



# AutoBright Foaming Upholstery Cleaner

JTC Import Export Pty Ltd

Chemwatch: 5388-52

Version No: 2.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: 28/01/2020

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

### Product Identifier

Product name	AutoBright Foaming Upholstery Cleaner
Synonyms	Product Code: 41281
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	Upholstery cleaner.
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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, Eye Irritation Category 2A, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation)
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	<b>WARNING</b>

### Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
AUH044	Risk of explosion if heated under confinement.

### Precautionary statement(s) Prevention

P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing mist/vapours/spray.

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P280	Wear protective gloves/protective clothing/eye protection/face protection.
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## Precautionary statement(s) Response

P321	Specific treatment (see advice on this label).
P362	Take off contaminated clothing and wash before reuse.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
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
111-76-2	1-5	<u>ethylene glycol monobutyl ether</u>
68515-73-1	<1	<u>decyl D-glucoside</u>
151-21-3	<1	<u>sodium lauryl sulfate</u>
532-32-1	<1	<u>sodium benzoate</u>
68476-85-7.	1-10	<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.

## SECTION 5 FIREFIGHTING MEASURES

## Extinguishing media

## AutoBright Foaming Upholstery Cleaner

**SMALL FIRE:**

- ▶ Water spray, dry chemical or CO2

**LARGE FIRE:**

- ▶ Water spray or fog.

**Special hazards arising from the substrate or mixture**

<b>Fire Incompatibility</b>	▶ 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**

<b>Fire Fighting</b>	<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>
<b>Fire/Explosion Hazard</b>	<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: carbon dioxide (CO2) sulfur oxides (SOx) other pyrolysis products typical of burning organic material.</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**

<b>Minor Spills</b>	<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>
<b>Major Spills</b>	<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**

<b>Safe handling</b>	<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> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ <b>DO NOT incinerate or puncture aerosol cans.</b></li> <li>▶ <b>DO NOT spray directly on humans, exposed food or food utensils.</b></li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> </ul>
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	<ul style="list-style-type: none"><li>▶ Work clothes should be laundered separately.</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	▶ 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	<ul style="list-style-type: none"><li>▶ Aerosol dispenser.</li><li>▶ Check that containers are clearly labelled.</li></ul>
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/m3	242 mg/m3 / 50 ppm	Not Available	Not Available
Australia Exposure Standards	hydrocarbon propellant	LPG (liquified petroleum gas)	1000 ppm / 1800 mg/m3	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
sodium lauryl sulfate	Sodium lauryl sulfate	3.9 mg/m3	43 mg/m3	260 mg/m3
sodium benzoate	Benzoic acid, sodium salt	56 mg/m3	620 mg/m3	810 mg/m3
hydrocarbon propellant	Liquified petroleum gas; (L.P.G.)	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm

Ingredient	Original IDLH	Revised IDLH
ethylene glycol monobutyl ether	700 ppm	Not Available
decyl D-glucoside	Not Available	Not Available
sodium lauryl sulfate	Not Available	Not Available
sodium benzoate	Not Available	Not Available
hydrocarbon propellant	2,000 ppm	Not Available

OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
decyl D-glucoside	E	≤ 0.01 mg/m³
sodium lauryl sulfate	E	≤ 0.01 mg/m³
sodium benzoate	E	≤ 0.01 mg/m³

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.
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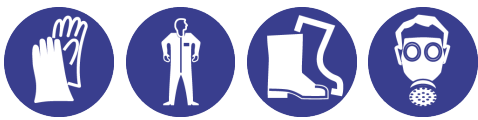
MATERIAL DATA

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.</p> <p>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> <table><tr><td>Type of Contaminant:</td><td>Speed:</td></tr><tr><td>aerosols, (released at low velocity into zone of active generation)</td><td>0.5-1 m/s</td></tr><tr><td>direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 f/min.)</td></tr></table> <p>Within each range the appropriate value depends on:</p> <table><tr><td>Lower end of the range</td><td>Upper end of the range</td></tr><tr><td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr></table>	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.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
Type of Contaminant:	Speed:										
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Lower end of the range	Upper end of the range										
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents										

