

Cetrigen Antibacterial Wound Spray

Virbac (Australia) Pty Limited

Chemwatch Hazard Alert Code: 2

Chemwatch: 5496-93

Version No: 3.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 10/26/2021

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L.GHS.AUS.EN

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

Product Identifier

| | |
|-------------------------------|------------------------------------|
| Product name | Cetrigen Antibacterial Wound Spray |
| Chemical Name | Not Applicable |
| Synonyms | APVMA No.: 45797 |
| Chemical formula | Not Applicable |
| Other means of identification | Not Available |

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

| | |
|--------------------------|--|
| Relevant identified uses | An antibacterial and insect repellent spray with purple dye for use on open wounds in all animals. SDS are intended for use in the workplace. For domestic-use products, refer to consumer labels. |
|--------------------------|--|

Details of the supplier of the safety data sheet

| | |
|-------------------------|--|
| Registered company name | Virbac (Australia) Pty Limited |
| Address | 361 Horsley Road Milperra NSW 2214 Australia |
| Telephone | 1800 242 100 |
| Fax | +61 2 9772 9773 |
| Website | au.virbac.com |
| Email | customercare@virbac.com.au |

Emergency telephone number

| | |
|-----------------------------------|----------------------------|
| Association / Organisation | Poisons Information Centre |
| Emergency telephone numbers | 13 11 26 |
| Other emergency telephone numbers | Not Available |

SECTION 2 Hazards identification

Classification of the substance or mixture


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

ChemWatch Hazard Ratings

| | Min | Max | |
|--------------|-----|-----|--------------|
| Flammability | 0 | | |
| Toxicity | 1 | | 0 = Minimum |
| Body Contact | 2 | | 1 = Low |
| Reactivity | 0 | | 2 = Moderate |
| Chronic | 0 | | 3 = High |
| | | | 4 = Extreme |

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

Label elements

| | |
|---------------------|---|
| Hazard pictogram(s) |  |
| Signal word | Warning |

Hazard statement(s)

Cetrigen Antibacterial Wound Spray

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|------|--|
| H315 | Causes skin irritation. |
| H319 | Causes serious eye irritation. |
| H412 | Harmful to aquatic life with long lasting effects. |

Precautionary statement(s) Prevention

| | |
|------|--|
| P273 | Avoid release to the environment. |
| P280 | Wear protective gloves, protective clothing, eye protection and face protection. |
| P264 | Wash all exposed external body areas thoroughly after handling. |

Precautionary statement(s) Response

| | |
|----------------|--|
| P305+P351+P338 | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| P337+P313 | If eye irritation persists: Get medical advice/attention. |
| P302+P352 | IF ON SKIN: Wash with plenty of water. |
| P332+P313 | If skin irritation occurs: Get medical advice/attention. |
| P362+P364 | Take off contaminated clothing and wash it before reuse. |

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

| | |
|------|--|
| P501 | Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation. |
|------|--|

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

| CAS No | %[weight] | Name |
|---------------|-----------|--|
| 57-55-6 | 10-30 | <u>propylene glycol</u> |
| 134-62-3 | 1-10 | <u>N,N-diethyl-m-toluamide</u> |
| 68439-49-6 | 1-5 | <u>alcohols C16-18 ethoxylated</u> |
| 1119-97-7 | <1 | <u>tetradecyltrimethylammonium bromide</u> |
| Not Available | >60 | Ingredients determined not to be hazardous |

Legend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures

Description of first aid measures

| | |
|---------------------|--|
| Eye Contact | <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| Skin Contact | <p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor. |
| 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"> ▶ For advice, contact a Poisons Information Centre or a doctor at once. ▶ Urgent hospital treatment is likely to be needed. ▶ 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. ▶ Transport to hospital or doctor without delay. |

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

Cetrigen Antibacterial Wound Spray

SECTION 5 Firefighting measures

Extinguishing media

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

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

In such an event consider:

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

Special hazards arising from the substrate or mixture

| | |
|-----------------------------|-------------|
| Fire Incompatibility | None known. |
|-----------------------------|-------------|

