Miteq Cleaner 305

Miteg P/L

Mited P/L	Chemwatch Hazard Alert Code: 1
Chemwatch: 4842-99	Issue Date: 12/09/2019
Version No: 4.1.1.1	Print Date: 17/09/2019
Safety Data Sheet according to WHS and ADG requirements	S.GHS.AUS.EN

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

Product Identifier

Product name	Miteq Cleaner 305
Synonyms	Rinse Free Liquid Detergent.
Other means of identification	Not Available

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

Relevant identified uses	A premium quality biodegradable floor cleaner.

Details of the supplier of the safety data sheet

Registered company name	Miteq P/L
Address	Unit 1, 45-49 Popes Road Keysborough VIC 3173 Australia
Telephone	(03) 9532 5747
Fax	Not Available
Website	www.miteq.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	Not Available
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification	Not Applicable
Label elements	
Hazard pictogram(s)	Not Applicable
	-
SIGNAL WORD	NOT APPLICABLE

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
111-76-2	5-10	ethylene glycol monobutyl ether
Not Available	5-10	surfactants proprietary
Not Available	1-5	wetting agents
8002-09-3	<2	pine oil
Not Available	>60	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	 If this product comes in contact with the eyes: 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	 If skin contact occurs: 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	 If fumes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
Ingestion	 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

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	
Advice for firefighters		
Fire Fighting	 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	 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. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO2) sulfur oxides (SOx)
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	 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. Slippery when spilt.
Major Spills	 Slippery when spilt. Minor hazard. 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	 DO NOT allow clothing wet with material to stay in contact with skin 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	 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

	 Polyethylene or polypropylene container.
Suitable container	Packing as recommended by manufacturer.

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Storage incompatibility

Avoid reaction with oxidising agents

Check all containers are clearly labelled and free from leaks.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

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

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
Ingredient	Original IDLH	Revised IDLH		
ethylene glycol monobutyl ether	700 ppm	Not Available		
pine oil	Not Available	Not Available		

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
Personal protection	
Eye and face protection	 Safety glasses with side shields. Chemical goggles. Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	 Wear chemical protective gloves, e.g. PVC. Wear safety footwear or safety gumboots, e.g. Rubber
Body protection	See Other protection below
Other protection	 ► Overalls. ► Eyewash unit.

Respiratory protection

Type A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Clear, blue thin liquid with pine fragrance; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	0.98
Odour	Not Available	Partition coefficient n-octanol / water	Not Available

Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	8.0-8.5	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	88 (approx.)	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	7.0-7.5 (1:80)
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	 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	Acute effects from inhalation of high vapour concentrations may be chest and nasal irritation with coughing, sneezing, headache and even nausea. Inhalation of vapour is more likely at higher than normal temperatures.		
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Ingestion may result in nausea, abdominal irritation, pain and diarrhoea		
Skin Contact	There is some evidence to suggest that this material can o	cause inflammation of the skin on contact in some persons.	
Eye	There is some evidence to suggest that this material can c	ause eye irritation and damage in some persons.	
Chronic	Long-term exposure to the product is not thought to produ Directives using animal models); nevertheless exposure b	ce chronic effects adverse to the health (as classified by EC y all routes should be minimised as a matter of course.	
	TOXICITY	IRRITATION	
Miteq Cleaner 305	Not Available	Not Available	
	тохісіту	IRRITATION	
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE	
	Inhalation (rat) LC50: 449.48655 mg/l/4H ^[2]	Eye (rabbit): 100 mg/24h-moderate	
ethylene glycol monobutyl ether	Oral (rat) LD50: 250 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]	
e.lobutyr ether		Skin (rabbit): 500 mg, open; mild	
		Skin: adverse effect observed (irritating) ^[1]	

Skin: no adverse effect observed (not irritating)^[1]