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	2: Contaminants of low toxicity or of nuisance value only. 3: Intermittent, low production. 4: Large hood or large air mass in motion	2: Contaminants of high toxicity 3: High production, heavy use 4: Small hood-local control only
	Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.	
Personal protection		
Eye and face protection	No special equipment for minor exposure i.e. when handling small quantities. <b>OTHERWISE:</b> For potentially moderate or heavy exposures: ▶ Safety glasses with side shields. ▶ <b>NOTE:</b> Contact lenses pose a special hazard; soft lenses may absorb irritants and <b>ALL</b> lenses concentrate them.	
Skin protection	See Hand protection below	
Hands/feet protection	▶ No special equipment needed when handling small quantities. ▶ <b>OTHERWISE:</b> ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear.	
Body protection	See Other protection below	
Other protection	No special equipment needed when handling small quantities. <b>OTHERWISE:</b> ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces.	

## 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:

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

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

## Respiratory protection

Type AX 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	AX-AUS	-	AX-PAPR-AUS / Class 1
up to 50 x ES	-	AX-AUS / Class 1	-
up to 100 x ES	-	AX-2	AX-PAPR-2 ^

^ - 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.

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

## Information on basic physical and chemical properties

Appearance	Liquid with slight odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	9-11	Decomposition temperature	Not Available

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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

## SECTION 11 TOXICOLOGICAL INFORMATION

## Information on toxicological effects

Inhaled	<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><b>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</b></p>
Ingestion	Not normally a hazard due to physical form of product.
Skin Contact	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</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>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p><b>WARNING: Aerosol containers may present pressure related hazards.</b></p>

AutoBright Foaming Upholstery Cleaner	TOXICITY	IRRITATION
	Not Available	Not Available
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>
decyl D-glucoside	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[2]</sup>	Not Available

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	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup>	
	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	
	Oral (rat) LD50: >5000 mg/kg <sup>[2]</sup>	
sodium lauryl sulfate	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 100 mg/24 hr-moderate
	Oral (rat) LD50: =200-2000 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (human): 25 mg/24 hr - mild
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
sodium benzoate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (rat) LD50: =2100 mg/kg <sup>[2]</sup>	Not Available
hydrocarbon propellant	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<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	

ETHYLENE GLYCOL MONOBUTYL ETHER	<p>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</p> <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):</p> <p>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.</p> <p>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.</p> <p><b>Acute Toxicity:</b> 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 &gt; 85 ppm (508 mg/m3) for EGHE, LC50 &gt; 400ppm (2620 mg/m3) for EGBEA to LC50 &gt; 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 <i>in vitro</i> than those of rats.</p> <p><b>Repeat dose toxicity:</b> 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 <i>in vitro</i> 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 <i>in vitro</i>.</p> <p><b>Mutagenicity:</b> In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in <i>S. typhimurium</i> strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. <i>In vitro</i> cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and <i>in vivo</i> micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.</p> <p><b>Carcinogenicity:</b> 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</p> <p><b>Reproductive and developmental toxicity.</b> 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).</p> <p>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.</p> <p>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).</p> <p>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.</p> <p>At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.</p> <p>Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.</p> <p>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 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</p>	



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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 process 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 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.

A high molecular weight polyglycoside was found to have a NOAEL of 250 mg/kg/day in a 90 day oral study in rats. Adverse treatment related effects were limited to the site of contact (forestomach) in animals treated at higher doses.

Alcohols with a chain length C18-C22 are of low acute toxicity and did not cause adverse effects when dosed at 1000 mg/bw/day in a 28 day study.

Absorption by oral route is expected to be good. For the substance per se, absorption by respiratory route is undetermined and absorption by dermal exposure is most probably limited; furthermore for both routes, absorption is virtually null for workers at the manufacturing steps as the substance is in the form of pearls.

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The components of the UVCB may undergo acido-basic, oxidoreductive reactions and deglycosylation, leading to the same endogenous metabolism as that of fatty acids and glucose. Elimination is expected to be mainly faecal (fatty acids and metabolites) and to a minor extent excretory (organic volatiles and carbon dioxide). No urinary excretion is expected, notably as the putative metabolite glucose, due to regulation of glycemia. The possibility of excretion into milk is undetermined.

