

Fudge Paintbox

PZ Cussons (PZ Cussons Beauty Australia)

Chemwatch Hazard Alert Code: 2

Chemwatch: 67415

Version No: 6.1.1.1

Safety Data Sheet according to WHS and ADG requirements

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Initial Date: Not Available

L.GHS.AUS.EN

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

Product Identifier

Product name	Fudge Paintbox
Synonyms	Color Refreshing Mask
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. MSDS are intended for use in the workplace. For domestic-use products, refer to consumer labels.
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Details of the manufacturer/importer

Registered company name	PZ Cussons (PZ Cussons Beauty Australia)
Address	Building A, Level 1, 13-15 Compark Circuit Mulgrave 3170 VIC Australia
Telephone	+61 3 8545 2700
Fax	+61 3 8545 2799
Website	www.pzcussons.com
Email	Not Available

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	1800 809 282
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 Model WHS Regulations and the ADG Code.


CHEMWATCH HAZARD RATINGS

	Min	Max	
Flammability	0		
Toxicity	2		
Body Contact	2		
Reactivity	1		
Chronic	2		

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

Poisons Schedule	Not Applicable
GHS Classification [1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Germ Cell Mutagen Category 2, STOT - SE (Resp. Irr.) Category 3, STOT - RE Category 2, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	
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SIGNAL WORD	WARNING
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Hazard statement(s)

H302	Harmful if swallowed
H315	Causes skin irritation
H319	Causes serious eye irritation

Continued...

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H317	May cause an allergic skin reaction
H341	Suspected of causing genetic defects
H335	May cause respiratory irritation
H373	May cause damage to organs through prolonged or repeated exposure
H400	Very toxic to aquatic life
H410	Very toxic to aquatic life with long lasting effects

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P362	Take off contaminated clothing.
P363	Wash contaminated clothing before reuse.
P302+P352	IF ON SKIN: Wash with plenty of water and soap
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
67762-27-0	5-10	<u>cetostearyl alcohol</u>
57-55-6	1-5	<u>propylene glycol</u>
112-02-7	1.8	<u>cetyltrimethylammonium chloride</u>
81-13-0	0.1-1	<u>d-panthenol</u>
100209-45-8	0.1-1	<u>vegetable protein, hydrolysed</u>
71750-80-6	0.1-1	<u>dimethylsiloxane, (aminoethylpropyl)dimethoxysilyloxy-</u>
69430-36-0	0.1-1	<u>keratin hydrolysates</u>
Not Available	0.1-1	wheat amino acids
Not Available	0.1-1	C11-15 Pareth-7
56-81-5	0.1-1	<u>glycerol</u>
24938-91-8	0.1-1	<u>tridecyl alcohol, ethoxylated</u>

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3055-99-0	0.1-1	<u>nonaethylene glycol monododecyl ether</u>
Not Available	0-1	HC Blue no.15
3844-45-9	0-1	<u>C.I. Acid Blue 9, disodium salt</u>
Not Available	0-1	Basic Orange 31
12270-25-6	0-1	<u>C.I. Basic Red 51</u>
6359-45-1	0-1	<u>C.I. Basic Violet 16, chloride</u>
Not Available	0-1	Basic Red 76
Not Available	0.1-1	HC Yellow no.2
Not Available	0-1	Basic Yellow 87
Not Available	0-1	Basic Yellow 57
3248-91-7	0-1	<u>C.I. Basic Violet 2</u>
4430-18-6	0-1	<u>C.I. Acid Violet 43</u>
Not Available	0.1-1	perfume
39236-46-9	0.1-1	<u>imidazolidinyl urea</u>
26172-55-4	0.1-1	<u>5-chloro-2-methyl-4-isothiazolin-3-one</u>
2682-20-4	0.1-1	<u>2-methyl-4-isothiazolin-3-one</u>
7732-18-5	balance	<u>water</u>

No hazardous ingredients present.

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<ul style="list-style-type: none"> ▶ If in eyes, hold eyelids apart and flush the eye continuously with running water. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ 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 or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
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

As in all cases of suspected poisoning, follow the ABCDEs of emergency medicine (airway, breathing, circulation, disability, exposure), then the ABCDEs of toxicology (antidotes, basics, change absorption, change distribution, change elimination).

For poisons (where specific treatment regime is absent):

BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 L/min.
- ▶ Monitor and treat, where necessary, for pulmonary oedema.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ Anticipate seizures.
- ▶ **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

BRONSTEIN, A.C. and CURRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

SECTION 5 FIREFIGHTING MEASURES

Continued...

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 | ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result |
|-----------------------------|--|

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 a significant fire risk, however containers may burn.
- Combustion products include: carbon dioxide (CO₂) sulfur oxides (SO_x) hydrogen cyanide nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.

SECTION 6 ACCIDENTAL RELEASE MEASURES**Personal precautions, protective equipment and emergency procedures****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.

Major Spills

- ▶ Absorb or contain isothiazolinone liquid spills with sand, earth, inert material or vermiculite.
- ▶ The absorbent (and surface soil to a depth sufficient to remove all of the biocide) should be shovelled into a drum and treated with an 11% solution of sodium metabisulfite (Na₂S₂O₅) or sodium bisulfite (NaHSO₃), or 12% sodium sulfite (Na₂SO₃) and 8% hydrochloric acid (HCl).
- ▶ Glutathione has also been used to inactivate the isothiazolinones.
- ▶ Use 20 volumes of decontaminating solution for each volume of biocide, and let containers stand for at least 30 minutes to deactivate microbicide before disposal.
- ▶ If contamination of drains or waterways occurs, advise emergency services.
- ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.

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

SECTION 7 HANDLING AND STORAGE**Precautions for safe handling****Safe handling**

- ▶ **DO NOT allow clothing wet with material to stay in contact with skin**
- ▶ Avoid all personal contact, including inhalation.
- ▶ Wear protective clothing when risk of exposure occurs.
- ▶ Use in a well-ventilated area.
- ▶ Avoid contact with moisture.
- ▶ Avoid contact with incompatible materials.
- ▶ **When handling, DO NOT eat, drink or smoke.**
- ▶ Keep containers securely sealed when not in use.
- ▶ Avoid physical damage to containers.
- ▶ Always wash hands with soap and water after handling.
- ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.
- ▶ Use good occupational work practice.
- ▶ Observe manufacturer's storage and handling recommendations contained within this MSDS.
- ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

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 MSDS.

Conditions for safe storage, including any incompatibilities**Suitable container**

- ▶ Polyethylene or polypropylene container.
- ▶ Packing as recommended by manufacturer.
- ▶ Check all containers are clearly labelled and free from leaks.

