

NV Chemicals Hi-Foam Car Wash

N.V. Chemicals (Aust) P/L

Chemwatch Hazard Alert Code: 2

Chemwatch: 4789-79

Version No: 6.1

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

Issue Date: 10/03/2023

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L.GHS.AUS.EN.E

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

Product Identifier

Product name	NV Chemicals Hi-Foam Car Wash
Chemical Name	Not Applicable
Synonyms	car shampoo
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Designed for cleaning and shampooing of cars. SDS are intended for use in the workplace ONLY. For domestic-use products, refer to consumer labels.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	N.V. Chemicals (Aust) P/L
Address	24 Lisa Place Coolaroo VIC 3048 Australia
Telephone	+61 3 9351 1100
Fax	+61 3 9351 1077
Website	http://www.nvchemicals.com.au/
Email	info@nvchemicals.com.au

Emergency telephone number

Association / Organisation	N.V.Chemicals(Aust) P/L
Emergency telephone numbers	0411 387 097
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

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

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

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Acute Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

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

H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H401	Toxic to aquatic life.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
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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
9004-82-4	10-30	<u>sodium lauryl ether sulfate</u>
1643-20-5	1-10	<u>lauryldimethylamine oxide</u>
61789-40-0	1-10	<u>cocamidopropylbetaine</u>
9016-45-9	1-10	<u>nonylphenol, ethoxylated</u>
50-00-0	<1	<u>formaldehyde</u>
7732-18-5	>60	<u>water</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

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

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.

Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.
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Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures**Extinguishing media**

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

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

In such an event consider:

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<p>The emulsion is not combustible under normal conditions. However, it will break down under fire conditions and the hydrocarbon component will burn.</p> <p>Decomposes on heating and produces toxic fumes of: carbon dioxide (CO₂) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</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"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	<p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage**Precautions for safe handling**

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

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	None known

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	formaldehyde.	Formaldehyde	1 ppm / 1.2 mg/m ³	2.5 mg/m ³ / 2 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
nonylphenol, ethoxylated	4.5 mg/m ³	49 mg/m ³	300 mg/m ³
nonylphenol, ethoxylated	43 mg/m ³	470 mg/m ³	5,400 mg/m ³
formaldehyde.	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
sodium lauryl ether sulfate	Not Available	Not Available
lauryldimethylamine oxide	Not Available	Not Available
cocamidopropylbetaine	Not Available	Not Available
nonylphenol, ethoxylated	Not Available	Not Available
formaldehyde.	20 ppm	Not Available
water	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sodium lauryl ether sulfate	E	≤ 0.01 mg/m ³
lauryldimethylamine oxide	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m ³)
cocamidopropylbetaine	E	≤ 0.1 ppm
nonylphenol, ethoxylated	E	≤ 0.1 ppm

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

MATERIAL DATA

None assigned. Refer to individual constituents.

NOTE D: Certain substances which are susceptible to spontaneous polymerisation or decomposition are generally placed on the market in a stabilised form. It is in this form that they are listed on Annex I

When they are placed on the market in a non-stabilised form, the label must state the name of the substance followed by the words "non-stabilised"


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 operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. 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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	<p>Type of Contaminant:</p> <p>solvent, vapours, degreasing etc., evaporating from tank (in still air).</p> <p>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</p> <p>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</p> <p>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</p> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="384 465 1145 618"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only	<p>Air Speed:</p> <p>0.25-0.5 m/s (50-100 f/min)</p> <p>0.5-1 m/s (100-200 f/min.)</p> <p>1-2.5 m/s (200-500 f/min.)</p> <p>2.5-10 m/s (500-2000 f/min.)</p>
Lower end of the range	Upper end of the range											
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents											
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity											
3: Intermittent, low production.	3: High production, heavy use											
4: Large hood or large air mass in motion	4: Small hood-local control only											
<p>Individual protection measures, such as personal protective equipment</p>												
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59]. 											
<p>Skin protection</p>	<p>See Hand protection below</p>											
<p>Hands/feet protection</p>	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>											
<p>Body protection</p>	<p>See Other protection below</p>											
<p>Other protection</p>	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. 											

- ▶ Skin cleansing cream.
- ▶ Eye wash unit.