REACH Dossier: Acetalization product between glucose and C16-18(even numbered)- alcohol (EC Number 927-870-2)

Alkyl glycosides (syn: alkyl polyglycosides, alkyl polyglycosides, APGs) are considered non-irritating to skin, but irritating to eyes at very high concentrations. A general classification of a 65% C8 alkyl glycoside solution according to the Substance Directive 67/548/EEC is Irritating (Xi) with the risk phrase R41 (Risk of serious damage to the eyes) or R36 (Irritating to the eyes) (Akzo Nobel 1998).

## DECYL D-GLUCOSIDE



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**Acute toxicity:**

In single dose dermal studies with caprylyl/capryl glucoside and C10-16 alkyl glucoside (both 50% a.i., n:1.6) in rabbits, the LD50 was greater than the 2000 mg/kg dose administered. In oral studies with the same test substances, none of the mice dosed with 2000 mg/kg caprylyl glucoside and none of the rats dosed with 5000 mg/kg C10-16 alkyl glucoside died during the study.

**Ocular:**

In system studies for ocular irritation, the ocular irritation potential of decyl, lauryl, C10-16 alkyl, and coco-glucosides was non to slightly irritating and of caprylyl/capryl glucoside was highly irritating. In a HET-CAM study with APG of varying proportions of alkyl chain length, the ocular irritation potential increased with the increased proportion of shorter-chain APGs. In studies using rabbits, neutralized lauryl glucoside produced slight ocular reactions. Caprylyl/capryl glucoside was severely irritating to rabbit eyes when tested undiluted; the irritation threshold value was 10% for 30% a.i. caprylyl/capryl glucoside and 5% for 60% a.i. caprylyl/capryl glucoside.

**Dermal:**

In an in vitro dermal absorption study using human skin samples, the mean absorbed dose of 10% caprylyl/capryl glucoside was 0.01%. APGs of varying chain length (C8/10 to C12/16; 15-70% a.i.) are potentially irritating with irritation potential decreasing with increasing chain length, and, independent of the degree of polymerisation, the irritation was concentration-dependent. The primary dermal irritation indices (PDII) ranged from 0.0 to 4.6 in rabbits. (A PDII of 2 was considered a positive responder).

In clinical studies, the dermal irritation of decyl, lauryl, and coco-glucosides was evaluated in epicutaneous patch (2.0% a.i.) and soap chamber tests (1.0% a.i.), and decyl glucoside was evaluated in a single insult occlusive patch test SIOPT (0.5% a.i.). At most, these ingredients were slightly irritating.

**Ingestion:**

In an oral study in which female mice were dosed by gavage with a 5% aq. solution of caprylyl [U-14C]glucoside, the highest levels of radioactivity at 2 h after dosing were found in the stomach, intestines, liver, and kidney. The radioactivity in the stomach was primarily unchanged substrate, while only a trace amount found in the liver was unchanged. Labeled glucose was found in all of these organs. In a feeding study in rats in which dietary sucrose was replaced with 10 or 20% ethyl glucoside for 39 days, 60-90% of the ingested ethyl glucoside was recovered in the urine.

**Repeat dose toxicity:**

In 2-wk repeated dose dermal studies in rabbits with 60% active caprylyl/capryl glucoside, occlusive applications produced testicular effects, while non-occlusive application did not. In the two occlusive studies, one with 0.09 and 1.8 g a.i./kg and the other with 0.14-1.25 g a.i./kg, an NOEL for testicular effects could not be established. In the non-occlusive study, the NOEL for systemic toxicity was 0.18 g a.i./kg caprylyl/capryl glucoside. Severe dermal irritation was observed in both occlusive studies, while slight to moderate irritation was reported in the non-occlusive study.

Dermal application of 60% active caprylyl/capryl glucoside, 0.9-1.8 g a.i./kg, under occlusive conditions may affect the testes and accessory sex glands of rabbits; however, it was not clear if the effects were test-article related or due to stress of the occlusive procedure and resulting irritation and weight loss. Lauryl glucoside, 100-1000 mg/kg by gavage, did not produce adverse reproductive or developmental effects. Lauryl glucoside, 0.1-10,000 nmol, did not have any effects in in vitro oestrogenicity assays.