Storage incompatibility

- ▶ Avoid reaction with oxidising agents

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

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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	propylene glycol	Propane-1,2-diol total: (vapour & particulates) / Propane-1,2-diol: particulates only	474 mg/m3 / 10 mg/m3 / 150 ppm	Not Available	Not Available	Not Available
Australia Exposure Standards	glycerol	Glycerin mist (a)	10 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
propylene glycol	Propylene glycol; (1,2-Propanediol)	30 mg/m3	1300 mg/m3	7900 mg/m3
cetyltrimethylammonium chloride	Hexadecyltrimethylammonium chloride	1.1 mg/m3	12 mg/m3	70 mg/m3
glycerol	Glycerine (mist); (Glycerol; Glycerin)	30 mg/m3	310 mg/m3	2500 mg/m3
nonaethylene glycol monododecyl ether	Brij-35; (alpha-Dodecyl-omega-hydroxypoly(oxyethylene))	1 mg/m3	11 mg/m3	200 mg/m3
5-chloro-2-methyl-4-isothiazolin-3-one	Chloro-2-methyl-4-isothiazolin-3-one, 5-	0.2 mg/m3	0.2 mg/m3	0.2 mg/m3

Ingredient	Original IDLH	Revised IDLH
cetostearyl alcohol	Not Available	Not Available
propylene glycol	Not Available	Not Available
cetyltrimethylammonium chloride	Not Available	Not Available
d-panthenol	Not Available	Not Available
vegetable protein, hydrolysed	Not Available	Not Available
dimethylsiloxane, (aminoethylpropyl)dimethoxysilyloxy-	Not Available	Not Available
keratin hydrolysates	Not Available	Not Available
wheat amino acids	Not Available	Not Available
C11-15 Pareth-7	Not Available	Not Available
glycerol	Not Available	Not Available
tridecyl alcohol, ethoxylated	Not Available	Not Available
nonaethylene glycol monododecyl ether	Not Available	Not Available
HC Blue no.15	Not Available	Not Available
C.I. Acid Blue 9, disodium salt	Not Available	Not Available
Basic Orange 31	Not Available	Not Available
C.I. Basic Red 51	Not Available	Not Available
C.I. Basic Violet 16, chloride	Not Available	Not Available
Basic Red 76	Not Available	Not Available
HC Yellow no.2	Not Available	Not Available
Basic Yellow 87	Not Available	Not Available
Basic Yellow 57	Not Available	Not Available
C.I. Basic Violet 2	Not Available	Not Available
C.I. Acid Violet 43	Not Available	Not Available
perfume	Not Available	Not Available
imidazolidinyl urea	Not Available	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Not Available	Not Available
2-methyl-4-isothiazolin-3-one	Not Available	Not Available
water	Not Available	Not Available

MATERIAL DATA

CEL Ceiling: 0.00006 mg/m3 (sensitiser)

(compare TLV-C subtilisins; proteolytic enzymes - 100% crystalline)

Exposure at or below the recommended TLV-C is thought to minimise the potential for allergic respiratory sensitization for the majority of immunologically normal persons and to minimise skin irritation and sensitization. TLV compliance is contingent on measurement of workplace air concentrations with a high volume sampler appropriate to capture these proteins for at least 60 minutes.


Although the recommended TLV-C is specifically prescribed for subtilisins, the Chemwatch recommendation (CEL) recognizes that all proteins have the potential to produce allergic responses. It should be noted, however, that proteins are typically poorly absorbed through the skin and after inhalation. Literature reports indicate that protein bioavailability, via the lung, is as low as 2%.

CEL TWA: 0.1 mg/m3; STEL 0.3 mg/m3 total isothiazolinones (Rohm and Haas)

(CEL = Chemwatch Exposure Limit)

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Exposure controls

<p>Appropriate engineering controls</p>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.</p> <p>An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1" data-bbox="359 571 1492 817"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="359 884 1492 1041"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)	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<p>Personal protection</p>																					
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ 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] 																				
<p>Skin protection</p>	<p>See Hand protection below</p>																				
<p>Hands/feet protection</p>	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> ▶ frequency and duration of contact, ▶ chemical resistance of glove material, ▶ glove thickness and ▶ dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> ▶ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ▶ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ▶ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. ▶ Contaminated gloves should be replaced. <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <ul style="list-style-type: none"> ▶ Butyl rubber gloves ▶ Nitrile rubber gloves 																				

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Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.
Thermal hazards	Not Available

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	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

Type AK-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 5 x ES	AK-AUS / Class 1 P2	-	AK-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	AK-2 P2	AK-PAPR-2 P2
up to 50 x ES	-	AK-3 P2	-
50+ x ES	-	Air-line**	-

* - Continuous-flow; ** - Continuous-flow or positive pressure demand

^ - 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)

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Coloured viscous emulsion with a green apple odour; miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	2.5-7.0	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	7000-16000 mPa.s @23C
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7

Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Isothiazolinones are moderately to highly toxic by oral administration. The major signs of toxicity were severe gastric irritation, lethargy, and ataxia</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Aqueous solutions of isothiazolinones may be irritating or even corrosive depending on concentration. Solutions containing more than 0.5% (5000 ppm active substance) may produce severe irritation of human skin whilst solutions containing more than 100 ppm may irritate the skin.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>Solutions containing isothiazolinones may produce corrosion of the mucous membranes and cornea. Instillation of 0.1 ml of an aqueous solution containing 560 ppm isothiazolinone into rabbit eye did not produce irritation whereas concentrations, typically around 3% and 5.5 %, were severely irritating or corrosive to the eye.. Symptoms included clouding of the cornea, chemosis and swelling of the eyelids.</p>
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p> <p>Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</p> <p>There is a possibility that unintended contact with this product (such as through a cut, needle stick, eye or mucous membrane, or inhalation) could result in allergic or hypersensitivity reactions. Such reactions are more likely following repeated exposures or in persons with a pre-existing allergy to certain proteins.</p> <p>Dusts produced by proteins are capable, under certain conditions, of sensitising workers by virtue of the bodies reaction to foreign proteins. Typical allergic asthma may be rapidly produced after exposure, with symptoms may include chronic cough, sputum production, fever, myalgia, fatigue, airway obstruction; chest radiographs may show a generalised reticulonodular pattern, or basal or apical fibrosis. In addition there may be retrosternal discomfort, headache, stomach-ache and general severe dyspnoea may develop giving a clinical picture similar to that of farmer's lung and allied conditions of extrinsic allergic alveolitis. No irritation is likely after brief skin contact, but prolonged contact in the presence of moisture may result in soreness, redness, inflammation and possible ulceration of the skin. Repeated attacks may lead to permanent impairment of lung function due to fibrotic change.</p> <p>NTP studies of nitro- and amino-anthraquinones, have demonstrated that each compound tested has some activity as a mutagen. Most compounds of this class that have been the subjects of two-year studies have also been found to be carcinogenic in one or more species. Sites of tumor development include the urinary bladder in rats and the liver of both rats and mice.</p> <p>The isothiazolinones are known contact sensitizers. Data are presented which demonstrate that, in comparison with the chlorinated and dichlorinated compounds which share immunological cross-reactivity, the non-chlorinated isothiazolinones have a lower potential for sensitization and no documented immunological cross-reaction with the chlorinated isothiazolinones. The risk of sensitization depends on how contact with the product occurs. The risk is greater when the skin barrier has been damaged and smaller when the skin is healthy. Dermatological studies have demonstrated that mixed isothiazolinone concentrations below 20 ppm may cause sensitisation and that allergic reactions can be provoked in sensitized persons even with concentrations in the range of 7-15 ppm active isothiazolinones.</p> <p>The isothiazolinones are a group of heterocyclic sulfur-containing compounds. In general all are electrophilic molecules containing an activated N-S bond that enables them with nucleophilic cell entities, thus exerting biocidal activity. A vinyl activated chlorine atom makes allows to molecule to exert greater antimicrobial efficiency but at the same time produces a greater potential for sensitisation.</p> <p>Several conclusions relating to the sensitising characteristics of the isothiazolinones may therefore be drawn* :</p> <ul style="list-style-type: none"> ▶ The strongest sensitizers are the chlorinated isothiazolinones. ▶ There are known immunological cross-reactions between at least 2 different chlorinated isothiazolinones. ▶ There appears to be no immunological cross reaction between non-chlorinated isothiazolinones and chlorinated isothiazolinones. ▶ Although classified as sensitizers, the nonchlorinated isothiazolinones are considerably less potent sensitizers than are the chlorinated isothiazolinones. ▶ By avoiding the use of chlorinated isothiazolinones, the potential to induce sensitisation is greatly reduced. ▶ Despite a significant percentage of the population having been previously sensitised to chlorinated and non-chlorinated species, it is likely that careful and judicious use of non-chlorinated isothiazolinones will result in reduced risk of allergic reactions in those persons. ▶ Although presently available data promise that several non-chlorinated isothiazolinones will offer effective antimicrobial protection in industrial and personal care products, it is only with the passage of time that proof of their safety in use or otherwise will become available. <p>* B.R. Alexander: Contact Dermatitis 2002, 46, pp 191-196</p> <p>Although there have been conflicting reports in the literature, it has been reported by several investigators that isothiazolinones are mutagenic in <i>Salmonella typhimurium</i> strains (Ames test). Negative results were obtained in studies of the DNA-damaging potential of mixed isothiazolinones (Kathon) in mammalian</p>