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

NV Chemicals Hi-Foam Car Wash

Material	CPI
BUTYL	A
NEOPRENE	A
VITON	A
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
TEFLON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

Type BKAX-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	BKAX-AUS P2	-	BKAX-PAPR-AUS / Class 1 P2
up to 50 x ES	-	BKAX-AUS / Class 1 P2	-
up to 100 x ES	-	BKAX-2 P2	BKAX-PAPR-2 P2 ^

^ - Full-face

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

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

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	Blue alkaline liquid; mixes with water to form a foaming solution.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	8-8.5	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	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"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information**Information on toxicological effects**

Inhaled	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
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>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>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</p> <p>Anionic surfactants/ hydrotropes generally produce skin reactions following the removal of natural oils. The skin may appear red and may become sore. Papular dermatitis may also develop. Sensitive individuals may exhibit cracking, scaling and blistering.</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	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. Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation. Direct eye contact with some concentrated anionic surfactants/ hydrotropes produces corneal damage, in some cases severe. Low concentrations may produce immediate discomfort, conjunctival hyperaemia, and oedema of the corneal epithelium. Healing may take several days. Temporary clouding of the cornea may occur.
Chronic	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</p> <p>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 (NO₃⁻) ions from cellular to interstitial fluids. Airborne sulfonates may be responsible for respiratory allergies and, in some instances, minor dermal allergies.</p> <p>There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.</p>

NV Chemicals Hi-Foam Car Wash	TOXICITY	IRRITATION
	Not Available	Not Available
sodium lauryl ether sulfate	TOXICITY	IRRITATION
	Oral (Rat) LD50: 1600 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit):25 mg/24 hr moderate
		Skin: adverse effect observed (irritating) ^[1]
lauryldimethylamine oxide	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 50 ug/24h - SEVERE

Continued...

NV Chemicals Hi-Foam Car Wash

	Oral (Rat) LD50: >600 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
		Skin (rabbit): 2 mg/24h - SEVERE
		Skin: adverse effect observed (irritating) ^[1]
cocamidopropylbetaine	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50: 2700 mg/kg ^[2]	Eye: primary irritant *
		Skin: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
		Skin: primary irritant *
nonylphenol, ethoxylated	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2943.2 mg/kg ^[2]	Eye (rabbit): 5 mg SEVERE
	Oral (Rat) LD50: 1310 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (human): 15 mg/3D mild
		Skin (rabbit): 500 mg mild
		Skin: adverse effect observed (irritating) ^[1]
formaldehyde.	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 270 mg/kg ^[2]	Eye (human): 4 ppm/5m
	Inhalation (Rat) LC50: <463 ppm4h ^[1]	Eye (rabbit): 0.75 mg/24H SEVERE
	Oral (Rat) LD50: 100 mg/kg ^[2]	Skin (human): 0.15 mg/3d-I mild
		Skin (rabbit): 2 mg/24H SEVERE
		Skin: adverse effect observed (corrosive) ^[1]
water	TOXICITY	IRRITATION
	Oral (Rat) LD50: >90000 mg/kg ^[2]	Not Available

