



NV Chemicals Auto Scrub

N.V. Chemicals (Aust) P/L

Chemwatch Hazard Alert Code: 2

Chemwatch: 5231-84

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 Auto Scrub
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

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

Registered company name	N.V. Chemicals (Aust) P/L
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		0 = Minimum 1 = Low 2 = Moderate 3 = High 4 = Extreme
Reactivity	0		
Chronic	2		

Poisons Schedule	Not Applicable
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
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	Warning
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Hazard statement(s)

H319	Causes serious eye irritation.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H402	Harmful to aquatic life.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.

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
9016-45-9	<10	<u>nonylphenol ethoxylates</u>
10213-79-3	<10	<u>sodium metasilicate, pentahydrate</u>
111-76-2	<10	<u>ethylene glycol monobutyl ether</u>
Not Available	<10	Ingredients determined not to be hazardous
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.

Indication of any immediate medical attention and special treatment needed

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Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

- ▶ There is no restriction on the type of extinguisher which may be used.
- ▶ Use extinguishing media suitable for surrounding area.

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	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered to be a significant fire risk. ▶ Expansion or decomposition on heating may lead to violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <p>Decomposes on heating and produces toxic fumes of: carbon dioxide (CO₂) phosphorus oxides (PO_x)</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	<p>Slippery when spilt.</p> <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>Slippery when spilt. Minor hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Control personal contact with the substance, by using protective equipment as required. ▶ Prevent spillage from entering drains or water ways. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▶ Wash area and prevent runoff into drains or waterways. ▶ 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"> ▶ Limit all unnecessary personal contact. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ When handling DO NOT eat, drink or smoke. ▶ Always wash hands with soap and water after handling. ▶ Avoid physical damage to containers. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
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.
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	▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m3	242 mg/m3 / 50 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
nonylphenol ethoxylates	43 mg/m3	470 mg/m3	5,400 mg/m3
sodium metasilicate, pentahydrate	6.6 mg/m3	73 mg/m3	440 mg/m3
sodium metasilicate, pentahydrate	3.8 mg/m3	42 mg/m3	250 mg/m3
ethylene glycol monobutyl ether	60 ppm	120 ppm	700 ppm

Ingredient	Original IDLH	Revised IDLH
nonylphenol ethoxylates	Not Available	Not Available
sodium metasilicate, pentahydrate	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available
water	Not Available	Not Available

Occupational Exposure Banding


Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
nonylphenol ethoxylates	E	≤ 0.1 ppm
sodium metasilicate, pentahydrate	E	≤ 0.01 mg/m ³

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.

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
Individual protection measures, such as personal protective equipment	
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields; or as required, ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ Eyewash 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:

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Material	CPI
BUTYL	A
NEOPRENE	B

Respiratory protection

Type A-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	A-AUS P2	-	A-PAPR-AUS / Class 1 P2

Continued...

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NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
VITON	C

up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-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)

* 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.

Ansell Glove Selection

Glove — In order of recommendation
AlphaTec® Solvex® 37-185
AlphaTec® 38-612
AlphaTec® 58-008
AlphaTec® 58-735
AlphaTec® Solvex® 37-675
AlphaTec® 58-530B
AlphaTec® 58-530W
AlphaTec® 79-700
DermaShield™ 73-711
MICROFLEX® 63-864

The suggested gloves for use should be confirmed with the glove supplier.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Orange liquid with musk fragrance; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Applicable
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	2.3 @ 20 C	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
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SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Not normally a hazard due to non-volatile nature of product
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Ingestion may result in nausea, abdominal irritation, pain and vomiting
Skin Contact	Limited 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. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	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.
Chronic	Long-term exposure to the product is not thought to produce chronic effects adverse to health (as classified by EC Directives using animal models); nevertheless exposure by all routes should be minimised as a matter of course.