Advice for firefighters

| | |
|------------------------------|--|
| Fire Fighting | <ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use. |
| Fire/Explosion Hazard | <ul style="list-style-type: none"> ▶ The material is not readily combustible under normal conditions. ▶ However, it will break down under fire conditions and the organic component may burn. ▶ Not considered to be a significant fire risk. ▶ Heat may cause expansion or decomposition with violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <p>Decomposes on heating and produces toxic fumes of: carbon dioxide (CO₂) hydrogen bromide nitrogen oxides (NO_x) sulfur oxides (SO_x) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</p> |
| HAZCHEM | Not Applicable |

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

| | |
|---------------------|--|
| Minor Spills | <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal. |
| Major Spills | <p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services. |

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

SECTION 7 Handling and storage

Precautions for safe handling

| | |
|----------------------|---|
| Safe handling | <ul style="list-style-type: none"> ▶ 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. ▶ Prevent concentration in hollows and sumps. |
|----------------------|---|

Cetrigen Antibacterial Wound Spray

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| | <ul style="list-style-type: none"> ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. |
| Other information | <ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry area protected from environmental extremes. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. <p>For major quantities:</p> <ul style="list-style-type: none"> ▶ Consider storage in banded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). ▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities. |

Conditions for safe storage, including any incompatibilities

| | |
|--------------------------------|--|
| Suitable container | <ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks. |
| Storage incompatibility | <p>Alcohols</p> <ul style="list-style-type: none"> ▶ are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents. ▶ reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen ▶ react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium ▶ should not be heated above 49 deg. C. when in contact with aluminium equipment |

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: particulates only | 10 mg/m ³ | Not Available | Not Available | Not Available |
| Australia Exposure Standards | propylene glycol | Propane-1,2-diol total: (vapour & particulates) | 150 ppm / 474 mg/m ³ | Not Available | Not Available | Not Available |

Emergency Limits

| Ingredient | TEEL-1 | TEEL-2 | TEEL-3 |
|-------------------------------------|-----------------------|-------------------------|-------------------------|
| propylene glycol | 30 mg/m ³ | 1,300 mg/m ³ | 7,900 mg/m ³ |
| alcohols C16-18 ethoxylated | 3.8 mg/m ³ | 42 mg/m ³ | 250 mg/m ³ |
| tetradecyltrimethylammonium bromide | 12 mg/m ³ | 130 mg/m ³ | 770 mg/m ³ |

| Ingredient | Original IDLH | Revised IDLH |
|-------------------------------------|---------------|---------------|
| propylene glycol | Not Available | Not Available |
| N,N-diethyl-m-toluamide | Not Available | Not Available |
| alcohols C16-18 ethoxylated | Not Available | Not Available |
| tetradecyltrimethylammonium bromide | Not Available | Not Available |

Occupational Exposure Banding

| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|-------------------------------------|-----------------------------------|----------------------------------|
| N,N-diethyl-m-toluamide | E | ≤ 0.1 ppm |
| alcohols C16-18 ethoxylated | E | ≤ 0.1 ppm |
| tetradecyltrimethylammonium bromide | E | ≤ 0.01 mg/m ³ |

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

MATERIAL DATA

Exposure controls

| | |
|---|--|
| Appropriate engineering controls | 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. |
|---|--|

Cetrigen Antibacterial Wound Spray

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

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.

Employers may need to use multiple types of controls to prevent employee overexposure.

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.

An approved self contained breathing apparatus (SCBA) may be required in some situations.

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.

| 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.) |

Within each range the appropriate value depends on:

| Lower end of the range | Upper end of the range |
|--|----------------------------------|
| 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents |
| 2: Contaminants of low toxicity or of nuisance value only. | 2: Contaminants of high toxicity |
| 3: Intermittent, low production. | 3: High production, heavy use |
| 4: Large hood or large air mass in motion | 4: Small hood-local control only |

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection



Eye and face protection

- ▶ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.
- ▶ Chemical goggles whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted.
- ▶ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.
- ▶ Alternatively a gas mask may replace splash goggles and face shields.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- ▶ Elbow length PVC gloves

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.