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

NV Chemicals Hi-Foam Car Wash	Not available.
SODIUM LAURYL ETHER SULFATE	<p>* [CESIO]</p> <p>Alkyl ether sulfates (alcohol or alkyl ethoxysulfates) (AES) (syn: AAASD ,alkyl alcohol alkoxyate sulfates, SLES) are generally classified according to Comité Européen des Agents de Surface et leurs Intermédiaires Organiques (CESIO) as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R36 (Irritating to eyes). An exception has been made for AES (2-3E0) in a concentration of 70-75% where R36 is substituted with R41 (Risk of serious damage to eyes).</p> <p>AES are not included in Annex 1 of the list of dangerous substances of Council Directive 67/548/EEC.</p> <p>In assessing this family the Cosmetic Ingredient Review (CIR) Expert Panel recognized that most of the acute oral toxicity, dermal irritation and sensitization, subchronic and chronic oral toxicity, reproductive and developmental toxicity, carcinogenicity, and photosensitization studies have been conducted on ammonium laureth sulfate and sodium laureth sulfate. Sodium and ammonium laureth sulfate have not evoked adverse responses in any toxicological testing, including acute oral toxicity, sub-chronic and chronic oral toxicity, reproductive and developmental toxicity, carcinogenicity, and photosensitization studies. These data, however, are considered a sufficient basis for concluding that the other ingredients are safe in the practices of use and concentration described in the safety assessment because of the fundamental chemical similarities between them and because they all are chemically similar salts (salts are expected to be dissociated in any product formulation independent of whether the salt is sodium, ammonium, magnesium, or zinc) of sulfated ethoxylated alcohols, and they all function as surfactants in cosmetic formulations. Based on these considerations, safety test data on one ingredient may be extrapolated to all of them. The panel noted that sodium laureth sulfate and ammonium laureth sulfate can produce eye and/or skin irritation in experimental animals and in some human test subjects; irritation may occur in some users of cosmetic formulations containing these ingredients. The irritant effects, however, are similar to those produced by other detergents, and the severity of the irritation appears to increase directly with concentration.</p> <p>Acute toxicity: AES are of low acute toxicity. Neat AES are irritant to skin and eyes. The irritation potential of AES containing solutions depends on concentration. Local dermal effects due to direct or indirect skin contact with AES containing solutions in hand-washed laundry or hand dishwashing are not of concern because AES is not a contact sensitizer and AES is not expected to be irritating to the skin at in-use concentrations. The available repeated dose toxicity data demonstrate the low toxicity of AES. Also, they are not considered to be mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. The consumer aggregate exposure from direct and indirect skin contact as well as from the oral route via dishware residues results in an estimated total body burden of 29 ug /kg bw/day. AES are easily absorbed in the intestine in rats and humans after oral administration. Radiolabelled C11 AE3S and C12 AE3S were extensively metabolized in rats and most of the 14C-activity was eliminated via the urine and expired air independently of the route of administration (oral, intraperitoneal or intravenous). The main urinary metabolite from C11 AE3S is propionic acid-3-(3EO)-sulfate. For C12 and C16 AE3S, the main metabolite is acetic acid-2-(3EO)-sulfate. The alkyl chain appears to be oxidised to CO₂ which is expired. The EO-chain seems to be resistant to metabolism.</p> <p>AES are better tolerated on the skin than, e.g., alkyl sulfates and it is generally agreed that the irritancy of AES is lower than that of other anionic surfactants. Alkyl chain lengths of 12 carbon atoms are considered to be more irritating to the skin compared to other chain lengths. The skin irritating properties of AES normally decrease with increasing level of ethoxylation. Undiluted AES should in general be considered strongly irritating. Even at concentrations of 10% moderate to strong effects can be expected. However, only mild to slight irritation was observed when a non-specified AES was applied at 1% to the skin.</p> <p>Subchronic toxicity: A 90-day subchronic feeding study in rats with 1% of AE3S or AE6S with alkyl chain lengths of C12-14 showed only an increased liver/body weight ratio. In a chronic oral study with a duration of 2 years, doses of C12-AE3S of 0.005 - 0.05% in the diet or drinking water had no effects on rats. The concentration of 0.5% sometimes resulted in increased kidney or liver weight.</p> <p>Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2 (CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day</p>

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(calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction.

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

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

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

NOTE: Some products containing AES/ SLES have been found to also contain traces (up to 279 ppm) of 1,4-dioxane; this is formed as a by-product during the ethoxylation step of its synthesis. The U.S. Food and Drug Administration recommends that these levels be monitored. The U.S. Environmental Protection Agency classifies 1,4-dioxane to be a probable human carcinogen (not observed in epidemiological studies of workers using the compound, but resulting in more cancer cases in controlled animal studies), and a known irritant with a no-observed-adverse-effects level of 400 milligrams per cubic meter at concentrations significantly higher than those found in commercial products. Under Proposition 65, 1,4-dioxane is classified in the U.S. state of California to cause cancer. The FDA encourages manufacturers to remove 1,4-dioxane, though it is not required by federal law.

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

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-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 autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing

Toxicokinetics:

Following oral exposure, AES is readily absorbed in the gastrointestinal tract in human and rat and excreted principally via the urine or faeces depending on the length of the ethoxylate chain but independently of the route of administration. Once absorbed, AES is extensively metabolized by beta- or omega oxidation. The alkyl chain appears to be oxidized to CO₂ which is expired. The EO-chain seems to be resistant to metabolism. Regarding the different anions, it is expected that the salts will be converted to the acid form in the stomach. This means that for all types of parent chemical the same compound structure eventually enters the small intestine. Hence, the situation will be similar for compounds originating from different salts and therefore no differences in uptake are anticipated.

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

Dermal absorption

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

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

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

There is also an *in vivo* study according to OECD guideline 427 for AES (C12-14; 2 EO) Na (CAS 68891-38-3) available (Aulmann, 1996).