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	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Skin (rabbit): 500 mg/24h-SEVERE
pine oil	Inhalation (rat) LC50: >3.79 mg/l/4H ^[2]	
	Oral (rat) LD50: 2100 mg/kg ^[2]	
Legend:	1. Value obtained from Europe ECHA Registered Substances - Unless otherwise specified data extracted from RTECS - Regi	- Acute toxicity 2.* Value obtained from manufacturer's SDS. ister of Toxic Effect of chemical Substances
ETHYLENE GLYCOL MONOBUTYL ETHER	NOTE: Changes in kidney, liver, spleen and lungs are observed substance by all routes. ** ASCC (NZ) SDS The material may produce source irritation to the eye causing to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or reper swelling, the production of vesicles, scaling and thickening of For ethylene glycol hoxal ethics and their acetates (EGM Typical members of this category are ethylene glycol propyler ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme <i>i</i> alcohols to aldehydes (which are transient metabolites). Furth dehydrogenase produces alkoxyacetic acids, which are the pr ethers. Acute Toxicity: Oral LD50 values in rats for all category mem values increasing with decreasing molecular weight. Four to s these chemicals in rats at the highest vapour concentrations j mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA was observed for any of these materials under these conditio (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category m toxicity. All category members cause reversible irritation to sk irritating than the other category members. EGPE and EGBE a of acute toxicity in rats, mice and rabbits are consistent with 1 CNS depression typical of organic solvents in general. Alkoxy butoxyacetic acid (BAA), are responsible for the red blood ce ingesting cleaning fluids containing 9-22% EGBE are similar t decreased blood haemoglobin and/or haemoglobinuria were o was due to haemolysis or haemodilution as a result of admini are many-fold more resistant to toxicity from EGPE and EGB Repeat dose toxicity: The fact that the NOAEL for repeated consistent with red blood cells being more sensitive to EGBE baboons were sensitive to the effects of BAA <i>in vitro</i> and dis (increased haematocrit and mean corpuscular hemoglobin), f and guinea pigs was less sensitive to haemolysis by BAA <i>in</i> on disgnificant increase in the incidence of liver haemangiosarcon female mice. It was decided that based on the mode of ac	ed in animals exposed to high concentrations of this pronounced inflammation. Repeated or prolonged exposure ated exposure and may produce on contact skin redness, if the skin. AAEs): ne ether (EGPE), ethylene glycol butyl ether (EGBE) and ADH-3, which catalyzes the conversion of their terminal ner, rapid conversion of the aldehydes by aldehyde redominant urinary metabolites of mono substituted glycol abers range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with six hour acute inhalation toxicity studies were conducted for practically achievable. Values range from LCO > 85 ppm (50 V to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality ons. Dermal LD50 values in rabbits range from 435 mg/kg bw members can be considered to be of low to moderate acute kin and eyes, with EGBEA less irritating and EGHE more are not sensitisers in experimental animals or humans. Signs haemolysis (with the exception of EGHE) and non-specific yacetic acid metabolites, propoxyacetic acid (PAA) and all hemolysis. Signs of toxicity in humans deliberately to those of rats, with the exception of haemolysis. Although observed in some of the human cases, it is not clear if this istration of large volumes of fluid. Red blood cells of human 8E <i>in vitro</i> than those of rats. 1 dose toxicity of EGBE is less than that of EGPE is than EGPE. Blood from mice, rats, hamsters, rabbits and played similar responses, which included erythrocyte swellin ollowed by hemolysis. Blood from humans, pigs, dogs, cats, <i>vitro</i> . ivation, EGBE tested negative for mutagenicity in Ames TA1535 and TA1537 and EGHE tested negative in strains ity and sister chromatid exchange assays with EGBE and abolic activation and in vivo micronucleus tests with EGBE is ris are not genotoxic. carcinogenicity study with EGBE in rats and mice a mas was seen in male mice and forestomach tumours in n data available, there was no significant hazard for human eproductive and developmental toxicity studies indicate that the reproductive system or developing fetus, developmenta toxicity studies

Ethylene glycol is quickly and extensively absorbed throughout the gastrointestinal tract. Limited information suggests that it is also absorbed through the airways; absorption through skin is apparently slow. Following absorption, it is