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.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- 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.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

Cetrigen Antibacterial Wound Spray

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| | <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> |
| 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. |

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:

Cetrigen Antibacterial Wound Spray

| Material | CPI |
|------------------|-----|
| BUTYL | C |
| NATURAL RUBBER | C |
| NATURAL+NEOPRENE | C |
| NEOPRENE | C |
| NEOPRENE/NATURAL | C |
| NITRILE | C |
| PE/EVAL/PE | C |
| PVA | C |
| PVC | 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)

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

| | | | |
|---|---|--|----------------|
| Appearance | Milky violet opaque liquid; mixes with water. | | |
| Physical state | Liquid | Relative density (Water = 1) | Not Available |
| Odour | Not Available | Partition coefficient n-octanol / water | Not Available |
| Odour threshold | Not Available | Auto-ignition temperature (°C) | Not Applicable |
| pH (as supplied) | 4.8-5.5 | Decomposition temperature | Not Available |
| Melting point / freezing point (°C) | Not Applicable | Viscosity (cSt) | Not Available |
| Initial boiling point and boiling range (°C) | Not Available | Molecular weight (g/mol) | Not Applicable |
| Flash point (°C) | Not Applicable | Taste | Not Available |
| Evaporation rate | Not Available | Explosive properties | Not Available |
| Flammability | Not Applicable | Oxidising properties | Not Available |
| Upper Explosive Limit (%) | Not Applicable | Surface Tension (dyn/cm or mN/m) | Not Available |
| Lower Explosive Limit (%) | Not Applicable | Volatile Component (%vol) | Not Available |

Cetrigen Antibacterial Wound Spray

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|--------------------------|---------------|----------------------|---------------|
| Vapour pressure (kPa) | Not Available | Gas group | Not Available |
| Solubility in water | Miscible | pH as a solution (%) | 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>The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severe damage to the health of the individual. Relatively small amounts absorbed through the lungs may prove fatal.</p> <p>Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.</p> |
| Ingestion | <p>Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.</p> <p>Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality</p> <p>Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.</p> <p>Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.</p> <p>Ingestion of propylene glycol produced reversible central nervous system depression in humans following ingestion of 60 ml. Symptoms included increased heart-rate (tachycardia), excessive sweating (diaphoresis) and grand mal seizures in a 15 month child who ingested large doses (7.5 ml/day for 8 days) as an ingredient of vitamin preparation.</p> <p>Excessive repeated ingestions may cause hypoglycaemia (low levels of glucose in the blood stream) among susceptible individuals; this may result in muscular weakness, incoordination and mental confusion.</p> <p>Very high doses given during feeding studies to rats and dogs produce central nervous system depression (although one-third of that produced by ethanol), haemolysis and insignificant kidney changes.</p> <p>In humans propylene glycol is partly excreted unchanged in the urine and partly metabolised as lactic and pyruvic acid. Lactic acidosis may result.</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>Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>Limited evidence suggests that repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> |
| Eye | <p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> |

Cetrigen Antibacterial Wound Spray

Chronic

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.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.

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.

Principal routes of exposure are usually by inhalation of mists or vapours from heated material and skin contact/absorption.

A 5 year old girl sprayed with Deet nightly for 3 months, developed headaches and slurred speech, progressing to athetosis (ceaseless slow, writhing motions especially of the hands), shaking, screaming and convulsion. She died 24 days after hospitalisation; autopsy revealed generalised oedema of the brain with intense congestion of the meninges. The effect was thought to represent sensitisation to the substance. Repeated application to human skin resulted in slight irritation and dryness of the face, desquamation around the nose and a slight tingling sensation. Incidences of sporadic allergy (anaphylaxis) and scarring dermatitis have been reported. Some individuals repeatedly exposed to the substance have shown encephalopathy and neurological symptoms (muscle cramp, urinary hesitation, insomnia, abnormal sweating, irritability, depression, paranoia, episodes of confusion, and aggressive behaviour).

An increased incidence sperm head abnormalities and period nausea, vomiting and nasal exudate were observed in animals following chronic exposure

Chronic intoxication with ionic bromides, historically, has resulted from medical use of bromides but not from environmental or occupational exposure; depression, hallucinosis, and schizophreniform psychosis can be seen in the absence of other signs of intoxication. Bromides may also induce sedation, irritability, agitation, delirium, memory loss, confusion, disorientation, forgetfulness (aphasias), dysarthria, weakness, fatigue, vertigo, stupor, coma, decreased appetite, nausea and vomiting, diarrhoea, hallucinations, an acne like rash on the face, legs and trunk, known as bronchoderma (seen in 25-30% of case involving bromide ion), and a profuse discharge from the nostrils (coryza). Ataxia and generalised hyperreflexia have also been observed. Correlation of neurologic symptoms with blood levels of bromide is inexact. The use of substances such as brompheniramine, as antihistamines, largely reflect current day usage of bromides; ionic bromides have been largely withdrawn from therapeutic use due to their toxicity.