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

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

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

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

References:

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<http://journals.sagepub.com/doi/pdf/10.1177/1091581810373151>

LAURYL DIMETHYLAMINE OXIDE

For amine oxides (AOs):

Substantial data exist for mammalian toxicity by *in vitro* and *in vivo* testing. Amine oxides are produced, and transported in aqueous solutions that are 25-35% concentration and most tests were conducted with aqueous solutions in that concentration range. Sometimes aqueous formulations were tested where the AO was at lesser concentrations than 25-35%. Whatever concentration were tested, results are reported below for the active ingredient, amine oxide, in mg AO/kg bw for dermal and oral acute toxicity results and mg AO/kg bw/day for repeated dose studies.

Toxicokinetic and metabolism studies indicate AOs are extensively metabolised and readily excreted after oral administration. Amine oxide was readily absorbed dermally by rats, mice and rabbits after 24 to 72 hours of exposure. After 8 hours of dermal exposure, humans absorbed <1%.

Acute toxicity: In rat oral acute toxicity limit tests, no deaths occurred at single doses of 600 mg C10-16 AO/kg bw or less (for CAS No 70592-80-2). In multi-dose studies, acute oral LD50 values for rats ranged from 846 mg AO/kg bw to 3873 mg AO/kg bw (both values for CAS No 61788-90-7), with several other AOs having rat oral LD50s falling within this range. In single dose acute dermal toxicity limit tests, no deaths occurred at a dose of 520 mg AO/kg bw (CAS No 70592-80-2). This dose was equivalent to 2 mL/kg of a 30% formulation. There were no deaths observed in a rat acute inhalation study to aerosol droplets of a consumer product providing a dose of 0.016 mg AO/L.

In a series of studies on rabbits, AOs of varying chain length showed consistent results and all

- ▶ were not irritating to the skin or eyes at low concentrations (1%),
- ▶ were moderately irritating at 5%, and
- ▶ more severely irritating when tested as produced (e.g., ~30% aqueous solutions).

In studies that included rinsing, eye irritation effects diminished with rinsing after 30 seconds of exposure and were slight with rinsing after 4 seconds of exposure. In Draize rabbit eye irritation tests using ~30% AO solutions, rabbits experienced severe to moderate irritation. (The maximum concentration of AO is 10% active in consumer products.) Accidental eye exposure in manufacturing employee incidents and consumer incidents established that eye irritation effects of exposure during manufacturing and use of products containing AO and other surfactants are moderate, transient and reversible

There is no indication of skin sensitisation for the AO category based on the available animal and human data.

Repeat dose toxicity: In four repeated-dose studies with rats and mice exposed to AO via oral and dermal routes (all with CAS No 70592-80-2), three dermal studies were designed to assess the effect of repeated exposure on skin at maximum doses of 1.5 mg AO/kg-bw/day. Higher doses were tested in a 90-day dietary study with rabbits. No treatment related clinical chemistry, hematology and histopathological changes were observed. In these studies, LOAELs ranged from 87 to 150 mg AO/kg bw/day with the highest oral NOEL below the lowest LOAEL as 80 mg AO/kg bw/day. Signs of toxicity observed in the oral study included suppressed mean body weight gain, lenticular opacities and diarrhea; in the dermal studies, local dermal irritation was evident.

Genetic toxicity: In five *in vitro* bacterial (*Salmonella*) mutagenicity studies, AO shows no evidence of mutagenicity either with or without S9 metabolic activation at concentrations up to 250 ug/plate (higher concentrations caused cytotoxicity).

Three *in vivo* studies investigated clastogenic effects on a close structural analog of the category, 1-(methylododecyl)dimethylamine-N-oxide including: a mouse micronucleus, a Chinese hamster micronucleus and a Chinese hamster cytogenetics study. These studies were all negative showing no increase in micronuclei or chromosome aberrations. An *in vivo* mouse dominant lethal assay showed no evidence of heritable effects. Two AOs (CAS No 1643-20-5 and CAS No 3332-27-2) were negative in an *in vitro* cell transformation assay tested at concentrations up to 20 ug/ml.

Carcinogenicity: The carcinogenic potential of amine oxides has been thoroughly investigated in three carcinogenicity studies in rats or mice by dermal, dietary, or drinking water routes. In all cases the substances demonstrated no evidence of a carcinogenic response.