In oral repeated dose toxicity studies, moderately-dilated renal tubules were observed in 3 of 6 rats fed 20% ethyl glucoside for 39 days, but in none of the rats fed 10% ethyl glucoside. Kidney weights were statistically significantly increased in the test animals. In rats dosed orally with 250-1000 mg/kg C12/16 APG for 13 wks, reversible irritation and ulceration of the stomach mucosa was observed, but there was no systemic toxicity reported for any group.

**Mutagenicity:**

Alkyl polyglucosides (polyglycoses; APGs) (chain length not specified), tested at 8-500 ug/l and 11-900 ug/plate in distilled water, were not mutagenic in Ames tests with or without metabolic activation. C10-16 APG, tested at concentrations of <= 160 ug/ml with and without metabolic activation, was not clastogenic.

**Sensitisation:**

Glucosides with alkyl chain lengths ranging from C8-C10 to >C18, as well as a C18 branched glucoside, were evaluated in both the guinea pig maximisation test (GPMT), at concentrations of 1.25-10% for intradermal induction, 5-100% for epidermal induction, and 2.5-50% for challenge, and the local lymph node assay (LLNA) at concentrations of 1.25-50%. None of the glucosides tested were irritants or sensitisers in the GPMT, but the LLNA indicated that one C12-C18 glucoside, C14 glucoside, and C18 branched glucoside may cause skin sensitization at concentrations of 8.4%, 5.9%, and 0.43%, respectively. The sensitization potential of C12/16 APG was evaluated in studies in guinea pigs using the Buehler method (test concentrations of 20%) and the Magnusson-Kligman protocol (1, 60, and 10% used for intracutaneous induction, epidermal induction, and epidermal challenge respectively). C12/16 APG was not a sensitiser in the Buehler or Magnusson-Kligman studies. In clinical testing, the sensitization potential of 0.5, 0.75, and 1.8% a.i. decyl glucoside (in formulation), 5% a.i. aq. decyl and lauryl glucoside, and 1% a.i. aq. coco-glucoside was evaluated in Human Repeat Insult Patch Tests (HRIPTs). These ingredients were not irritating or sensitising.

CIR Expert Panel Meeting, September 2011

**SODIUM LAURYL SULFATE**

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

for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates

Most chemicals of this category are not defined substances, but mixtures of homologues with different alkyl chain lengths. Alpha-olefin sulfonates are mixtures of alkene sulfonate and hydroxyl alkane sulfonates with the sulfonate group in the terminal position and the double bond, or hydroxyl group, located at a position in the vicinity of the sulfonate group. Common physical and/or biological pathways result in structurally similar breakdown products, and are, together with the surfactant properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

**Acute toxicity:** These substances are well absorbed after ingestion; penetration through the skin is however poor. After absorption, these chemicals are distributed mainly to the liver.

Acute oral LD50 values of alkyl sulfates in rats and/or mice were (in mg/kg):

C10-; 290-580

C10-16-; and C12-; 1000-2000

C12-14, C12-15, C12-16, C12-18 and C16-18-; >2000

C14-18, C16-18-; >5000

The clinical signs observed were non-specific (piloerection, lethargy, decreased motor activity and respiratory rate, diarrhoea). At necropsy the major findings were irritation of the gastrointestinal tract and anemia of inner organs.

Based on limited data, the acute oral LD50 values of alkane sulfonates and alpha-olefin sulfonates of comparable chain lengths are assumed to be in the same range.

The counter ion does not appear to influence the toxicity in a substantial way.

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Acute dermal LD50 values of alkyl sulfates in rabbits (mg/ kg):

C12-; 200

C12-13 and C10-16-;>500

Apart from moderate to severe skin irritation, clinical signs included tremor, tonic-clonic convulsions, respiratory failure, and body weight loss in the study with the C12- alkyl sulfate and decreased body weights after administration of the C10-16- alkyl sulfates. No data are available for alkane sulfonates but due to a comparable metabolism and effect concentrations in long-term studies effect concentrations are expected to be in the same range as found for alkyl sulfates.

There are no data available for acute inhalation toxicity of alkyl sulfates, alkane sulfonates or alpha-olefin sulfonates.