In test animals, brominated vegetable oils (BVOs), historically used as emulsifiers in certain soda-based soft drinks, produced damage to the heart and kidneys in addition to increasing fat deposits in these organs. In extreme cases BVO caused testicular damage, stunted growth and produced lethargy and fatigue.

Brominism produces slurred speech, apathy, headache, decreased memory, anorexia and drowsiness, psychosis resembling paranoid schizophrenia, and personality changes

Several cases of foetal abnormalities have been described in mothers who took large doses of bromides during pregnancy.

Reproductive effects caused by bromide (which crosses the placenta) include central nervous system depression, brominism, and bronchoderma in the newborn.

| Cetrigen Antibacterial Wound Spray | TOXICITY | IRRITATION |
|-------------------------------------|---|--|
| | Not Available | Not Available |
| propylene glycol | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 11890 mg/kg ^[2] | Eye (rabbit): 100 mg - mild |
| | Inhalation(Rat) LC50; >44.9 mg/L4h ^[2] | Eye (rabbit): 500 mg/24h - mild |
| | Oral(Rat) LD50; 20000 mg/kg ^[2] | Eye: no adverse effect observed (not irritating) ^[1] |
| | | Skin(human):104 mg/3d Intermittent Mod |
| | | Skin(human):500 mg/7days mild |
| | | Skin: no adverse effect observed (not irritating) ^[1] |
| N,N-diethyl-m-toluamide | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 3180 mg/kg ^[2] | Eye (rabbit) : 10 mg - moderate |
| | Oral(Rat) LD50; 1950 mg/kg ^[2] | Eye (rabbit): 100 mg |
| | | Skin (rabbit): 500 mg - moderate |
| alcohols C16-18 ethoxylated | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: >3000 mg/kg ^[1] | Eye : Severe (analogy) * |
| | Inhalation(Rat) LC50; >1.6 mg/l4h ^[1] | Skin: not irritating * (analogy) * |
| | Oral(Rat) LD50; 1260 mg/kg ^[2] | |
| tetradecyltrimethylammonium bromide | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 4300 mg/kg ^[1] | Eye: adverse effect observed (irritating) ^[1] |
| | Oral(Rat) LD50; 390 mg/kg ^[1] | Skin: adverse effect observed (irritating) ^[1] |

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

PROPYLENE GLYCOL

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. 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

Cetrigen Antibacterial Wound Spray

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.

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).

Propylene glycol shows no evidence of being a carcinogen or of being genotoxic.

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.

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

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 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 oestrogen 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 had 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 N,N-diethyl-m-toluamide (Deet)

Acute toxicity: Different preparations of Deet with different proportions of the m-isomer produced different oral LD50s. Rats killed by dosages in the LD50 range showed lacrimation, chromodacryorrhea, depression, prostration, tremors, and asphyxial convulsions.

Respiratory failure usually preceded cardiac failure.

In rabbits, an intravenous dosage of 75 mg/kg was rapidly fatal, but 50 mg/kg was not. Five doses at the rate of 25 mg/kg/day produced no cumulative effect, except for injury of the intima of some veins used for injection. Single dermal applications to rabbits at rates of 2 or 4 ml/kg produced no systemic effect, but did produce mild to moderate erythema. Repeated dermal application of 50% solutions for 13 weeks at the rate of 2 ml/kg/day produced no evidence of systemic toxicity but did produce desquamation, coriaceousness, dryness, and fissuring in the same species. Except for some scarring, these lesions cleared within 3 weeks. Instillation of Deet into the eyes of rabbits produced mild to moderate edema of the nictitating membrane, lacrimation, conjunctivitis, and some corneal injury, as revealed by fluorescein staining. After 5 days, all eyes appeared normal. No sensitisation was seen in guinea pigs.

Animals topically exposed to Deet have developed dermal and ocular reactions. Dermal effects including erythema, desquamation and scarring in rabbits and profuse sweating, irritation and exfoliation in horses have been reported following repeated applications of Deet at concentrations of 50 percent or greater. Direct ocular application of either diluted (30 or 40 percent Deet) or undiluted Deet in rabbits has produced edema, tearing, conjunctivitis, pus and clouding in the eyes.