Reproductive and developmental toxicity: No evidence of reproductive toxicity or fertility effects was observed in a study in which rats were given dietary doses of AO in the diet over two generations (CAS No 1643-20-5). No macroscopic or histopathological changes were attributable to treatment with the test substance. The maternal NOEL from this reproductive study was >40 mg AO/kg bw/day, which was the highest dose tested. At all treatment levels, the rate of bodyweight gain for the F1 and F2 offspring was reduced during the lactation period, however, this reduction was not greater than 10%. This effect appeared to be dose-related, but was not statistically significant until after weaning in the mid and high dose levels. This was not considered an adverse effect since the body weight change only reached statistical significance when the rat pups were getting the majority of their calories from solid food (Developmental NOEL >40 mg/kg bw/day).

In three developmental toxicity studies via gavage in rats and rabbits (with CAS No 1643-20-5 & 70592-80-2), effects such as decreased foetal weight or delayed ossification, were most often observed only at maternally toxic doses and were associated with the irritation effects of AO on the gastrointestinal tract. No decreases in litter size, no changes in litter parameters, no malformations or significant differences in skeletal defects were observed at oral doses up to 25 mg/kg bw/day in rats (based on decreased foetal weight at 100 mg/kg bw/day) and >160 mg/kg bw/day in rabbits (the highest dose tested).