In skin irritation tests using rabbits (aqueous solutions, OECD TG 404):

C8-14 and C8-16 (30%), C12-14 (90%), C14-18 (60%)- corrosive

Under occlusive conditions:

C12, and C12-14 (25%), C12-15-, C13-15 and C15-16 (5-7%) - moderate to strong irritants

Comparative studies investigating skin effects like transepidermal water loss, epidermal electrical conductance, skin swelling, extraction of amino acids and proteins or development of erythema in human volunteers consistently showed a maximum of effects with C12-alkyl sulfate, sodium; this salt is routinely used as a positive internal control giving borderline irritant reactions in skin irritation studies performed on humans. As the most irritant alkyl sulfate it can be concluded that in humans 20% is the threshold concentration for irritative effects of alkyl sulfates in general. No data were available with regard to the skin irritation potential of alkane sulfonates. Based on the similar chemical structure they are assumed to exhibit similar skin irritation properties as alkyl sulfates or alpha-olefin sulfonates of comparable chain lengths.

In eye irritation tests, using rabbits, C12-containing alkyl sulfates (>10% concentration) were severely irritating and produced irreversible corneal effects. With increasing alkyl chain length, the irritating potential decreases, and C16-18 alkyl sulfate sodium, at a concentration of 25%, was only a mild irritant.

Concentrated C14-16- alpha-olefin sulfonates were severely irritating, but caused irreversible effects only if applied as undiluted powder. At concentrations below 10% mild to moderate, reversible effects, were found. No data were available for alkane sulfonates

Alkyl sulfates and C14-18 alpha-olefin sulfonates were not skin sensitizers in animal studies. No reliable data were available for alkane sulfonates. Based on the similar chemical structure, no sensitisation is expected.

However anecdotal evidence suggests that sodium lauryl sulfate causes pulmonary sensitisation resulting in hyperactive airway dysfunction and pulmonary allergy accompanied by fatigue, malaise and aching. Significant symptoms of exposure can persist for more than two years and can be activated by a variety of non-specific environmental stimuli such as an exhaust, perfumes and passive smoking.

Absorbed sulfonates are quickly distributed through living systems and are readily excreted. Toxic effects may result from the effects of binding to proteins and the ability of sulfonates to translocate potassium and nitrate (NO<sub>3</sub><sup>-</sup>) ions from cellular to interstitial fluids. Airborne sulfonates may be responsible for respiratory allergies and, in some instances, minor dermal allergies. Repeated skin contact with some sulfonated surfactants has produced sensitisation dermatitis in predisposed individuals

**Repeat dose toxicity:** After repeated oral application of alkyl sulfates with chain lengths between C12 and C18, the liver was the only target organ for systemic toxicity. Adverse effects on this organ included an increase in liver weight, enlargement of liver cells, and elevated levels of liver enzymes. The LOAEL for liver toxicity (parenchymal hypertrophy and an increase in comparative liver weight) was 230 mg/kg/day (in a 13 week study with C16-18 alkyl sulfate, sodium). The lowest NOAEL in rats was 55 mg/kg/day (in a 13 week study with C12-alkyl sulfate, sodium). C14- and C14-16-alpha-olefin sulfonates produced NOAELs of 100 mg/kg/day (in 6 month- and 2 year studies). A reduction in body weight gain was the only adverse effect identified in these studies.

No data were available with regard to the repeated dose toxicity of alkane sulfonates. Based on the similarity of metabolic pathways between alkane sulfonates, alkyl sulfates and alkyl-olefin sulfonates, the repeated dose toxicity of alkane sulfonates is expected to be similar with NOAEL and LOAEL values in the same range as for alkyl sulfates and alpha-olefin sulfonates, i.e. 100 and 200-250 mg/kg/day, respectively, with the liver as potential target organ.