Repeated dermal application to horses produced hypersteatosis, an overactivity of the sebaceous glands, when the solution of Deet was 15% or higher.

Dermal application in humans of insect repellents containing Deet can produce a variety of skin reactions in humans. Cases of localized skin irritation, large painful blisters and permanent scarring of skin at the crease of the elbow have been reported in soldiers who applied solutions of 50 or 75 percent Deet. Results from questionnaire surveys conducted by the National Institute for Occupational Safety and Health (NIOSH) among Everglades National Park Employees indicated a variety of dermal reactions including rashes, irritation of skin and mucous membranes, and numb or burning sensations of the lips among park workers who were highly exposed to Deet-containing repellents. Urticaria or dermatitis, resulting from topical Deet exposure has been noted in both children and adults. In one instance involving only limited Deet exposure, the urticaria was accompanied by an anaphylactic reaction.

Controlled human exposure studies using 50 or 75 percent Deet have reproduced many of the dermal effects noted in field studies. The U.S. Army conducted an investigation in volunteers using 75 percent Deet applied to the upper arm and elbow's crease. Of the 77 volunteers, 37 (48%) had severe dermal reactions at the crease of the elbow. No dermal reactions were observed on the upper arm or in the control group of men tested with ethanol solvent alone.

Several cases of toxic encephalopathy associated with the use of Deet in children have been reported in the medical literature. The first reported case involved a 3.5 year old girl whose body, bedclothes and bedding were sprayed each night for two weeks with an insect repellent containing 15 percent Deet. Since then, five additional cases of toxic encephalopathy have been temporally associated with the use of Deet products in children, all of whom were females. The toxic encephalopathy was characterised by agitation, weakness, disorientation, ataxia, seizures, coma and in three cases resulted in death. Autopsies conducted on two fatalities indicated oedema of the brain, with one case presenting necrotic lesions in the cerebellum and spinal cord and an enlarged liver accompanied by microscopic changes. One child was reported to be heterozygous for ornithine carbamoyl transferase deficiency (a sex linked enzyme deficiency which may produce effects similar to those reported above) and it has been hypothesised that children with this enzyme disorder may be at greater risk of adverse reactions to Deet. This enzyme deficiency which usually causes infant death in males is of variable severity in females. Accidental and deliberate ingestion of Deet-containing products has produced neurotoxic effects similar to those described following dermal exposure.

Generalised seizures have also been temporally associated with the use of Deet-containing insect repellent on skin. These cases differ from those described above in that they involved males (four boys aged 3-7 years and one 29-year-old adult), had few associated neurotoxic effects and resolved rapidly. Lower exposure to Deet in these males (four of five males had either one or two dermal applications) may have accounted for the effects being less severe than in females. That the majority of identified neurotoxic cases involved children raises concerns that this subpopulation is at greater risk of adverse reaction following exposure to Deet than are adults. Signs and symptoms of more subtle neurotoxicity have also been associated with extensive dermal application of Deet in adults. Questionnaire results indicate that Everglades National Park employees having extensive Deet exposure were more likely to have insomnia, mood disturbances and impaired cognitive function than were lesser exposed co-workers. A young male who repeatedly applied Deet to his skin prior to spending prolonged periods in a sauna was reported to develop acute manic psychosis characterized by aggressive behavior, delusions and hyperactivity.

Either o-DET or p-DET, or both occur as impurities in commercial m-DET (Deet). A thorough study of the o- and p-isomers showed that the o-isomer is slightly more toxic than the others (oral LD50 1,210 mg/kg in rats). However, no alarming difference was found, and it was concluded that the presence of 5% of o-DET or p-DET as impurities in the

Chronic toxicity: When rats were fed Deet at a dietary level of 10,000 ppm for about 200 days, their growth rate was decreased without a decrease in food intake. There was a significant increase in the relative weight of the testes and liver in males, of the liver and spleen in females, and the kidneys of both males and females. Some of these changes were seen in lesser degree at a dietary level of 1,000 ppm.

N,N-DIETHYL-M-TOLUAMIDE

No gross or significant histological changes were seen at any dietary level and no changes of any kind were noted at 100 ppm or 500 ppm (about 25 mg/kg/day).