distributed throughout the body. In humans, it is initially metabolized by alcohol dehydrogenase to form glycoaldehyde, which is rapidly converted to glycolic acid and glycxal. These breakdown products are oxidized to glycxylate, which may be further metabolized to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate carbon dioxide, which is one of the major elimination products of ethylene glycol. In addition to exhaled carbon dioxide, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination is rapid and occurs within a few hours. Respiratory effects: Respiratory system involvement occurs 12-24 hours after swallowing sufficient amounts of ethylene glycol. Symptoms include hyperventilation, shallow rapid breathing, and generalized swelling of the lungs with calcium oxalate deposits occasionally appearing in the lungs. Respiratory system involvement appears to be dose-dependent and occurs at the same time as cardiovascular changes. Later, there may be other changes compatible with adult respiratory distress syndrome (ARDS). Swelling of the lung can be a result of heart failure, ARDS, or aspiration of stomach contents. Symptoms related to acidosis such as fast or excessive breathing are frequently observed; however, major symptoms such as swelling of the lung and inflammation of the bronchi and lungs are relatively rare, and are usually seen only in extreme poisoning. Cardiovascular effects: Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of ethylene glycol poisoning by swallowing, which is 12-24 hours after acute exposure. The symptoms of poisoning involving the heart include increased heart rate, heart enlargement and ventricular gallop. There may also be high or low blood pressure, which may progress to cardiogenic shock. In lethal cases, inflammation of the heart muscle has been observed at autopsy. Cardiovascular involvement appears to be rare and usually seen after swallowing higher doses of ethylene glycol. In summary, acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown. Gastrointestinal effects: Common early acute effects of swallowing ethylene glycol include nausea, vomiting with or without blood, heartburn and abdominal cramping and pain. One patient showed intermittent diarrhea and pain, and after surgery, deposition of oxalate crystals was shown to have occurred. Musculoskeletal effects: Reported musculoskeletal effects in cases of acute ethylene glycol poisoning include diffuse muscle tenderness and pain, associated with high levels of creatinine in the blood, and jerks and contractions associated with low calcium. Liver effects: Autopsies carried out on people who died following acute ethylene glycol poisoning showed deposition of calcium oxalate in the liver as well as hydropic and fatty degeneration and cell death (necrosis) of the liver. Kidney effects: Adverse kidney effects are seen during the third stage of ethylene glycol poisoning, 2-3 days after acute exposure. Calcium oxalate crystals are deposited in the tubules and are seen in the urine. There may also be degeneration and death of tubule cells, and inflammation of the tubule interstitium. If untreated, the degree of kidney damage progresses and leads to blood and protein in the urine, decreased kidney function, reduction in urine output and ultimately, kidney failure. With adequate supportive therapy, kidney function can return to normal or near normal. Metabolic effects: Metabolic changes can occur within 12 hours of exposure to ethylene glycol. There may be metabolic acidosis, caused by accumulation of glycolic acid in the blood and therefore a reduction in blood pH. The anion gap is increased, due to increased unmeasured anions (mainly glycolate). Effects on the nervous system: Adverse reactions involving the nervous system are among the first symptoms to appear in humans after ethylene glycol is swallowed. These early effects are also the only symptoms caused by unmetabolised ethylene glycol. Together with metabolic effects (see above), they occur from 0.5-12 hours after exposure and are considered to be part of the first stage in ethylene glycol poisoning. Inco-ordination, slurred speech, confusion and sleepiness are common in the early stages, as are irritation, restlessness and disorientation. Later, there may be effects on cranial nerves (which may be reversible over many months). Swelling of the brain (cerebrum) and crystal deposits of calcium oxalate in the walls of the small blood vessels of the brain were found at autopsy in people who died after acute ethylene alvcol poisonina. Reproductive effects: Animal testing showed that ethylene glycol may affect fertility, survival of fetuses and the male reproductive organs. Effects on development: Animal studies indicate that birth defects may occur after exposure in pregnancy; there may also be reduction in foetal weight. Cancer: No studies are known regarding cancer effects in humans or animal, after skin exposure to ethylene glycol. Genetic toxicity: No human studies available, but animal testing results are consistently negative. 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. No significant acute toxicological data identified in literature search. d-Limonene is readily absorbed by inhalation and swallowing. Absorption through the skin is reported to the lower than by PINE OIL inhalation. It is rapidly distributed to different tissues in the body, readily metabolized and eliminated, primary through the urine. Limonene shows low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data is available on the potential to cause eye and airway irritation. Autooxidised products of d-limonene have the potential to sensitise the skin. Limited data is available on the potential to cause respiratory sensitization in humans. Limonene will automatically oxidize in the presence of light in air, forming a variety of oxygenated monocyclic terpenes. When contact with these oxidation products occurs, the risk of skin sensitization is high. Limonene does not cause genetic toxicity of birth defects, and it is not toxic to the reproductive system.

Camphor appears to have moderate acute oral toxicity, and a higher toxicity when inhaled. Long term inhalation may cause emphysema. There is no observed tumour potential. Reproductive toxicity studies were not available for camphor, however, in developmental toxicity studies, it demonstrated no foetal toxicity.

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The terpenoid hydrocarbons are found in needle trees and deciduous plants. This category of chemicals shows very low acute toxicity. They are ecreted in the urine. They are unlikely to cause genetic damage, but animal testing shows that they do cause increased rates of kidney cancer. They have low potential to cause reproductive and developmental toxicity.

For terpenoid tertiary alcohols and their related esters:

These substances are metabolised in the liver and excreted primarily in the urine and faeces. A portion is also excreted unchanged. They have low short term toxicity when ingested or applied on the skin. However, repeated and long term use may cause dose dependent harm to both the foetus and mother.

Adverse reactions to fragrances in perfumes and fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, sensitivity to light, immediate contact reactions, and pigmented contact dermatitis. Airborne and connubial contact dermatitis occurs. Contact allergy is a lifelong condition, so symptoms may occur on re-exposure. Allergic contact dermatitis can be severe and widespread, with significant impairment of quality of life and potential consequences for fitness for work.