COCAMIDOPROPYLBETAINE

* [Van Waters and Rogers] ** [Canada Colors and Chemicals Ltd.] Toxicokinetics, metabolism and distribution. Absorption of the chemical across dermal and gastrointestinal membranes is possible based on the relatively low molecular weight of the chemical (500 Da) and given that it is a surfactant (EC, 2003). Acute toxicity. Acute oral toxicity studies in rats and mice indicated that the LD50 values of the chemical (at 30-35.61% concentration) ranged from 1800 mg/kg bw (male rats) up to 5000 mg/kg bw, with mortalities noted in most studies (CIR, 2010). Of note is an acute oral toxicity study conducted in Sprague-Dawley rats (5/sex) at a single dose of 1800 mg/kg bw (formulation containing 35.61% of the chemical), where no males but all five females died. Overall, the data suggests that mortality occurs following oral administration of the chemical and that it may be an acute oral toxicant. Therefore, based on these data the chemical may be harmful if swallowed. An acute dermal toxicity study in rats was conducted using 2000 mg/kg bw of a 31% formulation of the chemical (CIR, 2010). Irritation was observed, but there were no clinical signs of systemic toxicity or mortalities. The lack of effects in this study suggests that the chemical is likely to be of low acute dermal toxicity. Irritation. The chemical has a quaternary ammonium functional group, which is a structural alert for corrosion. Numerous skin irritation studies, conducted with formulations containing 7.5-30% of the chemical, indicated that the chemical has irritant properties. The studies were, in-general, conducted under occlusive conditions, with exposure times of up to 24 hours (7.5-10%). Based on the information available, the chemical is likely to be a skin irritant. Eye irritation studies with the chemical showed that corrosive and necrotic effects occurred at 30% whereas less severe effects were observed at lower concentrations of 2.3-10%. The chemical is classified with the risk phrase R36: Irritating to eyes, however, based on studies conducted on the chemical it may be a severe eye irritant. Sensitisation. The chemical has a quaternary ammonium functional group, which is a structural alert for sensitisation (Conflicting results have been obtained with the chemical in animal studies. Positive results were reported in an LLNA study (an EC3 value was not reported). In addition, positive results were obtained in two guinea pig maximisation studies conducted by a single laboratory, the first at 3% induction and 3% challenge, and the second at 0.15% induction and 0.015% challenge. However, there was no sensitisation in a guinea pig maximisation test when the chemical was tested at 6% induction and 1% challenge. In addition, no sensitisation was observed in another test in guinea pigs at 0.75% induction and 0.02% challenge. No evidence of sensitisation was reported in a HRIPT on a formulation containing the chemical at 0.6% concentration (a 10% dilution of a ~6% formulation) with 110 volunteers. In HRIPT studies on formulations containing the chemical, no evidence of sensitisation was reported at concentrations of 1.87% (88 subjects), 0.93% (93 subjects), 0.3% (100 subjects), 1.5-3.0% (141 subjects), 6.0% (210 subjects), 0.018% (27 subjects). However, positive results were observed in provocative studies conducted on formulations containing the chemical (at 0.3-1% concentration), conducted in subjects diagnosed with various forms of contact dermatitis, suggesting that the chemical may cause reactions in sensitive individuals. In one study authors note that sensitisation effects of the chemical (and related compounds) are most likely due to the impurities, including DMAPA and amidopropyl dimethylamines, however, they do not exclude the possibility of the causing the sensitisation. The potential for skin sensitisation, due to the presence of the above impurities in the chemical, will be limited by their reported low concentration. In summary, a definitive conclusion cannot be made on the skin sensitisation potential of the chemical. The available information suggests that skin sensitisation is possible. Although there are some inconsistencies in the results reported for studies conducted on the chemical, the scientific data points towards the positive findings being caused by impurities, in particular DMAPA and amidopropyl dimethylamines, which are present in the chemical at low concentrations. Repeated Dose Toxicity. In a 28-day repeated dose oral toxicity study, rats were administered a 30.6% solution of the chemical at 0, 100, 500 or 1000 mg/kg bw/day. Inflammation of the non-glandular stomach was noted in animals of the high-dose group, although this effect was attributed to the irritant properties of the test material. Mortality was also observed in this study at all treatment levels but there was no dose-response relationship. In another 28-day repeated dose oral toxicity study, rats were administered a solution containing the chemical (concentration not stated) at 0, 250, 500 or 1000 mg/kg bw/day. The NOEL was reported as 500 mg/kg bw/day, which appears to be based on non-systemic irritant effects on the non-glandular stomach. No mortalities were observed. In a 90-day repeated dose oral toxicity study, rats were administered a solution containing the chemical (concentration not stated) at 0, 250, 500 or 1000 mg/kg bw/day. There were no mortalities and the noted effects are isolated to the stomach region and appear to be irritant in nature. The NOEL established by the study authors was 250 mg/kg bw/day, based on these effects. Mutagenicity. The chemical was not mutagenic in numerous bacterial reverse mutation assays. Negative results were also obtained for the chemical in a mouse lymphoma test and a micronucleus test in mice. Carcinogenicity. No signs of carcinogenicity were noted in a 20 month dermal study in mice (3 applications/week) for a hair dye formulation containing the chemical at a concentration of 0.09%. The formation of nitrosamines is possible. Secondary amides (and the identified impurities) may serve as substrates for N-nitrosation, therefore formulation with N-nitrosating agents should be avoided. Possible cross-reactions to several fatty acid amidopropyl dimethylamines were observed in patients that were reported to have allergic contact dermatitis to a baby lotion that contained 0.3% oleamidopropyl dimethylamine. Stearamidopropyl dimethylamine at 2% in hair conditioners was not a contact sensitizer when tested neat or diluted to 30%. However, irritation reactions were observed. A 10-year retrospective study found that out of 46 patients with confirmed allergic eyelid dermatitis, 10.9% had relevant reactions to oleamidopropyl dimethylamine and 4.3% had relevant reactions to cocamidopropyl dimethylamine. Several cases of allergic contact dermatitis were reported in patients from the Netherlands that had used a particular type of body lotion that contained oleamidopropyl dimethylamine. In 12 patients tested with their personal cosmetics, containing the fatty acid amidopropyl dimethylamine cocamidopropyl betaine (CAPB), 9 had positive reactions to at least one dilution and 5 had irritant reactions. All except 3 patients, who were not tested, had 2 or 3+ reaction to the 3,3-dimethylaminopropylamine (DMAPA, the reactant used in producing fatty acid amidopropyl dimethylamines) at concentrations as low as 0.05%. The presence of DMAPA was investigated via thin-layer chromatography in the personal cosmetics of 4 of the patients that had positive reactions. DMAPA was measured in the products at 50 - 150 ppm suggesting that the sensitising agent in CAPB-induced allergy is DMAPA. The sensitisation potential of a 4% aqueous liquid fabric softener formulation containing 0.5% stearyl/palmitylamidopropyl dimethylamine was investigated using. The test material caused some irritation in most volunteers. After a rest period of 2 weeks, the subjects received challenge patches with the same concentration of test material on both arms. Patch sites were graded 48 and 96 h after patching. Eight subjects reacted at challenge, and 7 of the eight submitted to rechallenge with 4% and 0.4% aqueous formulations. No reactions indicative of sensitisation occurred at rechallenge. The test formulation containing stearyl/palmitylamidopropyl dimethylamine had no significant sensitisation potential.subjects.

Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41.

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.

