical of burning organic material.</p>
HAZCHEM	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>▶ Wear protective clothing, impervious gloves and safety glasses.</li> <li>▶ Shut off all possible sources of ignition and increase ventilation.</li> <li>▶ Wipe up.</li> <li>▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.</li> <li>▶ Undamaged cans should be gathered and stowed safely.</li> </ul>
Major Spills	<ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water courses</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Water spray or fog may be used to disperse / absorb vapour.</li> <li>▶ Absorb or cover spill with sand, earth, inert materials or vermiculite.</li> <li>▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.</li> <li>▶ Undamaged cans should be gathered and stowed safely.</li> <li>▶ Collect residues and seal in labelled drums for disposal.</li> </ul>

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>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> </ul>
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## HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

- ▶ When handling, **DO NOT** eat, drink or smoke.
- ▶ **DO NOT** incinerate or puncture aerosol cans.
- ▶ **DO NOT** spray directly on humans, exposed food or food utensils.
- ▶ Avoid physical damage to containers.
- ▶ Always wash hands with soap and water after handling.
- ▶ Work clothes should be laundered separately.
- ▶ Use good occupational work practice.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

## Other information

- ▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can

## Conditions for safe storage, including any incompatibilities

## Suitable container

- ▶ Aerosol dispenser.
- ▶ Check that containers are clearly labelled.

## Storage incompatibility

- ▶ Avoid reaction with oxidising agents

## 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	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m <sup>3</sup>	242 mg/m <sup>3</sup> / 50 ppm	Not Available	Not Available
Australia Exposure Standards	hydrocarbon propellant	LPG (liquefied petroleum gas)	1000 ppm / 1800 mg/m <sup>3</sup>	Not Available	Not Available	Not Available

## EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
lauryl alcohol, ethoxylated	Brij-35; (alpha-Dodecyl-omega-hydroxypoly(oxyethylene))	2.9 mg/m <sup>3</sup>	31 mg/m <sup>3</sup>	200 mg/m <sup>3</sup>
ammonium hydroxide	Ammonium hydroxide	61 ppm	330 ppm	2,300 ppm
sodium nitrite	Sodium nitrite	6.4 mg/m <sup>3</sup>	71 mg/m <sup>3</sup>	240 mg/m <sup>3</sup>
hydrocarbon propellant	Liquefied petroleum gas; (L.P.G.)	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm

Ingredient	Original IDLH	Revised IDLH
sodium percarbonate	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available
lauryl alcohol, ethoxylated	Not Available	Not Available
ammonium hydroxide	Not Available	Not Available
sodium nitrite	Not Available	Not Available
hydrocarbon propellant	2,000 ppm	Not Available

## OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sodium percarbonate	E	≤ 0.01 mg/m <sup>3</sup>
lauryl alcohol, ethoxylated	E	≤ 0.1 ppm
ammonium hydroxide	E	≤ 0.1 ppm
sodium nitrite	E	≤ 0.01 mg/m <sup>3</sup>

## 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

NOTE K: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.1%w/w 1,3-butadiene (EINECS No 203-450-8). - European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

## Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.</p> <p>Provide adequate ventilation in warehouse or closed storage areas.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>
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## HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

	Type of Contaminant:	Speed:
	aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s
	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
Personal protection	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.	
		
Eye and face protection	No special equipment for minor exposure i.e. when handling small quantities. <b>OTHERWISE:</b> For potentially moderate or heavy exposures: <ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ <b>NOTE:</b> Contact lenses pose a special hazard; soft lenses may absorb irritants and <b>ALL</b> lenses concentrate them.</li> </ul>	
Skin protection	See Hand protection below	
Hands/feet protection	<ul style="list-style-type: none"> <li>▶ No special equipment needed when handling small quantities.</li> <li>▶ <b>OTHERWISE:</b></li> <li>▶ For potentially moderate exposures:</li> <li>▶ Wear general protective gloves, eg. light weight rubber gloves.</li> <li>▶ For potentially heavy exposures:</li> <li>▶ Wear chemical protective gloves, eg. PVC. and safety footwear.</li> </ul>	
Body protection	See Other protection below	
Other protection	No special equipment needed when handling small quantities. <b>OTHERWISE:</b> <ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eyewash unit.</li> <li>▶ Do not spray on hot surfaces.</li> </ul>	

## Recommended material(s)

GLOVE SELECTION INDEX

## Respiratory protection

Type KAX-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	KAX-AUS P2	-	KAX-PAPR-AUS / Class 1 P2
up to 50 x ES	-	KAX-AUS / Class 1 P2	-
up to 100 x ES	-	KAX-2 P2	KAX-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(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