Essentially identical results were found in other subacute dermal and feeding studies each carried out on rats, rabbits, and dogs. In these oral studies, 2,000 ppm proved to be a no-effect-level. Oral administration of Deet to dogs at rates of 100 and 300 mg/kg/day caused tremor and hyperactivity and occasional vomiting, but no other effects. Blood studies (hemoglobin, haematocrit, sedimentation rate, platelet counts, total and differential white cell counts) on dogs receiving 300 mg/kg orally or dermally or on rabbits receiving 300 mg/kg dermally revealed no effect on the haematopoietic system. Gross and microscopic examination of the organs of all three species revealed only slight kidney damage in rabbits typical of that associated with burns of the skin. Thirteen other organs, including liver, spleen, and bone marrow, were normal in the three species.

No systemic toxicity was observed in rats exposed 8 hours/day, 5 days/week for 7 weeks to air saturated with Deet. No toxic effects were observed in rats exposed for 6 hours to an aerosol of Deet. No gross or significant histological changes were seen.

Organ Toxicity: Hypertrophy of the kidneys and liver and effects of mild central nervous system stimulation including tremors and hyperactivity were noted in animals following repeated exposure. Significant testicular hypertrophy was observed in male rats repeatedly fed a diet containing from 48 to 531 mg/kg/day of Deet.

Reproductive Effects: When Deet was applied to the skin of rats at the rate of 1,000 mg/kg/day throughout pregnancy, implantation was reduced significantly. Prenatal mortality was 34.1%, compared with 20.9% in the control. Mortality between birth and weaning was 44.0%, compared to 15.7% in the control. Injury was less (but probably significant) at a dosage of 100 mg/kg/day throughout pregnancy.

Teratogenic Effects: A dermal teratology study was conducted on rabbits. Groups of 20 pregnant rabbits received daily dermal applications of 0, 50, 100, 500, 1000, or 5000 mg Deet/kg/day in ethanol on shaved backs from day 0 through day 29 of gestation. There were no significant differences between control and treated animals with respect to the fertility index, number of implantations per animal, or number of fetuses per animal. In addition, treatment did not change fetal weight, fetal length or placental weights and no increases in the incidence of skeletal or soft tissue anomalies were observed in treated groups when compared with untreated controls. This study demonstrated that Deet has no teratogenic or embryotoxic effects in rabbits exposed dermally to technical Deet.

An additional supplementary teratology study was conducted on rats. Groups of 20 pregnant rats were daily administered 10 ml of peanut oil containing 0, 8, 20 or 80 mg/kg/Deet by gavage from day 5 through day 15 of gestation. No significant differences were reported between control and treated mothers with respect to fertility, fetuses per litter, foetal weight or fetal survival. However, the study did show decreases in number of implantation sites per dam and number of fetuses per animal. In addition, a related increase was observed in the number of resorptions per dam.

Carcinogenicity: Researchers fed Deet to male and female rats in the diet for two years at doses of 10, 30, or 100 mg/kg/day, and 30, 100, or 400 mg/kg/day, respectively. Researchers fed mice 250, 500, or 1,000 mg/kg/day for 18 months, and dogs 30, 100, or 400 mg/kg/day. No specific target organ toxicity or oncogenicity was observed in any of the animals. Researchers often use studies designed to test for mutagenicity to screen chemicals for carcinogenicity. Sufficient evidence indicates that DEET does not have significant potential for mutagenicity.

Fate in Humans and Animals: Deet is absorbed promptly from the skin and distributed to all organs including the brain and the foetus. The compound is excreted in the milk but primarily in the urine.

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

Reproductive effector in rats

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

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

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

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

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

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

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

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

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

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

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

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their

ALCOHOLS C16-18 ETHOXYLATED

Cetrigen Antibacterial Wound Spray

corresponding aldehydes in the oxidation mixture .

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

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

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

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

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

>20 EO is not classified (CESIO 2000)

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

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

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

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

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

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

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

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

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

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

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

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

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

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

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

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats. In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular

Cetrigen Antibacterial Wound Spray

hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

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

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

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

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.