Fudge Paintbox

cells *in vitro* and of cytogenetic effects and DNA-binding *in vivo*. The addition of rat liver S-9 (metabolic activation) reduced toxicity but did not eliminate mutagenicity. These compounds bind to the proteins in the S-9. At higher concentrations of Kathon the increase in mutagenicity may be due to an excess of unbound active compounds.

A study of cutaneous application of Kathon CG in 30 months, three times per week at a concentration of 400 ppm (0.04%) a.i. had no local or systemic tumourigenic effect in male mice. No dermal or systemic carcinogenic potential was observed.

Reproduction and teratogenicity studies with rats, given isothiazolinone doses of 1.4-14 mg/kg/day orally from day 6 to day 15 of gestation, showed no treatment related effects in either the dams or in the foetuses

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

Fudge Paintbox	TOXICITY	IRRITATION
	Not Available	Not Available
cetostearyl alcohol	TOXICITY	IRRITATION
	Oral (mouse) LD50: 15000 mg/kg ^[2]	Not Available
propylene glycol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg - mild
	Oral (rat) LD50: 20000 mg/kg ^[2]	Eye (rabbit): 500 mg/24h - mild
		Skin(human):104 mg/3d Intermit Mod Skin(human):500 mg/7days mild
cetyltrimethylammonium chloride	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 4300 mg/kg ^[2] Oral (rat) LD50: 250 mg/kg ^[2]	Not Available
d-panthenol	TOXICITY	IRRITATION
	Oral (mouse) LD50: 15000 mg/kg ^[2]	Eye (rabbit): 0.5 mg - mild Skin (rabbit): 500 mg/4h - mild
vegetable protein, hydrolysed	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^{**[2]}	Eye (rabbit): not irritating * Skin (rabbit): not irritating *
dimethylsiloxane, (aminoethylpropyl)dimethoxysilyloxy-	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg ^{**[2]}	* GE Silicones Eye (rabbit): SEVERE * Skin (rabbit): moderate *
keratin hydrolysates	TOXICITY	IRRITATION
	Not Available	Not Available
glycerol	TOXICITY	IRRITATION
	dermal (guinea pig) LD50: 54000 mg/kg ^[1] Oral (rat) LD50: >20<39800 mg/kg ^[1]	Not Available
tridecyl alcohol, ethoxylated	TOXICITY	IRRITATION
	Oral (rat) LD50: 7400 mg/kg ^[2]	Skin (rabbit): 2000 mg/4w mild
nonaethylene glycol monododecyl ether	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2] Oral (rat) LD50: 1000 mg/kg ^[2]	Not Available
C.I. Acid Blue 9, disodium salt	TOXICITY	IRRITATION
	Not Available	Not Available
C.I. Basic Red 51	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2] Oral (rat) LD50: 250 mg/kg ^[2]	Not Available
C.I. Basic Violet 16, chloride	TOXICITY	IRRITATION
	Not Available	Not Available

C.I. Basic Violet 2	TOXICITY	IRRITATION
	Not Available	Not Available
C.I. Acid Violet 43	TOXICITY	IRRITATION
	Not Available	Not Available
imidazolidinyl urea	TOXICITY	IRRITATION
	Oral (rat) LD50: 11300 mg/kg ^[2]	Nil reported
5-chloro-2-methyl-4-isothiazolin-3-one	TOXICITY	IRRITATION
	Not Available	Not Available
2-methyl-4-isothiazolin-3-one	TOXICITY	IRRITATION
	Not Available	Not Available
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 msds. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

Fudge Paintbox	<p>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.</p> <p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>No significant acute toxicological data identified in literature search.</p>
CETOSTEARYL ALCOHOL	<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 severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p> <p>No significant acute toxicological data identified in literature search.</p>
PROPYLENE GLYCOL	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>The acute oral toxicity of propylene glycol is very low, and large quantities are required to cause perceptible health damage in humans. Serious toxicity generally occurs only at plasma concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time. It would be nearly impossible to reach toxic levels by consuming foods or supplements, which contain at most 1 g/kg of PG. Cases of propylene glycol poisoning are usually related to either inappropriate intravenous administration or accidental ingestion of large quantities by children. The potential for long-term oral toxicity is also low. Because of its low chronic oral toxicity, propylene glycol was classified by the U. S. Food and Drug Administration as "generally recognized as safe" (GRAS) for use as a direct food additive.</p> <p>Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce slight transient conjunctivitis (the eye recovers after the exposure is removed). Exposure to mists may cause eye irritation, as well as upper respiratory tract irritation. Inhalation of the propylene glycol vapours appears to present no significant hazard in ordinary applications. However, limited human experience indicates that inhalation of propylene glycol mists could be irritating to some individuals. It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations.</p> <p>Propylene glycol is metabolised in the human body into pyruvic acid (a normal part of the glucose-metabolism process, readily converted to energy), acetic acid (handled by ethanol-metabolism), lactic acid (a normal acid generally abundant during digestion), and propionaldehyde (a potentially hazardous substance).</p> <p>Propylene glycol shows no evidence of being a carcinogen or of being genotoxic.</p> <p>Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but that they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis to propylene glycol may be greater than 2% in patients with eczema.</p> <p>One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic reactions, such as rhinitis or hives in children</p> <p>Another study suggested that the concentrations of PGEs (counted as the sum of propylene glycol and glycol ethers) in indoor air, particularly bedroom air, is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema, and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use</p>

Fudge Paintbox

of water-based paints and water-based system cleansers.

Patients with vulvodynia and interstitial cystitis may be especially sensitive to propylene glycol. Women suffering with yeast infections may also notice that some over the counter creams can cause intense burning. Post menopausal women who require the use of an estrogen cream may notice that brand name creams made with propylene glycol often create extreme, uncomfortable burning along the vulva and perianal area. Additionally, some electronic cigarette users who inhale propylene glycol vapor may experience dryness of the throat or shortness of breath. As an alternative, some suppliers will put Vegetable Glycerin in the "e-liquid" for those who are allergic (or have bad reactions) to propylene glycol.