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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:

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

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

## Information on basic physical and chemical properties

Appearance	Clear liquid with fresh odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	0.95
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	11	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

## SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> <li>▶ Elevated temperatures.</li> <li>▶ Presence of open flame.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

## HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

## SECTION 11 TOXICOLOGICAL INFORMATION

## Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. <b>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</b>
Ingestion	Not normally a hazard due to physical form of product.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. <b>WARNING: Aerosol containers may present pressure related hazards.</b>

HomeBright Foaming Super Oxygen Toilet Bowl Cleaner	TOXICITY	IRRITATION
	Inhalation (None) LC50: 150 mg/l (dust&mist)*[2]	Not Available
	Oral (None) LD50: 13357 mg/kg*[2]	
sodium percarbonate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup>	Not Available
	Oral (rat) LD50: 893 mg/kg <sup>[1]</sup>	
ethylene glycol monobutyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 100 mg SEVERE
	Inhalation (rat) LC50: 449.48655 mg/l/4h <sup>[2]</sup>	Eye (rabbit): 100 mg/24h-moderate
	Oral (rat) LD50: 250 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 500 mg, open; mild
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
lauryl alcohol, ethoxylated	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 0.75 mg/24h SEVERE
	Oral (rat) LD50: 1000 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 500 mg/24h mild
		Skin (rabbit): 75 mg/24h mild
ammonium hydroxide	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 1997.718 mg/l/4h <sup>[2]</sup>	Eye (rabbit): 0.25 mg SEVERE
	Oral (rat) LD50: 350 mg/kg <sup>[2]</sup>	Eye (rabbit): 1 mg/30s SEVERE
sodium nitrite	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 0.0055 mg/l/4h <sup>[2]</sup>	Eye (rabbit): 500 mg/24hr - mild
	Oral (rat) LD50: =85 mg/kg <sup>[2]</sup>	
hydrocarbon propellant	TOXICITY	IRRITATION
	Not Available	Not Available

## Legend:

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

## SODIUM PERCARBONATE

For sodium percarbonate:  
Sodium percarbonate is an inorganic, water soluble solid of relatively low molecular weight. Dermal absorption is assumed to be low due to the

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## HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

hydrophilic character and the ionic structure of the substance. When sodium percarbonate is getting into contact with body fluids it will dissociate into hydrogen peroxide, carbonate and sodium ions which are all naturally present in the human body. For hydrogen peroxide a high degradation capacity is present in the blood and tissues, making it unlikely that hydrogen peroxide is systemically available. As carbonate is a part of the natural buffer systems in the organism it is unlikely that it is absorbed through sodium percarbonate exposure in amounts that would disturb the normal acid/base balance of the body. Similarly for sodium percarbonate exposure is not expected to contribute significantly to the sodium load of the body. The mode

of action is characterized by the local irritation potential in particular to mucous membranes. No systemic effects are anticipated because it is unlikely that the substance is systemically available.

Acute oral LD50 values ranged between 1034 and 2200 mg/kg bw, while the acute dermal LD50 was > 2000 mg/kg bw. The existing animal data on acute toxicity show that sodium percarbonate has a local effect and that systemic effects are not to be expected. In animal tests a slight irritating effect on the skin was reported for solid sodium percarbonate and it was highly irritating to the rabbit eye (not rinsed). Sodium percarbonate did not have sensitising properties in a test with guinea pigs. The acute studies indicate that most of the acute and local effects can be explained by the release of hydrogen peroxide.

Although a repeated dose study is not available for sodium percarbonate, effects can be predicted based on the release of hydrogen peroxide, carbonate and sodium. As it is expected that repeated dose toxicity of sodium percarbonate will mainly be mediated by hydrogen peroxide, no observed adverse effect levels can be defined on the basis of its hydrogen peroxide content. Based on the 90-day drinking water study according to OECD guidelines and GLP with hydrogen peroxide and catalase deficient mice, the predicted NOAEL of sodium percarbonate would be 308 ppm (81 to 115 mg/kg bw/day for males and females, respectively).

Data on the mutagenicity will be similar to those of hydrogen peroxide due to the release of hydrogen peroxide in aqueous media. The available studies on hydrogen peroxide, most of them, in particular the *in vivo* studies, were performed according to OECD guidelines and GLP, are not in support of significant genotoxicity/mutagenicity under *in vivo* conditions. Therefore sodium percarbonate is also unlikely to have any *in vivo* genotoxic potential.