Remarks: Patch test on human volunteers did not demonstrate sensitization properties. * Cognis MSDS for Ceteraeth -20 The skin sensitising potential was assessed with C16-18AE (CAS 68439-49-6) in a Buehler Test according to OECD Guideline 406. In this study 20 female guinea pigs were induced by an epicutaneous occlusive dressing with 100% test substance (in maize oil) for 6 h on Day 0, 7 and 14. Two weeks after the last induction animals were challenged by epicutaneous occlusive exposure for 6 h to 100% test substance (in maize oil). 24 and 48 h after patch removal the application site was assessed for signs of local irritation. No dermal reactions were observed in any test animal at any time point. Available oral toxicity studies provide a coherent picture on the subchronic and chronic oral toxicity of AE. Based on the described effects and argumentations, the dietary NOAEL of 500 mg/kg bw/day (Shell, 1982) representing an average of all NOAELs, was chosen for the risk assessment. The clastogenic potential was assessed in a chromosomal aberration test with C16-18AE (CAS 68439-49-6) in mammalian cells according to OECD Guideline 473. Chinese hamster ovary cells (CHO) were exposed to 313, 625, 1250, 2500 and 5000 µg/mL in the presence and 1.25, 2.5, 5, 10, 20, 39 and 78 µg/mL in the absence of metabolic activation. Positive and vehicle (1% ethanol) control cultures were included in each assay. No increases in the number of chromosome aberrations in the presence or absence of metabolic activation were seen at any concentration tested. Appropriate reference mutagens used as positive controls showed a significant increase in chromosome aberrations, thus indicating the sensitivity of the assay, and the efficacy of the S9-mix. Hence, the test substance can not be regarded as clastogenic. The mutagenic potential in mammalian cells was assessed with C16-18AE (CAS 68439-49-6) by a HPRT-assay according to OECD Guideline 476. Following pre-tests with the concentration ranging from 1-100 µg/mL, the latter being the solubility limit of the test substance, Chinese hamster ovary cells were exposed for 4 h to concentrations of 1.8, 6, 18, 60 and 100 µg/mL in the absence and presence of metabolic activation by rat liver S9-mix. No dose-related increases in mutant colony numbers were obtained in two independent experiments with the test substance in either the presence or absence of S9-mix. Appropriate reference mutagens used as positive controls produced highly significant increases in mutation frequency, thus indicating the sensitivity of the assay. Therefore, the test substance is regarded as not mutagenic in mammalian cells. In conclusion, C16AE (CAS 52609-19-5) is regarded as non-genotoxic a reproductive toxicity study on a structurally similar material, C14-15AE7 (CAS 68951-67-7) was conducted at dietary levels of 25, 50 and 250 mg/kg bw/day. The 2-generation study (Procter and Gamble Ltd., 1977: Long term reproduction and teratology study in rats with Neodol 45-7; unpublished report) did not show any potential for reproductive toxicity at the tested dose levels. The NOAEL for reproductive effects was greater than the highest tested dose of 250 mg/kg bw/day. Although the study was pre-GLP and not in full compliance with current OECD guidelines, the study provided sufficient information and was assessed to be scientifically reliable. The comparable toxicokinetic and metabolic profiles, as well as their toxicological similarities for this and other toxicological endpoints, support the conclusion that insights from the reproductive toxicity study on higher ethoxylated AE are applicable to AE with an ethoxylation degree of 1 - 2.5. * REACH Dossier

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 alkyltrimethylammonium 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 alkyltrimethylammonium 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 alkyltrimethylammonium 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 alkyltrimethylammonium 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

**TETRADECYLTRIMETHYLAMMONIUM
BROMIDE**

Cetrigen Antibacterial Wound Spray

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 alkyltrimethylammonium salts are less irritating to the rabbit eye than the shorter alkyl chain homologues. C10 ATMAC, C12 ATMAC, 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 ATMAC 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 ATMAC 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 ATMAC 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 ATMAC 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 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.

PROPYLENE GLYCOL & N,N-DIETHYL-M-TOLUAMIDE

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.

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

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

SECTION 12 Ecological information

Toxicity

Cetrigen Antibacterial Wound Spray

| Cetrigen Antibacterial Wound Spray | Endpoint | Test Duration (hr) | Species | Value | Source |
|------------------------------------|---------------|--------------------|---------------|---------------|---------------|
| | Not Available | Not Available | Not Available | Not Available | Not Available |

| propylene glycol | Endpoint | Test Duration (hr) | Species | Value | Source |
|------------------|-----------|--------------------|-------------------------------|------------|--------|
| | NOEC(ECx) | 336h | Algae or other aquatic plants | <5300mg/l | 1 |
| | EC50 | 72h | Algae or other aquatic plants | 19300mg/l | 2 |
| | LC50 | 96h | Fish | >10000mg/l | 2 |
| | EC50 | 48h | Crustacea | >114.4mg/L | 4 |
| | EC50 | 96h | Algae or other aquatic plants | 19000mg/l | 2 |