Adverse responses to intravenous administration of drugs which use PG as an excipient have been seen in a number of people, particularly with large dosages thereof. Responses may include "hypotension, bradycardia... QRS and T abnormalities on the ECG, arrhythmia, cardiac arrest, serum hyperosmolality, lactic acidosis, and haemolysis". A high percentage (12% to 42%) of directly-injected propylene glycol is eliminated/secreted in urine unaltered depending on dosage, with the remainder appearing in its glucuronide-form. The speed of renal filtration decreases as dosage increases, which may be due to propylene glycol's mild anesthetic / CNS-depressant -properties as an alcohol. In one case, intravenous administration of propylene glycol-suspended nitroglycerin to an elderly man may have induced coma and acidosis.

Propylene glycol is an approved food additive for dog food under the category of animal feed and is generally recognized as safe for dogs with an LD50 of 9 mL/kg. The LD50 is higher for most laboratory animals (20 mL/kg)

Similarly, propylene glycol is an approved food additive for human food as well. The exception is that it is prohibited for use in food for cats due to links to Heinz body anemia.

for acid mists, aerosols, vapours

Data from assays for genotoxic activity *in vitro* suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events *in vivo* in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH *in vivo* differ from exposures *in vitro* in that, *in vivo*, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than *in vitro*.

For alkytrimethylammonium chloride (ATMAC)

Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41. In addition, certain surfactants will satisfy the criteria for classification as Corrosive with R34 in addition to the acute toxicity.

According to Centre Europeen des Agents de Surface et de leurs Intermediaires Organiques (CESIO), C8-18 alkytrimethylammonium chloride (ATMAC) (i.e., lauryl, coco, soya, and tallow) are classified as Corrosive (C) with the risk phrases R22 (Harmful if swallowed) and R34 (Causes burns). C16 ATMAC is classified as Harmful (Xn) with the risk phrases R22 (Harmful if swallowed), R38 (Irritating to skin), and R41 (Risk of serious damage to eyes). C20-22 ATMAC are classified as Irritant (Xi) with R36/38 (Irritating to eyes and skin).

Toxicokinetics and Acute Toxicity: The few available absorption studies conducted with cationic surfactants indicate that absorption occurs in small amounts through the skin. Percutaneous absorption of radiolabelled C12 alkytrimethylammonium bromide (ATMAB) in 3% aqueous solution (applied to an 8 cm² area with occlusion) in the rat was low and corresponded to 0.6% of the applied 14C activity in 72 hours. Most of the absorbed surfactant was excreted in the urine, i.e. 0.35% of the applied 14C activity within the first 24 hours, whereas 13.2% remained on the skin after rinsing. Cutaneous application of the surfactant without rinsing resulted in a greater degree of percutaneous absorption (3.15%) in 48 hours. In the rat elimination after parenteral administration was rapid and was effected primarily via the urine, - more than 80% of the radioactivity was eliminated within 24 hours of application. About 80% of the 14C activity was found in the gastrointestinal tract 8 hours after oral administration of 14C-labelled C16 ATMAB. Only small amounts of the applied radioactivity were found in the urine and in the blood plasma. This indicates poor intestinal absorption. Similar small amounts of 14C were found in the liver, kidneys, spleen, heart, lungs and skeletal muscles. Within 3 days of ingestion, 92% of the administered radioactivity had been excreted in the faeces and 1% in the urine. No appreciable enterohepatic circulation of the radioactivity was found.

The acute oral toxicity of alkytrimethylammonium salts is somewhat higher than the toxicity of anionic and nonionic surfactants. This may be due to the strongly irritating effect which cationic surfactants exhibit on the mucous membrane of the gastrointestinal tract (SFT 1991). Cationic surfactants are generally about 10 times more toxic when administered by the intravenous route compared to oral administration.

Skin and Eye Irritation: Skin irritation depends on surfactant concentration. Regardless of the structure, cationic surfactants lead to serious destruction of the skin at high concentrations. Solutions of approximately 0.1% are rarely irritating, whereas irritation is usually pronounced at concentrations between 1.0 and 10.0% surfactant. C16 ATMAC was severely irritating to rabbit skin in a concentration of 2.5%. The surfactant was applied to intact and abraded sites and scored after 34 hours. Then the skin was rinsed and then scored again after 48 hours. The erythema and Eschar Index was 3.75 (maximum 4) and the edema Index was 2.0 (maximum 4).

With regard to eye irritation, cationic surfactants are the most irritating of the surfactants. The longer chained alkytrimethylammonium salts are less irritating to the rabbit eye than the shorter alkyl chain homologues. C10 ATMAB, C12 ATMAB, and C16 ATMAC were tested in concentrations between 0.1 and 1.0% in water and were found to be significantly irritating or injurious to the rabbit eye. A 5% solution of C18 ATMAC was instilled into the eyes of guinea pigs, and this concentration was very irritating with a total PII (The Primary Irritation Index) score of 96 (maximum 110).

A homologous series of ATMAB produced very little swelling of the stratum corneum and some homologues produced a shrinkage of the stratum corneum after prolonged exposure.

Many proteins in the skin are considerably more resistant to the denaturing effects of cationic surfactants compared to those of anionic surfactants. As cationic surfactants frequently have a lower critical micelle concentration than the anionic surfactants, a saturation of the surfactant/protein complex is prevented by the formation of micelles.

Compared to a representative anionic surfactant, the cooperative binding with subsequent protein denaturation requires about a tenfold higher concentration of a cationic surfactant. Contrary to the irreversible denaturing effect of sodium dodecyl sulfate, the adverse effects of some cationic surfactants on proteins may be reversible. Cationic surfactants can interact with proteins or peptides by polar and hydrophobic binding. Polar interactions result in electrostatic bonds between the negatively charged groups of the protein molecule and the positively charged surfactant molecule.

Sensitisation: A repeated insult patch test of C16 ATMAC was conducted with 114 volunteers. Seventeen days after the last induction of 0.25% surfactant, a challenge patch of 0.25% was applied. No sensitization was observed.

Sub-chronic toxicity: C16 ATMAB was administered at concentrations of 10, 20, and 45 mg/kg/day via the drinking water to rats for one year. The only effect observed was a decrease in body weight gain in the 45 mg/day dose group.

Reproductive Toxicity: No embryo toxic effects were seen, when C18 ATMAC was applied dermally to pregnant rats during the period of major organogenesis (day 6-15 of gestation). The concentrations of C18 ATMAC were 0.9, 1.5 and 2.5%. There was no increase in the incidence of fetal malformations. C16 ATMAB was not teratogenic in rats after oral doses. Mild embryonic effects were observed with 50 mg/kg/day, but these effects were attributed to maternal toxicity rather than to a primary embryonic effect. Lower doses of C16 ATMAB showed no embryo toxic or teratogenic effects.

Mutagenicity: C16 ATMAC was studied in *in vitro* short-term tests to detect potential mutagenic effects. Cultures of Syrian golden hamster embryo cells were used for an *in vitro* bioassay. No *in vitro* transformation of hamster embryo cells was induced, and C16 ATMAC was not mutagenic in *Salmonella typhimurium* (Inoue and Sunakawa 1980). No mutagenic effects or genetic damages were

CETYLTRIMETHYLAMMONIUM CHLORIDE

indicated in a survey of nine short-term genotoxicity tests with C16 and C18 ATMAC (Yam *et al.* 1984). Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljøministeriet (Danish Environmental Protection Agency)

For quaternary ammonium compounds (QACs):

Quaternary ammonium compounds (QACs) are cationic surfactants. They are synthetic organically tetra-substituted ammonium compounds, where the R substituents are alkyl or heterocyclic radicals. A common characteristic of these synthetic compounds is that one of the R's is a long-chain hydrophobic aliphatic residue.