Carcinogenicity studies with animals and sodium percarbonate are not available. The only component that could give rise to some concerns with regard to this endpoint is hydrogen peroxide. A local carcinogenic effect was observed in the duodenum of a catalase-deficient mouse strain administered 0.4 % hydrogen peroxide in drinking water. Although an underlying genotoxic mechanism cannot be excluded, the weight of evidence at this time does not suggest that the carcinogenic properties of hydrogen peroxide should be regarded as practically significant. Neither an animal study on toxicity to reproduction nor a study on developmental toxicity is available for sodium percarbonate. A developmental toxicity study with sodium carbonate, which was well documented and meets basic scientific principles, revealed no substance related foetotoxic, embryotoxic or teratogenic effects. From the nature of the substance it is to be anticipated that neither sodium percarbonate nor hydrogen peroxide and sodium carbonate will be systemically available under human exposure conditions and are thus unlikely to reach the gonads and the developing embryo or fetus. Therefore the substance is unlikely to have any relevant potential for toxicity to reproduction or developmental toxicity.

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. \*\* ASCC (NZ) SDS

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.

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

**Acute Toxicity:** Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

**Repeat dose toxicity:** The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.

**Mutagenicity:** In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

**Carcinogenicity:** In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity

**Reproductive and developmental toxicity.** The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to

ETHYLENE GLYCOL  
MONOBUTYL ETHER



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endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO<sub>2</sub>, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO<sub>2</sub>, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

**Respiratory Effects.** Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

**Cardiovascular Effects.** Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

**Gastrointestinal Effects.** Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

**Musculoskeletal Effects.** Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

**Hepatic Effects.** Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

**Renal Effects.** Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

**Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

**Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion. Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

**Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

**Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

**Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

**Genotoxic Effects:** Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

LAURYL ALCOHOL,  
ETHOXYLATED

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

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-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

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combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules ( $n = 195$  to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used.

Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology <http://doi.org/10.5487/TR.2015.31.2.105>

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

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

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

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-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.

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

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

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

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

>20 EO is not classified (CESIO 2000)

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

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

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO<sub>2</sub>). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO<sub>2</sub>). The metabolism of C12 AE yields PEG, carboxylic acids, and CO<sub>2</sub> as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

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

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

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

**Skin absorption:** Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm<sup>2</sup>/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm<sup>2</sup>/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

**Metabolism:** The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via

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

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

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

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

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

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

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

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

haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

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

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

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

The material may 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.

## SODIUM NITRITE

Tumorigenic - Carcinogenic by RTECS criteria.

Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells *in vivo*. Such findings are often supported by positive results from *in vitro* mutagenicity studies.

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

## HYDROCARBON PROPELLANT

for Petroleum Hydrocarbon Gases:

In many cases, there is more than one potentially toxic constituent in a refinery gas. In those cases, the constituent that is most toxic for a particular endpoint in an individual refinery stream is used to characterize the endpoint hazard for that stream. The hazard potential for each mammalian endpoint for each of the petroleum hydrocarbon gases is dependent upon each petroleum hydrocarbon gas constituent endpoint toxicity values (LC50, LOAEL, etc.) and the relative concentration of the constituent present in that gas. It should also be noted that for an individual petroleum hydrocarbon gas, the constituent characterizing toxicity may be different for different mammalian endpoints, again, being dependent upon the concentration of the different constituents in each, distinct petroleum hydrocarbon gas.

All Hydrocarbon Gases Category members contain primarily hydrocarbons (i.e., alkanes and alkenes) and occasionally asphyxiant gases like hydrogen. The inorganic components of the petroleum hydrocarbon gases are less toxic than the C1 - C4 and C5 - C6 hydrocarbon components to both mammalian and aquatic organisms. Unlike other petroleum product categories (e.g. gasoline, diesel fuel, lubricating oils, etc.), the inorganic and hydrocarbon constituents of hydrocarbon gases can be evaluated for hazard individually to then predict the screening level hazard of the Category members

**Acute toxicity:** No acute toxicity LC50 values have been derived for the C1 -C4 and C5- C6 hydrocarbon (HC) fractions because no mortality was observed at the highest exposure levels tested (~ 5 mg/l) for these petroleum hydrocarbon gas constituents. The order of acute toxicity of petroleum hydrocarbon gas constituents from most to least toxic is:

C5-C6 HCs (LC50 > 1063 ppm) > C1-C4 HCs (LC50 > 10,000 ppm) > benzene (LC50 = 13,700 ppm) > butadiene (LC50 = 129,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).