For betaines:

Several sources revealing data on skin irritation, skin sensitisation and dermal absorption in humans are available for CAS 683-10-3, the C12-alkyldimethyl betaine, which is the most frequently occurring betaine because it is one of the components of most of the substances of the alkyldimethyl betaine group, among those also Betaines, C12-14 (even numbered)-alkyldimethyl. Therefore, read-across of exposure-related observations in humans from CAS 683-10-3 is justified. Data from several human closed patch tests demonstrate skin irritation in humans ranging from mild to strong under occlusive conditions even with concentrations as low as 1%. In contrast, exposure to 0.1% under open conditions did not induce any positive skin reactions.

Information on skin sensitisation in humans is available from a closed human patch test with CAS 683-10-3. After an induction period of 6 days with 0.1% of active ingredient, followed by a treatment-free period of 10 days, the volunteers were challenged under occlusive conditions for 24 hours. No reactions were observed immediately after challenge; during the next 4 days only irritation reactions were observed.

This finding was further supported by industrial medical monitoring data. Workers involved in the production of CAS 683-10-3 are routinely checked every 3 years for signs of skin sensitisation, respiratory irritation, skin irritation and eye irritation. During these examinations no signs of the aforementioned disorders were observed which were related to the test substance.

Moreover, a study focusing on dermal uptake of C12-alkyldimethyl betaine) into human skin and the effects of surfactants on skin barrier function demonstrated that only up to 0.4% of the applied dose was absorbed within 30 minutes of exposure, with absorbance being dependent on the concentration applied. Tape stripping of the skin revealed that the administered test substance was primarily located in the outer stratum corneum layer

Test material does not demonstrate mutagenic or clastogenic effects in bacteria or mammalian cells in vitro

" REACH Dossier

Amphoteric surfactants are easily absorbed in the intestine and are excreted partly unchanged via the faeces. Metabolisation to CO₂ and short-chained fatty acids also occur. No tendency to accumulation in the organism or storage of betaines in certain organs has been detected. Betaines generally have a low acute toxicity. E.g., LD50 values for cocoamidopropylbetaine (30% solution) by oral administration have been determined to 4,910 mg/kg body weight in rats.

Betaines do not carry any net charge, and, therefore, they can only form hydrophobic bonds with proteins in the skin. This may be the explanation for the low protein denaturation potential of betaines as the ion-binding of other surfactants contributes to denaturation. In combination with anionic surfactants a positive synergistic effect with regard to skin compatibility is often found. Compared to a 20% solution of C12 alkyl sulfate (AS; sodium lauryl sulfate) alone, decreased erythema was observed for the combination of 20% C12 AS and 10% cocoamidopropyl betaine one hour after the removal of patches. The combination of cocoamidopropyl betaine and C12 AS also reduced swelling of the skin, and generally interactions between amphoteric and AS produce less swelling and result in milder skin reactions. Concentrated betaines are expected to be irritant to skin and eyes. Diluted solutions (3-10%) are not irritant to skin, but they are mildly irritant to the eyes (4.5%)

No evidence of delayed contact hypersensitivity was found in guinea pigs after topically administered solutions of 10% cocoamidopropyl betaine by using the Magnusson-Kligman maximization test. Various instances of contact allergy to cocoamidopropyl betaine have been reported. In all of the reports it was concluded that the observed skin reactions were due to the presence of 3-dimethylaminopropylamine which is an impurity in cocoamidopropyl betaine. This impurity is an intermediate in the synthesis of alkylamidopropyl dimethylamines that are intermediates in the synthesis of the corresponding alkylamido betaines.

Cocoamidopropyl betaine was proven to be non-mutagenic to Salmonella typhimurium in the Ames Salmonella/microsome reverse mutation assay. Short-term genotoxicity tests have shown negative results of mutagenicity for lauryl betaine in various strains of Salmonella typhimurium.

NONYLPHENOL, ETHOXYLATED

For nonylphenol and its compounds:

Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor),. Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogenous hormone for binding with the estrogen receptors ERalpha and ERbeta.

Effects in pregnant women.

Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These "low doses" ranged from 10-13-10-9 M, which is lower than what is generally found in the environment.

Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications.

Effects on metabolism

Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats.

Cancer

Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease

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 autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing.

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

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

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

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

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

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

>20 EO is not classified (CESIO 2000)

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

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

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

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein *in vitro* and their effect on the skin.

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

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

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

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

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

of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

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

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

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

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

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

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

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

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

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

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

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGME 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.

for nonylphenol:

Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.