The cationic surface active compounds are in general more toxic than the anionic and non-ionic surfactants. The positively-charged cationic portion is the functional part of the molecule and the local irritation effects of QACs appear to result from the quaternary ammonium cation.

Due to their relative ability to solubilise phospholipids and cholesterol in lipid membranes, QACs affect cell permeability which may lead to cell death. Further QACs denature proteins as cationic materials precipitate protein and are accompanied by generalised tissue irritation.

It has been suggested that the experimentally determined decrease in acute toxicity of QACs with chain lengths above C16 is due to decreased water solubility.

In general it appears that QACs with a single long-chain alkyl groups are more toxic and irritating than those with two such substitutions,

The straight chain aliphatic QACs have been shown to release histamine from minced guinea pig lung tissue. However, studies with benzalkonium chloride have shown that the effect on histamine release depends on the concentration of the solution. When cell suspensions (11% mast cells) from rats were exposed to low concentrations, a decrease in histamine release was seen. When exposed to high concentrations the opposite result was obtained.

In addition, QACs may show curare-like properties (specifically benzalkonium and cetylpyridinium derivatives, a muscular paralysis with no involvement of the central nervous system. This is most often associated with lethal doses. Parenteral injections in rats, rabbits and dogs have resulted in prompt but transient limb paralysis and sometimes fatal paresis of the respiratory muscles. This effect seems to be transient.

From human testing of different QACs the generalised conclusion is obtained that all the compounds investigated to date exhibit similar toxicological properties.

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

for Fatty Nitrogen-Derived Cationics (FND Cationics):

Overall, the available data support the conclusion that, because of their closely-related structures, FND Cationics possess similar environmental fate and ecotoxicity across the category.

Environmental fate:

FND Cationics are considered to be essentially nonvolatile. Water solubility estimates varied from insoluble to slightly soluble, with higher solubility predictions tending to occur for lower molecular weight chemicals. Log Kow values less than 5 were predicted for all of the chemicals that could be modeled.

Measurement and prediction of physical/chemical properties for surfactants are complicated by their behavior in test systems and the environment, and the Kow is not an appropriate hydrophobicity parameter for reliably predicting environmental behavior. Although predictions vary, the overall data and knowledge of the chemicals support the conclusion that the FND Cationics have closely related structures and behave similarly from the perspective of physical/chemical properties.

Fugacity models predict virtually no occurrence of the FND Cationics in air. Nonetheless, modeling of these and similar substances indicates that these chemicals would be expected to degrade relatively rapidly upon exposure to light (t1/2 values ranging from approximately 2.8 to 5.9 hours).

Predicted distribution of the chemicals in the environment was to water and/or sediment compartments based on the assumption that release of the chemicals to the environment is exclusively via water. For chemicals with higher predicted water solubility (lower Kow), the water compartment was favoured. Measured biodegradation rates were variable and frequently confounded by adsorption.

Overall, the FND Cationic Category chemicals are biodegradable.

Cationic substances in the environment instantaneously form complexes with naturally occurring negatively charged constituents in sewage, soils, sediments, and with dissolved humic substances in surface waters. This complexation behavior results in reduced bioavailability in actual environmental conditions that is not adequately represented by standard laboratory assays and/or predictions by various QSAR models.

Ecotoxicity:

These chemicals, by the nature of their surfactant properties, are toxic to aquatic organisms at low concentrations

Measured aquatic toxicity values indicated acute LC50 and EC50 values generally less than approximately 25 mg/l for fish, daphnid and algae. Other species may be less sensitive to the toxicity of these surfactants with acute LC50 values of 36 and > 50 mg/l recorded for shrimp and crabs, respectively. Chronic toxicity to aquatic organisms varied considerably, with NOECs ranging from 4.15 ug/l to 12.7 mg/l. These studies of aquatic toxicity, many of which were conducted in natural waters with and without added effluents, indicate that the source and composition of the test water dramatically affects the toxicity of the test substance.

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

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

Changes in motor activity ** Akzo * for hexadecylammonium chloride Cetyltrimethylammonium chloride is expected to produce similar toxic effects

D-PANTHENOL

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

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.

VEGETABLE PROTEIN, HYDROLYSED

* Croda SDS for Hydrobrazilnut AA

**DIMETHYLSILOXANE,
(AMINOETHYLPROPYL)DIMETHOXYSILOXY-**

For siloxanes:

Effects which based on the reviewed literature do not seem to be problematic are acute toxicity, irritant effects, sensitization and genotoxicity.

Some studies indicate that some of the siloxanes may have endocrine disrupting properties, and reproductive effects have caused concern about the possible effects of the siloxanes on humans and the environment.

Only few siloxanes are described in the literature with regard to health effects, and it is therefore not possible to make broad conclusions and comparisons of the toxicity related to short-chained linear and cyclic siloxanes based on the present evaluation.

Data are primarily found on the cyclic siloxanes D4 (octamethylcyclotetrasiloxane) and D5 (decamethylcyclopentasiloxane) and the short-linear HMDS (hexamethyldisiloxane).

These three siloxanes have a relatively low order of acute toxicity by oral, dermal and inhalatory routes and do not require classification for this effect.

They are not found to be irritating to skin or eyes and are also not found sensitizing by skin contact. Data on respiratory sensitization have not been identified.

Subacute and subchronic toxicity studies show that the liver is the main target organ for D4 which also induces liver cell enzymes.

This enzyme induction contributes to the elimination of the substance from the tissues. Primary target organ for D5 exposure by inhalation is the lung. D5 has an enzyme induction profile similar to that of D4. Subacute and subchronic inhalation of HMDS affect in particular the lungs and kidneys in rats.

None of the investigated siloxanes show any signs of genotoxic effects *in vitro* or *in vivo*. Preliminary results indicate that D5 has a potential carcinogenic effect.

D4 is considered to impair fertility in rats by inhalation and is classified as a substance toxic to reproduction in category 3 with the risk phrase R62 ('Possible risk of impaired fertility').

The results of a study to screen for oestrogen activity indicate that D4 has very weak oestrogenic and antioestrogenic activity and is a partial agonist (enhances the effect of the estrogen). It is not uncommon for compounds that are weakly oestrogenic to also have antioestrogenic properties. Comparison of the oestrogenic potency of D4 relative to ethinyloestradiol (steroid hormone) indicates that D4 is 585,000 times less potent than ethinyloestradiol in the rat strain Sprague-Dawley and 3.7 million times less potent than ethinyloestradiol in the Fisher-344 rat strain. Because of the lack of effects on other endpoints designated to assess oestrogenicity, the oestrogenicity as mode of action for the D4 reproductive effects has been questioned. An indirect mode of action causing a delay of the LH (luteinising hormone) surge necessary for optimal timing of ovulation has been suggested as the mechanism.

Based on the reviewed information, the critical effects of the siloxanes are impaired fertility (D4) and potential carcinogenic effects (uterine tumours in females). Furthermore there seem to be some effects on various organs following repeated exposures, the liver (D4), kidney (HMDS) and lung (D5 and HMDS) being the target organs.

A possible oestrogenic effect contributing to the reproductive toxicity of D4 is debated. There seems however to be some indication that this toxicity may be caused by another mechanism than oestrogen activity

For alkoxysilanes:

Low molecular weight alkoxysilanes (including alkyl orthosilicates) are a known concern for lung toxicity, due to inhalation of vapours or aerosols causing irreversible lung damage at low doses.