**Genotoxicity:** Alkyl sulfates of different chain lengths and with different counter ions were not mutagenic in standard bacterial and mammalian cell systems both in the absence and in the presence of metabolic activation. There was also no indication for a genotoxic potential of alkyl sulfates in various in vivo studies on mice (micronucleus assay, chromosome aberration test, and dominant lethal assay). alpha-Olefin sulfonates were not mutagenic in the Ames test, and did not induce chromosome aberrations in vitro. No genotoxicity data were available for alkane sulfonates. Based on the overall negative results in the genotoxicity assays with alkyl sulfates and alpha-olefin sulfonates, the absence of structural elements indicating mutagenicity, and the overall database on different types of sulfonates, which were all tested negative in mutagenicity assays, a genotoxic potential of alkane sulfonates is not expected.

**Carcinogenicity:** Alkyl sulfates were not carcinogenic in feeding studies with male and female Wistar rats fed diets with C12-15 alkyl sulfate sodium for two years (corresponding to doses of up to 1125 mg/kg/day).

alpha-Olefin sulfonates were not carcinogenic in mice and rats after dermal application, and in rats after oral exposure.

No carcinogenicity studies were available for the alkane sulfonates.

**Reproductive toxicity:** No indication for adverse effects on reproductive organs was found in various oral studies with different alkyl sulfates. The NOAEL for male fertility was 1000 mg/kg/day for sodium dodecyl sulfate. In a study using alpha-olefin sulfonates in male and female rats, no adverse effects were identified up to 5000 ppm.

**Developmental toxicity:** In studies with various alkyl sulfates (C12 up to C16-18- alkyl) in rats, rabbits and mice, effects on litter parameters were restricted to doses that caused significant maternal toxicity (anorexia, weight loss, and death).

The principal effects were higher foetal loss and increased incidences of total litter losses. The incidences of malformations and visceral and skeletal anomalies were unaffected apart from a higher incidence of delayed ossification or skeletal variation in mice at > 500 mg/kg bw/day indicative of a delayed development. The lowest reliable NOAEL for maternal toxicity was about 200 mg/kg/day in rats, while the lowest NOAELs in offspring were 250 mg/kg/day in rats and 300 mg/kg/day for mice and rabbits.

For alpha-olefin sulfonates (C14-16-alpha-olefin sulfonate, sodium) the NOAEL was 600 mg/kg/day both for maternal and developmental toxicity. No data were available for the reproductive and developmental toxicity of alkane sulfonates. Based on the available data, the similar toxicokinetic properties and a comparable metabolism of the alkyl sulfates and alkane sulfonates, alkane sulfonates are not considered to be developmental toxicants.

Although the database for category members with C<12 is limited, the available data are indicating no risk as the substances have comparable toxicokinetic properties and metabolic pathways. In addition, longer-term studies gave no indication for adverse effects on reproductive organs with different alkyl sulfates

Alkyl sulfates (AS) anionic surfactants 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 R41 (Risk of serious damage to eyes). An exception has been made for C12 AS which is classified as Harmful (Xn) with the risk phrases R22 (Harmful if swallowed) and R38 and R41 (CESIO 2000). AS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC.

AS are readily absorbed from the gastrointestinal tract after oral administration. Penetration of AS through intact skin appears to be minimal. AS are extensively metabolized in various species resulting in the formation of several metabolites. The primary metabolite is butyric acid-4-sulfate.

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The major site of metabolism is the liver. AS and their metabolites are primarily eliminated via the urine and only minor amounts are eliminated via the faeces. In rats about 70-90% of the dose was eliminated via the urine within 48 hours after oral, intravenous or intraperitoneal administration of 1 mg of AS per rat. The acute toxicity of AS in animals is considered to be low after skin contact or oral intake.

For a homologous series of AS (C8 to C16), maximum swelling of stratum corneum (the outermost layer of epidermis) of the skin was produced by the C12 homologue. This is in accordance with the fact that the length of the hydrophobic alkyl chain influences the skin irritation potential. Other studies have shown that especially AS of chain lengths C11, C12 and C13 remove most amino acids and soluble proteins from the skin during washing.

Concentrated samples of AS are skin irritants in rabbits and guinea pigs. AS are non-irritant to laboratory animals at a 0.1% concentration. C12 AS is used in research laboratories as a standard substance to irritate skin and has been shown to induce an irritant eczema. AS were found, by many authors, to be the most irritating of the anionic surfactants, although others have judged the alkyl sulfates only as irritant as laurate (fatty acid soap).