Nonylphenol was not mutagenic to *Salmonella typhimurium*, TA100, TA1535, TA98, TA1537 and *Escherichia coli* WP2 uvrA, with or without an exogenous metabolic activation system.

Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.

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.

FORMALDEHYDE.

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

SODIUM LAURYL ETHER SULFATE & WATER

No significant acute toxicological data identified in literature search.

SODIUM LAURYL ETHER SULFATE & NONYLPHENOL, 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 autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

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

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

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

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

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

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

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

SODIUM LAURYL ETHER SULFATE & COCAMIDOPROPYL BETAINE

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

NV Chemicals Hi-Foam Car Wash

LAURYL DIMETHYLAMINE OXIDE & NONYLPHENOL, ETHOXYLATED & FORMALDEHYDE.	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
LAURYL DIMETHYLAMINE OXIDE & FORMALDEHYDE.	The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
COCAMIDOPROPYL BETAINE & FORMALDEHYDE.	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

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

NV Chemicals Hi-Foam Car Wash	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
sodium lauryl ether sulfate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Fish	0.26mg/L	5
	EC50	48h	Crustacea	2.43-4.01mg/l	4
lauryldimethylamine oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	2.4mg/l	2
	EC50	72h	Algae or other aquatic plants	0.015mg/l	2
	EC50	48h	Crustacea	2.9mg/l	2
	EC10(ECx)	72h	Algae or other aquatic plants	0.002mg/l	2
cocamidopropylbetaine	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	1-10mg/l	Not Available
	EC0(ECx)	96h	Algae or other aquatic plants	0.09mg/l	1
	EC50	72h	Algae or other aquatic plants	1-10mg/l	1
	EC50	96h	Algae or other aquatic plants	0.55mg/l	1
	EC50	48h	Crustacea	6.5mg/l	1
nonylphenol, ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<0.2	7
	EC50(ECx)	48h	Crustacea	86mg/l	Not Available
	EC50	96h	Algae or other aquatic plants	12mg/l	4
	EC50	48h	Crustacea	86mg/l	Not Available
	LC50	96h	Fish	1-1.8mg/L	4
formaldehyde.	Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	96h	Algae or other aquatic plants	0.005mg/l	4	

Continued...

NV Chemicals Hi-Foam Car Wash

	EC50	72h	Algae or other aquatic plants	1.034-1.984mg/l	4
	EC50	96h	Algae or other aquatic plants	0.375-0.579mg/l	4
	EC50	48h	Crustacea	3.26mg/l	4
	LC50	96h	Fish	0.727-9.193mg/L	4
water	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
lauryldimethylamine oxide	LOW	LOW
nonylphenol, ethoxylated	LOW	LOW
formaldehyde.	LOW (Half-life = 14 days)	LOW (Half-life = 2.97 days)
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
lauryldimethylamine oxide	HIGH (LogKOW = 4.673)
nonylphenol, ethoxylated	LOW (BCF = 16)
formaldehyde.	LOW (LogKOW = 0.35)

Mobility in soil

Ingredient	Mobility
lauryldimethylamine oxide	LOW (Log KOC = 18660)
nonylphenol, ethoxylated	LOW (Log KOC = 940)
formaldehyde.	HIGH (Log KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
sodium lauryl ether sulfate	Not Available
lauryldimethylamine oxide	Not Available
cocamidopropylbetaine	Not Available
nonylphenol, ethoxylated	Not Available
formaldehyde.	Not Available
water	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sodium lauryl ether sulfate	Not Available
lauryldimethylamine oxide	Not Available
cocamidopropylbetaine	Not Available
nonylphenol, ethoxylated	Not Available
formaldehyde.	Not Available
water	Not Available

SECTION 15 Regulatory information

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

sodium lauryl ether sulfate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

lauryldimethylamine oxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

cocamidopropylbetaine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

nonylphenol, ethoxylated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

formaldehyde. is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDLS	No (sodium lauryl ether sulfate; lauryldimethylamine oxide; cocamidopropylbetaine; nonylphenol, ethoxylated; formaldehyde.; water)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes

NV Chemicals Hi-Foam Car Wash

National Inventory	Status
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium lauryl ether sulfate; lauryldimethylamine oxide)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	01/11/2009

SDS Version Summary

Version	Date of Update	Sections Updated
5.1	23/12/2022	Classification review due to GHS Revision change.
6.1	10/03/2023	Classification change due to full database hazard calculation/update.

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
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration

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

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