Alkoxysilane groups that rapidly hydrolyse when in contact with water, result in metabolites that may only cause mild skin irritation.

Although there appears to be signs of irritation under different test conditions, based on the available information, the alkoxysilanes cannot be readily classified as a skin irritant.

The trimethoxysilane group of chemicals have previously been associated with occupational eye irritation in exposed workers who experienced severe inflammation of the cornea. Based on the collective information, these substances are likely to be severe irritants to the eyes.

Methoxysilanes are generally reported to possess higher reactivity and toxicity compared to ethoxysilanes; some methoxysilanes appear to be carcinogenic. In the US, alkoxysilanes with alkoxy groups greater than C2 are classified as moderate concern.

Based on available information on methoxysilanes, the possibility that this family causes skin sensitisation cannot be ruled out.

Amine-functional methoxysilanes have previously been implicated as a cause of occupational contact dermatitis, often as a result of repeated skin exposure with workers involved in the manufacture or use of the resins containing the chemical during fibreglass production.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

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.

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).

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.

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 of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

GLYCEROL

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

For glycerol:

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Acute toxicity: Glycerol is of a low order of acute oral and dermal toxicity with LD50 values in excess of 4000 mg/kg bw. At very high dose levels, the signs of toxicity include tremor and hyperaemia of the gastro-intestinal tract. Skin and eye irritation studies indicate that glycerol has low potential to irritate the skin and the eye. The available human and animal data, together with the very widespread potential for exposure and the absence of case reports of sensitisation, indicate that glycerol is not a skin sensitiser.

Repeat dose toxicity: Repeated oral exposure to glycerol does not induce adverse effects other than local irritation of the gastro-intestinal tract. The overall NOEL after prolonged treatment with glycerol is 10,000 mg/kg bw/day (20% in diet). At this dose level no systemic or local effects were observed. For inhalation exposure to aerosols, the NOAEC for local irritant effects to the upper respiratory tract is 165 mg/m³ and 662 mg/m³ for systemic effects.

Genotoxicity: Glycerol is free from structural alerts, which raise concern for mutagenicity. Glycerol does not induce gene mutations in bacterial strains, chromosomal effects in mammalian cells or primary DNA damage *in vitro*. Results of a limited gene mutation test in mammalian cells were of uncertain biological relevance. *In vivo*, glycerol produced no statistically significant effect in a chromosome aberrations and dominant lethal study. However, the limited details provided and the absence of a positive control, prevent any reliable conclusions to be drawn from the *in vivo* data. Overall, glycerol is not considered to possess genotoxic potential.

Carcinogenicity: The experimental data from a limited 2 year dietary study in the rat does not provide any basis for concerns in relation to carcinogenicity. Data from non-guideline studies designed to investigate tumour promotion activity in male mice suggest that oral administration of glycerol up to 20 weeks had a weak promotion effect on the incidence of tumour formation.

Reproductive and developmental toxicity: No effects on fertility and reproductive performance were observed in a two generation study with glycerol administered by gavage (NOAEL 2000 mg/kg bw/day). No maternal toxicity or teratogenic effects were seen in the rat, mouse or rabbit at the highest dose levels tested in a guideline comparable teratogenicity study (NOEL 1180 mg/kg bw/day).

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.

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.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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.

No significant acute toxicological data identified in literature search.

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

TRIDECYL ALCOHOL, ETHOXYLATED

NONAETHYLENE GLYCOL MONODODECYL ETHER

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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.

C.I. ACID BLUE 9, DISODIUM SALT

The substance is classified by IARC as Group 3:
NOT classifiable as to its carcinogenicity to humans.
Evidence of carcinogenicity may be inadequate or limited in animal testing.

C.I. BASIC RED 51

The acute oral LD50 was set at 250 - 500 mg/kg bw in females and at 500 - 1000 mg/kg bw in males. The acute dermal LD50 is greater than 2000 mg/kg bw. The NOAEL was set at 12.25 mg/kg bw/day (repeated dose oral toxicity study). In light of the effects on the thyroid and pituitary (sub-chronic oral toxicity study), the NOAEL was set at 10 mg/kg bw/day. Basic Red 51 was not toxic to embryo or foetus and was not teratogenic. The NOEL for the maternal effects was set at 20 mg/kg bw/day and at 180 mg/kg bw/day for foetal effects. Basic Red 51 was not irritating to the skin and moderately irritating to the eyes. It is not considered to be a sensitiser. A total of 0.018% of the applied dose is reported to have penetrated, corresponding to a percutaneous absorption of 0.040 ig/cm². However, the substance was not tested in the presence of an oxidising agent. Basic Red 51 has been tested in prokaryotic and mammalian cells for gene mutation, and in mammalian cells for chromosomal aberration *in vitro*. Two tests have been performed (bone marrow micronucleus and UDS tests). The test for gene mutation in prokaryotes has been found positive in the presence of a reducing metabolic activation system. test for gene mutation in mammalian cells showed that the test agent is non mutagenic under both activation conditions. test for clastogenicity in human lymphocytes is negative, with only a normal activation system. micronucleus test in mice gave negative results; no firm evidence that the bone marrow was reached by the test agent was noted. *in vivo/in vitro* UDS on rats hepatocytes is negative for the treatment of 16 hours; the effect of 2 hours treatment could not be evaluated due to the absence of a concurrent positive control. This is why the study is considered as inadequate according to the OECD guideline 486. Considering that the metabolic behaviour suspected in the strain TA 98, could have influenced specifically the results observed and considering the minor inadequacy of the UDS test and the absence of toxicokinetics data in the micronucleus assay, it may be concluded that there are insufficient data to evaluate the mutagenic potential of this dye Opinion of the SCCNFP: October 2003