A structure/effect relationship with regard to the length of the alkyl chain can also be observed on mucous membranes. The maximum eye irritation occurs at chain lengths of C10 to C14. In acute ocular tests, 10% C12 AS caused corneal damage to the rabbit eyes if not irrigated.

Another study showed that a 1.0% aqueous C12 AS solution only had a slight effect on rabbit eyes, whereas 5% C12 AS caused temporary conjunctivitis, and 25% C12 AS resulted in corneal damage.

In a 13-week feeding study, rats were fed dietary levels of 0, 40, 200, 1,000 or 5,000 ppm of C12 AS. The only test material related effect observed was an increase in absolute organ weights in the rats fed with the highest concentration which was 5,000 ppm. The organ weights were not further specified and no other abnormalities were found.

In a mutagenicity study, rats were fed 1.13 and 0.56% C12 AS in the diet for 90 days. This treatment did not cause chromosomal aberrations in the bone marrow cells.

Mutagenicity studies with *Salmonella typhimurium* strains (Ames test) indicate no mutagenic effects of C12 AS. The available long-term studies in experimental animals (rats and mice) are inadequate to evaluate the carcinogenic potential of AS. However, in studies in which animals were administered AS in the diet at levels of

up to 4% AS, there was no indication of increased risk of cancer after oral ingestion.

No specific teratogenic effects were observed in rabbits, rats or mice when pregnant animals were dosed with 0.2, 2.0, 300 and 600 mg C12 AS/kg body weight/day by gavage during the most important period of organogenesis (day 6 to 15 of pregnancy for mice and rats and day 6 to 18 of pregnancy for rabbits). Reduced litter size, high incidence of skeletal abnormalities and foetal loss were observed in mice at 600 mg C12 AS/kg/day, a dose level which also caused severe toxic effects in the parent animals in all three species. An aqueous solution of 2% AS was applied (0.1 ml) once daily to the dorsal skin (2 x 3 cm) of pregnant mice from day 1 to day 17 of gestation. A solution of 20% AS was tested likewise from day 1 to day 10 of gestation. The

mice were killed on days 11 and 18, respectively. A significant decrease in the number of implantations was observed when mice were treated with 20% AS compared to a control group which was dosed with water. No evidence of teratogenic effects was noted.

When aqueous solutions of 2% and 20% AS (0.1 ml) were applied once per day to the dorsal skin (2 x 3 cm) of pregnant ICR/Jc1 mice from day 12 to day 17 of gestation no effects on pregnancy outcome were detected. Treatment with 20% AS resulted in growth retardation of suckling mice, but this effect disappeared after weaning. A 10% AS solution (0.1 ml) was applied twice daily to the dorsal skin (2 x 3 cm) of pregnant ICR/Jc1 mice during the preimplantation period (days 0-3 of gestation). A significant number of embryos collected on day 3 as severely deformed or remained at the morula stage. The number of embryos in the oviducts was significantly greater for the mice dosed with AS as compared to the control mice. No pathological changes were detected in the major organs of the dams

**NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.

Eye (None) None: None None rabbit None 250 ugSkin (rabbit):25 mg/24 hr-moderate Skin (None) None: None rabbit None 50 mg/24Eye (rabbit) 10: mg-

## SODIUM BENZOATE

For benzoates:

**Acute toxicity:** Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol.

The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds.

Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye.

**Sensitisation:** The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers.

**Repeat dose toxicity:** For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed.

For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur.

**Mutagenicity:** All chemicals showed no mutagenic activity in *in vitro* Ames tests. Various results were obtained with other *in vitro* genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity *in vivo*. While some mixed and/or equivocal *in vitro* chromosomal/chromatid responses have been observed, no genotoxicity was observed in the *in vivo* cytogenetic, micronucleus, or other assays. The weight of the evidence of the *in vitro* and *in vivo* genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.

In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts.