**Repeat dose toxicity:** With the exception of the asphyxiant gases, repeated dose toxicity has been observed in individual selected petroleum hydrocarbon gas constituents. Based upon LOAEL values, the order of order of repeated-dose toxicity of these constituents from most toxic to the least toxic is:

Benzene (LOAEL >=10 ppm) >C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) > C5-C6 HCs (LOAEL = 6,625 ppm) > butadiene (LOAEL = 8,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).

**Genotoxicity:**

***In vitro*:** The majority of the Petroleum Hydrocarbon Gases Category components are negative for *in vitro* genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian *in vitro* test systems.

***In vivo*:** The majority of the Petroleum Hydrocarbon Gases Category components are negative for *in vivo* genotoxicity. The exceptions are benzene and 1,3-butadiene, which are genotoxic in *in vivo* test systems

**Developmental toxicity:** Developmental effects were induced by two of the petroleum hydrocarbon gas constituents, benzene and the C5 -C6 hydrocarbon fraction. No developmental toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas

## HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

	<p>constituents tested for this effect. The asphyxiant gases have not been tested for developmental toxicity. Based on LOAEL and NOAEL values, the order of acute toxicity of these constituents from most to least toxic is:</p> <p>Benzene (LOAEL = 20 ppm) &gt; butadiene (NOAEL &gt;=1,000 ppm) &gt; C5-C6 HCs (LOAEL = 3,463 ppm) &gt; C1-C4 HCs (NOAEL &gt;=5,000 ppm; assumed to be 100% 2-butene) &gt; asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p><b>Reproductive toxicity:</b> Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the C1-C4 hydrocarbon fraction). No reproductive toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for reproductive toxicity. Based on LOAEL and NOAEL values, the order of reproductive toxicity of these constituents from most to least toxic is:</p> <p>Benzene (LOAEL = 300 ppm) &gt; butadiene (NOAEL &gt;=6,000 ppm) &gt; C5-C6 HCs (NOAEL &gt;=6,521 ppm) &gt; C1-C4 HCs (LOAEL = 9,000 ppm; assumed to be 100% isobutane) &gt; asphyxiant gases (hydrogen, carbon dioxide, nitrogen)</p>
<b>SODIUM PERCARBONATE &amp; HYDROCARBON PROPELLANT</b>	No significant acute toxicological data identified in literature search.
<b>ETHYLENE GLYCOL MONOBUTYL ETHER &amp; LAURYL ALCOHOL, ETHOXYLATED &amp; AMMONIUM HYDROXIDE</b>	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
<b>LAURYL ALCOHOL, ETHOXYLATED &amp; AMMONIUM HYDROXIDE</b>	<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>

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

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

## SECTION 12 ECOLOGICAL INFORMATION

## Toxicity

HomeBright Foaming Super Oxygen Toilet Bowl Cleaner	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
sodium percarbonate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	48	Crustacea	4.9mg/L	2
	NOEC	48	Crustacea	=2mg/L	1
ethylene glycol monobutyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-700mg/L	2
	EC50	48	Crustacea	ca.1-800mg/L	2
	EC50	72	Algae or other aquatic plants	1-840mg/L	2
	NOEC	24	Crustacea	>1-mg/L	2
lauryl alcohol, ethoxylated	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.5mg/L	4
	EC50	72	Algae or other aquatic plants	2.06mg/L	2
	BCF	72	Fish	1mg/L	4
	NOEC	504	Crustacea	0.24mg/L	5
ammonium hydroxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	15mg/L	4
	NOEC	72	Fish	3.5mg/L	4
sodium nitrite	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.048mg/L	4
	EC50	48	Crustacea	ca.12.5100mg/L	1
	EC50	96	Algae or other aquatic plants	12.537mg/L	3

Continued...

HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

	NOEC	96	Fish	0.02mg/L	4
hydrocarbon propellant	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	24.11mg/L	2
	EC50	96	Algae or other aquatic plants	7.71mg/L	2
	LC50	96	Fish	24.11mg/L	2
	EC50	96	Algae or other aquatic plants	7.71mg/L	2
<b>Legend:</b> <i>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</i>					

Harmful to aquatic organisms.  
**DO NOT** discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
lauryl alcohol, ethoxylated	LOW	LOW
sodium nitrite	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
lauryl alcohol, ethoxylated	LOW (LogKOW = 3.6722)
sodium nitrite	LOW (LogKOW = 0.0564)