C.I. BASIC VIOLET 16, CHLORIDE

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a

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	<p>non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>No significant acute toxicological data identified in literature search.</p> <p>Data for C.I. Basic Violet phosphate has been used to classify the substance.</p>
C.I. BASIC VIOLET 2	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. Substance has been investigated as a tumorigen.</p>
IMIDAZOLIDINYL UREA	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>For imidazolidinyl urea and diazolidinyl urea: Imidazolidinyl urea releases formaldehyde into cosmetics at temperatures above 10 °C. A 1974 study found formaldehyde release occurs at the non-physiological conditions of 60 °C and a pH of 6. In water-containing cosmetics like shampoos, formaldehyde release increases with a rise in pH and temperature of the solution as well as a longer storage period</p> <p>Acute toxicity: The prevalence of positive reactions to imidazolidinyl urea was 1.9 percent and 3.2 percent in patients with contact dermatitis in two independent studies. Concomitant positive reactions have also been reported for imidazolidinyl urea and formaldehyde as well as imidazolidinyl urea and <i>N</i>-(3-chloroallyl)hexaminium chloride (Quaternium-15) /</p> <p>A British study conducted between 1982-1993 showed that the frequency of preservative allergy to imidazolidinyl urea in 5,167 patients with contact dermatitis was 0.99%. Furthermore, the face and the hands were the sites of allergy for 69% and 19% of patients, respectively. Curiously imidazolidinyl urea did not induce contact sensitivity in several reports. In a study, 200 subjects were given repeated insult patch tests with a 10% aqueous solution of imidazolidinyl urea three times per week for five weeks. These subjects were challenged for 24 hours after treatment and no evidence of primary irritation or sensitization occurred</p> <p>Imidazolidinyl urea sensitised 60-70% of female Dunkin Hartley guinea pigs in a dose-dependent manner. The animals were patch tested with 1, 5, and 10% imidazolidinyl urea in petrolatum and read after 48 hours. Imidazolidinyl urea was found to be a sensitizer after topical applications of 25 µl of 10, 25, or 50 percent to CBA/Ca mice daily for three days induced significant radiolabelled thymidine incorporation into local lymph nodes four days after the last treatment with imidazolidinyl urea</p> <p>Skin irritation: Imidazolidinyl urea was non-irritating after an application of 0.1 ml of a solution containing 5, 10, or 20 percent of this compound in the right eye of albino rabbits. In another study, imidazolidinyl urea did not produce any irritation after application to the shaved backs of albino rabbits at concentrations of 0, 1, 2.5, and 5 percent</p> <p>Imidazolidinyl urea was described as not phototoxic in female Hartley guinea pigs after intradermal injections of 1-5% doses into the shaved backs and subsequent irradiation with FL20E and FL20BLB light for 30 minutes. The animals were again injected and irradiated 24 and 48 hours after the initial injection with no reaction</p> <p>Subchronic Toxicity: Imidazolidinyl urea was applied in powder form at concentrations of 20, 45, 90, and 200 mg/kg/day to the shaved backs of five male and female albino rabbits for 6 hr/day, 5 d/wk, for three weeks. The only treatment-related effects reported were a slight to mild inflammatory and focal ulcerative effect.</p> <p>Seven male and female rats that were fed 6, 28, 130, or 600 mg/kg of imidazolidinyl urea daily for 90 days showed no differences in the haematology, urinalysis, and pathology profiles when compared to controls. However, imidazolidinyl urea induced a decrease in weight gain in males fed diets over 28 mg/kg/day</p> <p>Reproductive and/or Developmental Effects: Imidazolidinyl urea induced a slight increase in the number of resorptions and/or foetal deaths <i>in utero</i> on day 17 in female albino mice that were orally intubated with 30, 95, and 300 mg/kg from day 6 to 15 of gestation. However, no different abnormalities in soft or skeletal tissue with respect to controls were found. This compound was found to be slightly foetotoxic but not teratogenic in mice</p> <p>Genetic toxicity: Imidazolidinyl urea was mutagenic in <i>Salmonella typhimurium</i> strains TA98 and TA100 at concentrations up to 1,500 µg/plate in the presence or absence of rodent liver S-9 fraction</p> <p>Diazolidinyl urea was mutagenic in <i>Salmonella typhimurium</i> strains TA98, TA100, and TA102 with and without metabolic activation. This compound induced micronuclei in Chinese hamster V79 cells with and without metabolic activation. Diazolidinyl urea also inhibited the formation of microtubuli at 3 mmol/l. In contrast, the Cosmetic Ingredients Review Expert Panel (1990) found that diazolidinyl urea was not mutagenic in <i>S. typhimurium</i> and that this compound did not induce micronuclei</p> <p>Cytotoxicity: Imidazolidinyl urea induced a significant dose- and time-dependent decrease in cell viability of HL60 cells after 3, 6, or 24 hours of incubation at a concentration range of 0.01-1%. Apoptotic markers of cell death, DNA subdiploid content, internucleosomal DNA fragmentation, and caspase activation were observed in HL60 cells treated with low concentrations of imidazolidinyl urea (0.01% and 0.1%). However, at higher concentrations (0.5-1%), the mechanism of cell death was necrosis.</p> <p>Imidazolidinyl urea was not cytotoxic to normal human fibroblasts after incubation with 1-30% solutions for up to 30 minutes</p> <p>Formaldehyde generators (releasers) are often used as preservatives (antimicrobials, biocides, microbiocides). Formaldehyde may be generated following hydrolysis. The most widely used antimicrobial compounds function by releasing formaldehyde once inside the microbe cell. Some release detectable levels of formaldehyde into the air space, above working solutions, especially when pH has dropped.</p> <p>Many countries are placing regulatory pressure on suppliers and users to replace formaldehyde generators.</p> <p>Formaldehyde generators are a diverse group of chemicals that can be recognised by a small, easily detachable formaldehyde moiety, prepared by reacting an amino alcohol with formaldehyde ("formaldehyde-condensates"),</p>

	<p>There is concern that when formaldehyde-releasing preservatives are present in a formulation that also includes amines, such as triethanolamine (TEA), diethanolamine (DEA), or monoethanolamine (MEA), nitrosamines can be formed; nitrosamines are carcinogenic substances that can potentially penetrate skin.</p> <p>One widely-discussed hypothesis states that formaldehyde-condensate biocides, such as triazines and oxazolindines, may cause an imbalance in the microbial flora of in-use metalworking fluids (MWFs). The hypothesis further asserts that this putative microbial imbalance favours the proliferation of certain nontuberculosis mycobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, in a small percentage of susceptible workers. Symptoms of HP include flu-like illness accompanied by chronic dyspnea, i.e., difficult or laboured respiration</p> <p>According to Annex VI of the Cosmetic Directive 76/768/EC, the maximum authorised concentration of free formaldehyde is 0.2% (2000 ppm). In addition, the provisions of Annex VI state that, <i>All finished products containing formaldehyde or substances in this Annex and which release formaldehyde must be labelled with the warning "contains formaldehyde" where the concentration of formaldehyde in the finished product exceeds 0.05%.</i></p> <p>Formaldehyde-releasing preservatives have the ability to release formaldehyde in very small amounts over time. The use of formaldehyde-releasing preservatives ensures that the actual level of free formaldehyde in the products is always very low but at the same time sufficient to ensure absence of microbial growth. The formaldehyde reacts most rapidly with organic and inorganic anions, amino and sulfide groups and electron-rich groups to disrupt metabolic processes, eventually causing death of the organism.</p>
<p>5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE</p>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>No significant acute toxicological data identified in literature search.</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 cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p> <p>Considered to be the major sensitiser in Kathon CG (1)</p>
<p>2-METHYL-4-ISOTHIAZOLIN-3-ONE</p>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>No significant acute toxicological data identified in literature search.</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 cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p> <p>Considered to be a minor sensitiser in Kathon CG (1)</p>
<p>KERATIN HYDROLYSATES & C.I. ACID VIOLET 43 & WATER</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>C.I. BASIC RED 51</p>	<p>in vivo</p>
<p>C.I. BASIC RED 51</p>	<p>in vitro</p>

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C.I. BASIC RED 51	The
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE	(1). Bruze etal - Contact Dermatitis 20: 219-39, 1989

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 required to make classification available
 ✘ – Data available but does not fill the criteria for classification
 ⊘ – Data Not Available to make classification

CMR STATUS

Not Applicable

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
cetostearyl alcohol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
propylene glycol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
cetyltrimethylammonium chloride	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
d-panthenol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
vegetable protein, hydrolysed	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
dimethylsiloxane, (aminoethylpropyl)dimethoxysilyloxy-	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
keratin hydrolysates	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
wheat amino acids	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
C11-15 Pareth-7	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
glycerol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
tridecyl alcohol, ethoxylated	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
nonaethylene glycol monododecyl ether	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
HC Blue no.15	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Acid Blue 9, disodium salt	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
Basic Orange 31	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Basic Red 51	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Basic Violet 16, chloride	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
Basic Red 76	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
HC Yellow no.2	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
Basic Yellow 87	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
Basic Yellow 57	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Basic Violet 2	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Acid Violet 43	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
perfume	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
imidazolidinyl urea	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
2-methyl-4-isothiazolin-3-one	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
water	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
 Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
 Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
 For organic cationics
 Cationic substances, and their polymers and those polymers that are reasonably anticipated to become cationic in the natural aquatic environment (pH range 4-9) may be environmental hazards. Exempt from this concern are those polymers to be used only in solid phase, such as ion-exchange resins, and where the FGEW (Functional Group Equivalent Weight) of cationic groups is not 5000 and above.
 The numerous studies of aquatic toxicity, many of which were conducted in natural waters with and without added effluents, indicate that the source and composition of the test water dramatically affects the toxicity of the test substance. These results are consistent with the known behavior of these materials in the environment. Cationic substances in the environment instantaneously form

complexes with naturally occurring negatively charged constituents in sewage, soils, sediments, and with dissolved humic substances in surface waters. This complexation behavior results in reduced bioavailability in actual environmental conditions that is not adequately represented by standard laboratory assays and/or predictions by various QSAR models.