If the perfume contains a sensitizing component, intolerance to perfumes by inhalation may occur. Symptoms may include general unwellness, coughing, phlegm, wheezing, chest tightness, headache, shortness of breath with exertion, acute respiratory illness, hayfever, asthma and other respiratory diseases. Perfumes can induce excess reactivity of the airway without producing allergy or airway obstruction. Breathing through a carbon filter mask had no protective effect. Occupational asthma caused by perfume substances, such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms, even though the exposure is below occupational exposure limits. Prevention of contact sensitization to fragrances is an important objective of public health risk management. Hands: Contact sensitization may be the primary cause of hand eczema or a complication of irritant or atopic hand eczema. However hand eczema is a disease involving many factors, and the clinical significance of fragrance contact allergy in severe, chronic hand eczema may not be clear.

Underarm: Skin inflammation of the armpits may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a skin specialist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

Face: An important manifestation of fragrance allergy from the use of cosmetic products is eczema of the face. In men, after-shave products can cause eczema around the beard area and the adjacent part of the neck. Men using wet shaving as opposed to dry have been shown to have an increased risk of allergic to fragrances.

Irritant reactions: Some individual fragrance ingredients, such as citral, are known to be irritant. Fragrances may cause a dose-related contact urticaria (hives) which is not allergic; cinnamal, cinnamic alcohol and Myroxylon pereirae are known to cause hives, but others, including menthol, vanillin and benzaldehyde have also been reported.

Pigmentary anomalies: Type IV allergy is responsible for "pigmented cosmetic dermatitis", referring to increased pigmentation on the face and neck. Testing showed a number of fragrance ingredients were associated, including jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol and geranium oil. Light reactions: Musk ambrette produced a number of allergic reactions mediated by light and was later banned from use in Europe. Furocoumarins (psoralens) in some plant-derived fragrances have caused phototoxic reactions, with redness. There are now limits for the amount of furocoumarins in fragrances. Phototoxic reactions still occur, but are rare.

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

Fragrance allergens act as haptens, which are small molecules that cause an immune reaction only when attached to a carrier protein. However, not all sensitizing fragrance chemicals are directly reactive, but some require previous activation. A prehapten is a chemical that itself causes little or no sensitization, but it is transformed into a hapten outside the skin by a chemical reaction (oxidation in air or reaction with light) without the requirement of an enzyme.

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

Prehaptens: Most terpenes with oxidisable allylic positions can be expected to self-oxidise on air exposure. Depending on the stability of the oxidation products that are formed, the oxidized products will have differing levels of sensitization potential. Tests shows that air exposure of lavender oil increased the potential for sensitization.

Prohaptens: Compounds that are bioactivated in the skin and thereby form haptens are referred to prohaptens. The possibility of a prohapten being activated cannot be avoided by outside measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Various enzymes play roles in both activating and deactivating prohaptens. Skin-sensitizing prohaptens can be recognized and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or studies of sensitization.

QSAR prediction: Prediction of sensitization activity of these substances is complex, especially for those substances that can act both as pre- and prohaptens.

The material may cause severe skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. Repeated exposures may produce severe ulceration. 55rad

Acute Toxicity	×	Carcinogenicity	×
Skin Irritation/Corrosion	×	Reproductivity	×

Serious Eye Damage/Irritation	×	STOT - Single Exposure	×
Respiratory or Skin sensitisation	×	STOT - Repeated Exposure	×
Mutagenicity	×	Aspiration Hazard	×
	Legen	d: 🗙 – Data either not availa	ble 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
Miteq Cleaner 305	Not Available	Not Available	Not Available	Not Available	Not Available
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
ethylene glycol monobutyl ether	LC50	96	Fish	1-700mg/L	2
	EC50	48	Crustacea	ca.1-800mg/L	2
	EC50	72	Algae or other aquatic plants	1-840mg/L	2
	NOEC	24	Crustacea	>1-mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
pine oil	LC50	96	Fish	18.35mg/L	4
	EC50	48	Crustacea	24.5mg/L	4
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic				
	Ioxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)

Bioaccumulative potential

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

Mobility in soil

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

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods		
Product / Packaging disposal	 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. 	

SECTION 14 TRANSPORT INFORMATION

 Marine Pollutant
 NO

 HAZCHEM
 Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

ETHYLENE GLYCOL MONOBUTYL ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

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

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule ${\bf 6}$

GESAMP/EHS Composite List - GESAMP Hazard Profiles

International Air Transport Association (IATA) Dangerous Goods Regulations International Maritime Dangerous Goods Requirements (IMDG Code) United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (pine oil; ethylene glycol monobutyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (pine oil)
Japan - ENCS	No (pine oil)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	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 and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	12/09/2019
Initial Date	01/11/2009

Other information

Ingredients with multiple cas numbers

Name	CAS No
pine oil	8002-09-3, 6878-34-8

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.

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