NV Chemicals Auto Scrub	TOXICITY	IRRITATION
	Not Available	Not Available
nonylphenol ethoxylates	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2943.2 mg/kg ^[2] Oral (Rat) LD50: 1310 mg/kg ^[2]	Eye (rabbit): 5 mg SEVERE Skin (human): 15 mg/3D mild
sodium metasilicate, pentahydrate	TOXICITY	IRRITATION
	Oral (Rat) LD50: 1153 mg/kg ^[2]	Skin (human): 250 mg/24h SEVERE Skin (rabbit): 250 mg/24h SEVERE
ethylene glycol monobutyl ether	TOXICITY	IRRITATION
	Dermal (Guinea Pig) LD50: 210 mg/kg ^[2] Inhalation (Rat) LC50: 450 ppm/4h ^[2]	Eye (rabbit): 100 mg SEVERE * [Union Carbide] Eye (rabbit): 100 mg/24h-moderate
	Oral (Rat) LD50: 250 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1] Skin (rabbit): 500 mg, open; mild
		Skin: adverse effect observed (irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
water	TOXICITY	IRRITATION
	Oral (Rat) LD50: >90000 mg/kg ^[2]	Not Available

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

NONYLPHENOL ETHOXYLATES

Oral (rat) TDL0: 150 mg/kg/3D-I Skin (rabbit): 500 mg mild

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

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>

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

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

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

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

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

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

>20 EO is not classified (CESIO 2000)

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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	<p>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.</p> <p>Nonylphenol was not mutagenic to <i>Salmonella typhimurium</i>, TA100, TA1535, TA98, TA1537 and <i>Escherichia coli</i> WP2 uvrA, with or without an exogenous metabolic activation system.</p> <p>Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.</p>
<p>SODIUM METASILICATE, PENTAHYDRATE</p>	<p>sodium metasilicate anhydrous:</p> <p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.</p> <p>Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).</p> <p>The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.</p>
<p>ETHYLENE GLYCOL MONOBUTYL ETHER</p>	<p>NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS</p> <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):</p> <p>Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.</p> <p>EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.</p> <p>Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitizers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE <i>in vitro</i> than those of rats.</p> <p>Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA <i>in vitro</i> and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA <i>in vitro</i>.</p> <p>Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in <i>S. typhimurium</i> strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. <i>In vitro</i> cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and <i>in vivo</i> micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.</p> <p>Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity</p> <p>Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).</p> <p>Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.</p> <p>The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE). Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species. At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.</p> <p>Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.</p> <p>Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.</p> <p>1: NTP Toxicology Program Technical report Series 484, March 2000.</p>
<p>WATER</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>NONYLPHENOL ETHOXYLATES & SODIUM METASILICATE, PENTAHYDRATE</p>	<p>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</p>

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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.

**SODIUM METASILICATE,
PENTAHYDRATE &
ETHYLENE GLYCOL
MONOBUTYL ETHER**

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.

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

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

SECTION 12 Ecological information

Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
NV Chemicals Auto Scrub	Not Available	Not Available	Not Available	Not Available	Not Available
nonylphenol ethoxylates	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
sodium metasilicate, pentahydrate	LC50	96h	Fish	180mg/l	1
	EC50(ECx)	48h	Crustacea	22.94-49.01mg/l	4
	EC50	72h	Algae or other aquatic plants	207mg/l	2
	EC50	48h	Crustacea	22.94-49.01mg/l	4
ethylene glycol monobutyl ether	LC50	96h	Fish	1700mg/l	Not Available
	EC50	48h	Crustacea	164mg/l	2
	EC50	72h	Algae or other aquatic plants	623mg/l	2
	EC10(ECx)	48h	Crustacea	7.2mg/l	2
	EC50	96h	Algae or other aquatic plants	720mg/l	2
water	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
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
nonylphenol ethoxylates	LOW (BCF = 1.4)
ethylene glycol monobutyl ether	LOW (BCF = 2.51)

Mobility in soil

Continued...

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (Log KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ Bury residue in an authorised landfill. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	
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
nonylphenol ethoxylates	Not Available
sodium metasilicate, pentahydrate	Not Available
ethylene glycol monobutyl ether	Not Available
water	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
nonylphenol ethoxylates	Not Available
sodium metasilicate, pentahydrate	Not Available
ethylene glycol monobutyl ether	Not Available
water	Not Available

SECTION 15 Regulatory information

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

nonylphenol ethoxylates is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

sodium metasilicate, pentahydrate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

ethylene glycol monobutyl ether 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 6

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

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

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	08/12/2016

SDS Version Summary

Version	Date of Update	Sections Updated
5.1	20/08/2021	Classification change due to full database hazard calculation/update.
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

- ▶ AIIC: 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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