Ecotoxicity:

These chemicals, by the nature of their surfactant properties, are toxic to aquatic organisms at low concentrations. Cationic groups such as alkylsulfoniums, alkylphosphoniums and quaternary ammonium polymers are highly toxic to fish and other aquatic organisms. Similarly potentially cationic groups such as amines and isocyanates are of concern. Some cationics, however, may fall into the category of PLCs (polymers of low concern) provided they possess low charge density, and/or are not water-soluble or are not self-dispersing polycarboxylates or poly- (aromatic or aliphatic) sulfonate polymers.

The toxicity of quaternary ammonium compounds is known to be greatly reduced in the environment because of preferential binding to dissolved organics in surface water

The isothiazolinones are very toxic to marine organisms (fish, Daphnia magna and algae)

The high water solubility and low log Kow values of several chlorinated and non-chlorinated indicate a low potential for bioaccumulation.

Studies of 5-chloro-2-methyl-4-isothiazolin-3-one (CMI) in bluegill sunfish (*Lepomis macrochirus*) show BCF values of 102, 114 and 67 at nominal concentrations of 0.02, 0.12 and 0.8 mg/l. The BCF for 2-methyl-4-isothiazolin-3-one (MI) was determined at 2.3 at a nominal concentration of 0.12 mg/l

Primary biodegradation of MI and CMI occurred with half-lives of less than 24 hours in aerobic and anoxic sediments, and within a period of less than one week the parent compounds were depleted to very low levels that could not be clearly distinguished from analytical artifacts. The ultimate aerobic biodegradability of both MI and CMI attained levels of > 55% within 29 days.

Furthermore, the proposed metabolites of MI and CMI are considered to have a low aquatic toxicity on the basis of QSAR estimates and the measured toxicity of the structurally related N-(n-octyl) malonamic acid.

Proteins are generally easily biodegradable.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene glycol	LOW	LOW
d-panthenol	LOW	LOW
glycerol	LOW	LOW
nonaethylene glycol monododecyl ether	LOW	LOW
C.I. Basic Violet 2	HIGH	HIGH
C.I. Acid Violet 43	HIGH	HIGH
imidazolidinyl urea	HIGH	HIGH
5-chloro-2-methyl-4-isothiazolin-3-one	HIGH	HIGH
2-methyl-4-isothiazolin-3-one	HIGH	HIGH
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
cetostearyl alcohol	MEDIUM (BCF = 1300)
propylene glycol	LOW (BCF = 1)
d-panthenol	LOW (LogKOW = -1.9222)
glycerol	LOW (LogKOW = -1.76)
nonaethylene glycol monododecyl ether	LOW (LogKOW = 3.6722)
C.I. Basic Violet 2	HIGH (LogKOW = 4.8356)
C.I. Acid Violet 43	LOW (LogKOW = 3.0778)
imidazolidinyl urea	LOW (LogKOW = -8.2787)
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (LogKOW = 0.0444)
2-methyl-4-isothiazolin-3-one	LOW (LogKOW = -0.8767)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
propylene glycol	HIGH (KOC = 1)
d-panthenol	LOW (KOC = 10)
glycerol	HIGH (KOC = 1)
nonaethylene glycol monododecyl ether	LOW (KOC = 10)
C.I. Basic Violet 2	LOW (KOC = 1426000)
C.I. Acid Violet 43	LOW (KOC = 421.8)
imidazolidinyl urea	LOW (KOC = 10)
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (KOC = 45.15)
2-methyl-4-isothiazolin-3-one	LOW (KOC = 27.88)
water	LOW (KOC = 14.3)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Fudge Paintbox

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

Labels Required

Marine Pollutant	
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 73 / 78 and the IBC code

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	C.I. Acid Violet 43	X

SECTION 15 REGULATORY INFORMATION

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

cetostearyl alcohol(67762-27-0) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
propylene glycol(57-55-6) is found on the following regulatory lists	"Australia Exposure Standards", "Australia Inventory of Chemical Substances (AICS)", "Australia Hazardous Substances Information System - Consolidated Lists"
cetyltrimethylammonium chloride(112-02-7) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
d-panthene(81-13-0) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
vegetable protein, hydrolysed(100209-45-8) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
dimethylsiloxane, (aminoethylpropyl)dimethoxysilyloxy-(71750-80-6) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
keratin hydrolysates(69430-36-0) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
glycerol(56-81-5) is found on the following regulatory lists	"Australia Exposure Standards", "Australia Inventory of Chemical Substances (AICS)"
tridecyl alcohol, ethoxylated(24938-91-8) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
nonaethylene glycol monododecyl ether(3055-99-0) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
C.I. Acid Blue 9, disodium salt(3844-45-9) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)", "International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs"

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C.I. Basic Red 51(12270-25-6) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
C.I. Basic Violet 16, chloride(6359-45-1) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
C.I. Basic Violet 2(3248-91-7) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
C.I. Acid Violet 43(4430-18-6) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
imidazolidinyl urea(39236-46-9) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)", "Australia Hazardous Substances Information System - Consolidated Lists"
5-chloro-2-methyl-4-isothiazolin-3-one(26172-55-4) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
2-methyl-4-isothiazolin-3-one(2682-20-4) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
water(7732-18-5) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (tridecyl alcohol, ethoxylated; dimethylsiloxane, (aminoethylpropyl)dimethoxysilyloxy-)
Japan - ENCS	N (keratin hydrolysates; water; imidazolidinyl urea; vegetable protein, hydrolysed; dimethylsiloxane, (aminoethylpropyl)dimethoxysilyloxy-)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	N (vegetable protein, hydrolysed)
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
cetostearyl alcohol	67762-27-0, 8005-44-5
cetyltrimethylammonium chloride	112-02-7, 139272-33-6, 53023-95-3, 79728-63-5
d-panthenol	16485-10-2, 17307-32-3, 81-13-0
glycerol	29796-42-7, 30049-52-6, 37228-54-9, 56-81-5, 75398-78-6, 78630-16-7, 8013-25-0
tridecyl alcohol, ethoxylated	24938-91-8, 9067-13-4
C.I. Acid Blue 9, disodium salt	3844-45-9, 70992-30-2
C.I. Basic Red 51	12270-25-6, 77061-58-6
C.I. Basic Violet 16, chloride	51258-23-2, 56451-40-2, 6359-45-1
C.I. Basic Violet 2	100359-07-7, 3248-91-7
C.I. Acid Violet 43	12701-65-4, 4430-18-6, 63310-00-9

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.

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

The (M)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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