**Developmental toxicity:** In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed.

**NOTE:** Oral doses of 8-10g may cause nausea and vomiting, though tolerance in human is 50 g/day. Use in food limited to 0.1%. [ICI]

## 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.

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	<p>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</p> <p><b>Acute toxicity:</b> 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 &gt; 1063 ppm) &gt; C1-C4 HCs (LC50 &gt; 10,000 ppm) &gt; benzene (LC50 = 13,700 ppm) &gt; butadiene (LC50 = 129,000 ppm) &gt; asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p><b>Repeat dose toxicity:</b> 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 .&gt;=10 ppm) &gt;C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) &gt; C5-C6 HCs (LOAEL = 6,625 ppm) &gt; butadiene (LOAEL = 8,000 ppm) &gt; asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p><b>Genotoxicity:</b> <i>In vitro:</i> The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vitro</i> genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian <i>in vitro</i> test systems. <i>In vivo:</i> The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vivo</i> genotoxicity. The exceptions are benzene and 1,3-butadiene, which are genotoxic in <i>in vivo</i> test systems</p> <p><b>Developmental toxicity:</b> 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 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: 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: 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>
DECYL D-GLUCOSIDE & SODIUM BENZOATE	<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>
DECYL D-GLUCOSIDE & HYDROCARBON PROPELLANT	No significant acute toxicological data identified in literature search.

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

AutoBright Foaming Upholstery Cleaner	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

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

decyl D-glucoside	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	62.249mg/L	3
	EC50	96	Algae or other aquatic plants	187.298mg/L	3
	LC50	96	Fish	96.64mg/L	2
	EC50	48	Crustacea	31.62mg/L	2
	EC50	72	Algae or other aquatic plants	7.03mg/L	2
	EC10	504	Crustacea	1.76mg/L	2
	NOEC	504	Crustacea	1mg/L	2

## AutoBright Foaming Upholstery Cleaner

sodium lauryl sulfate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.59mg/L	4
	EC50	48	Crustacea	0.67mg/L	4
	EC50	96	Algae or other aquatic plants	1.2mg/L	4
	BCF	1	Fish	0.85mg/L	4
	EC15	24	Crustacea	0.17mg/L	4
	NOEC	0.08	Fish	0.000013mg/L	4
sodium benzoate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	650mg/L	2
	EC50	72	Algae or other aquatic plants	>30.5mg/L	2
	EC10	72	Algae or other aquatic plants	6.5mg/L	2
	NOEC	72	Algae or other aquatic plants	0.09mg/L	2
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

**Legend:** 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

**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)
decyl D-glucoside	LOW	LOW
sodium lauryl sulfate	HIGH	HIGH

## Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
decyl D-glucoside	LOW (LogKOW = 1.916)
sodium lauryl sulfate	LOW (BCF = 7.15)

## Mobility in soil

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (KOC = 1)
decyl D-glucoside	LOW (KOC = 10)
sodium lauryl sulfate	LOW (KOC = 10220)

## 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 incinerate or puncture aerosol cans.</b></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

Continued...



AutoBright Foaming Upholstery Cleaner

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

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

ETHYLENE GLYCOL MONOBUTYL ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List	GESAMP/EHS Composite List - GESAMP Hazard Profiles
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes	IMO IBC Code Chapter 17: Summary of minimum requirements
Australia Exposure Standards	IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Inventory of Chemical Substances (AICS)	International Air Transport Association (IATA) Dangerous Goods Regulations
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Part 2, Section Seven - Appendix I	International Maritime Dangerous Goods Requirements (IMDG Code)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

DECYL D-GLUCOSIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)	IMO IBC Code Chapter 17: Summary of minimum requirements
GESAMP/EHS Composite List - GESAMP Hazard Profiles	IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk

SODIUM LAURYL SULFATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...



## AutoBright Foaming Upholstery Cleaner

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)

GESAMP/EHS Composite List - GESAMP Hazard Profiles  
 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

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

Australia Inventory of Chemical Substances (AICS)  
 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

**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 (decyl D-glucoside; sodium benzoate; hydrocarbon propellant; ethylene glycol monobutyl ether)
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 (decyl D-glucoside)
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>	28/01/2020
<b>Initial Date</b>	28/01/2020

**SDS Version Summary**

Version	Issue Date	Sections Updated
2.1.1.1	28/01/2020	Fire Fighter (fire/explosion hazard), Ingredients, Supplier Information

**Other information**

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

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  
 BCF: BioConcentration Factors  
 BEI: Biological Exposure Index

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