Mobility in soil

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (KOC = 1)
lauryl alcohol, ethoxylated	LOW (KOC = 10)
sodium nitrite	LOW (KOC = 23.74)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"><li>Consult State Land Waste Management Authority for disposal.</li><li>Discharge contents of damaged aerosol cans at an approved site.</li><li>Allow small quantities to evaporate.</li><li><b>DO NOT</b> incinerate or puncture aerosol cans.</li><li>Bury residues and emptied aerosol cans at an approved site.</li></ul>
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SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	Class : 2.2
	Subrisk : Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions : 63 190 277 327 344 381
	Limited quantity : 1000ml



## HomeBright Foaming Super Oxygen Toilet Bowl Cleaner

## Air transport (ICAO-IATA / DGR)

UN number	1950		
UN proper shipping name	Aerosols, non-flammable		
Transport hazard class(es)	ICAO/IATA Class	2.2	
	ICAO / IATA Subrisk	Not Applicable	
	ERG Code	2L	
Packing group	Not Applicable		
Environmental hazard	Not Applicable		
Special precautions for user	Special provisions	A98 A145 A167 A802	
	Cargo Only Packing Instructions	203	
	Cargo Only Maximum Qty / Pack	150 kg	
	Passenger and Cargo Packing Instructions	203	
	Passenger and Cargo Maximum Qty / Pack	75 kg	
	Passenger and Cargo Limited Quantity Packing Instructions	Y203	
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G	

## Sea transport (IMDG-Code / GGVSee)

UN number	1950		
UN proper shipping name	AEROSOLS		
Transport hazard class(es)	IMDG Class	2.2	
	IMDG Subrisk	Not Applicable	
Packing group	Not Applicable		
Environmental hazard	Not Applicable		
Special precautions for user	EMS Number	F-D , S-U	
	Special provisions	63 190 277 327 344 381 959	
	Limited Quantities	1000 ml	

## 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

## SODIUM PERCARBONATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List  
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes  
 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  
 International Air Transport Association (IATA) Dangerous Goods Regulations  
 International Maritime Dangerous Goods Requirements (IMDG Code)  
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

## ETHYLENE GLYCOL MONOBUTYL ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List  
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes  
 Australia Exposure Standards  
 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) - Part 2, Section Seven - Appendix I  
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

GESAMP/EHS Composite List - GESAMP Hazard Profiles  
 IMO IBC Code Chapter 17: Summary of minimum requirements  
 IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs  
 International Air Transport Association (IATA) Dangerous Goods Regulations  
 International Maritime Dangerous Goods Requirements (IMDG Code)  
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

## LAURYL ALCOHOL, ETHOXYLATED IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List  
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes  
 Australia Inventory of Chemical Substances (AICS)  
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2  
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4  
 International Air Transport Association (IATA) Dangerous Goods Regulations  
 International Maritime Dangerous Goods Requirements (IMDG Code)  
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

## AMMONIUM HYDROXIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

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Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List  
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes  
 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

**SODIUM NITRITE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List  
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes  
 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 2  
 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

IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk  
 International Air Transport Association (IATA) Dangerous Goods Regulations  
 International Maritime Dangerous Goods Requirements (IMDG Code)  
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7  
 GESAMP/EHS Composite List - GESAMP Hazard Profiles  
 IMO IBC Code Chapter 17: Summary of minimum requirements  
 IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk  
 International Air Transport Association (IATA) Dangerous Goods Regulations  
 International Maritime Dangerous Goods Requirements (IMDG Code)  
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

**HYDROCARBON PROPELLANT IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List  
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes  
 Australia Dangerous Goods Code (ADG Code) - Packing Instruction - Liquefied and Dissolved Gases  
 Australia Exposure Standards  
 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  
 Chemical Footprint Project - Chemicals of High Concern List  
 International Air Transport Association (IATA) Dangerous Goods Regulations  
 International Maritime Dangerous Goods Requirements (IMDG Code)  
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

**National Inventory Status**

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (lauryl alcohol, ethoxylated; hydrocarbon propellant; sodium nitrite; ammonium hydroxide; ethylene glycol monobutyl ether; sodium percarbonate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium percarbonate)
Vietnam - NCI	Yes
Russia - ARIPS	Yes
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory 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)

**SECTION 16 OTHER INFORMATION**

<b>Revision Date</b>	04/02/2020
<b>Initial Date</b>	04/02/2020

**SDS Version Summary**

Version	Issue Date	Sections Updated
2.1.1.1	04/02/2020	Acute Health (eye), Fire Fighter (fire/explosion hazard), Toxicity and Irritation (Toxicity Figure)

**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

Continued...



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**HomeBright Foaming Super Oxygen Toilet Bowl Cleaner**

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OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

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TEL (+61 3) 9572 4700.