| N,N-diethyl-m-toluamide | Endpoint | Test Duration (hr) | Species | Value | Source |
|-------------------------|-----------|--------------------|-----------|-----------------|--------|
| | BCF | 1008h | Fish | 0.8-2.4 | 7 |
| | EC50 | 48h | Crustacea | 55.776-99.6mg/L | 4 |
| | LC50 | 96h | Fish | 70.965mg/L | 4 |
| | NOEC(ECx) | 504h | Crustacea | 3.7mg/l | 4 |

| alcohols C16-18 ethoxylated | Endpoint | Test Duration (hr) | Species | Value | Source |
|-----------------------------|-----------|--------------------|-------------------------------|----------|--------|
| | EC20(ECx) | 72h | Algae or other aquatic plants | 0.06mg/l | 2 |
| | EC50 | 72h | Algae or other aquatic plants | >100mg/l | 2 |
| | LC50 | 96h | Fish | 108mg/l | 2 |

| tetradecyltrimethylammonium bromide | Endpoint | Test Duration (hr) | Species | Value | Source |
|-------------------------------------|-----------|--------------------|---------|-----------|--------|
| | LC50 | 96h | Fish | >1.81mg/l | 2 |
| | EC10(ECx) | 96h | Fish | 0.72mg/l | 2 |

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|-------------------------|-------------------------|------------------|
| propylene glycol | LOW | LOW |
| N,N-diethyl-m-toluamide | HIGH | HIGH |

Bioaccumulative potential

| Ingredient | Bioaccumulation |
|-------------------------|-----------------|
| propylene glycol | LOW (BCF = 1) |
| N,N-diethyl-m-toluamide | LOW (BCF = 2.4) |

Mobility in soil

| Ingredient | Mobility |
|-------------------------|-------------------|
| propylene glycol | HIGH (KOC = 1) |
| N,N-diethyl-m-toluamide | LOW (KOC = 536.6) |

SECTION 13 Disposal considerations

Waste treatment methods

| Product / Packaging disposal | Disposal instructions |
|------------------------------|---|
| | <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed. ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. |

SECTION 14 Transport information**Labels Required**

| | |
|-------------------------|----------------|
| Marine Pollutant | NO |
| HAZCHEM | Not Applicable |

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

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

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

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

Not Applicable

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

| Product name | Group |
|-------------------------------------|---------------|
| propylene glycol | Not Available |
| N,N-diethyl-m-toluamide | Not Available |
| alcohols C16-18 ethoxylated | Not Available |
| tetradecyltrimethylammonium bromide | Not Available |

Transport in bulk in accordance with the ICG Code

| Product name | Ship Type |
|-------------------------------------|---------------|
| propylene glycol | Not Available |
| N,N-diethyl-m-toluamide | Not Available |
| alcohols C16-18 ethoxylated | Not Available |
| tetradecyltrimethylammonium bromide | Not Available |

SECTION 15 Regulatory information

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

propylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

N,N-diethyl-m-toluamide is found on the following regulatory lists

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

Australian Inventory of Industrial Chemicals (AIIC)

alcohols C16-18 ethoxylated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

tetradecyltrimethylammonium bromide is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

| National Inventory | Status |
|---|--|
| Australia - AIIC / Australia Non-Industrial Use | Yes |
| Canada - DSL | Yes |
| Canada - NDSL | No (propylene glycol; N,N-diethyl-m-toluamide; alcohols C16-18 ethoxylated; tetradecyltrimethylammonium bromide) |
| China - IECSC | Yes |
| Europe - EINEC / ELINCS / NLP | Yes |
| Japan - ENCS | Yes |
| Korea - KECI | Yes |
| New Zealand - NZIoC | Yes |
| Philippines - PICCS | No (tetradecyltrimethylammonium bromide) |
| USA - TSCA | Yes |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (alcohols C16-18 ethoxylated; tetradecyltrimethylammonium bromide) |
| Vietnam - NCI | Yes |
| Russia - FBEPH | No (alcohols C16-18 ethoxylated) |

| National Inventory | Status |
|--------------------|---|
| Legend: | Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration. |

SECTION 16 Other information

| | |
|---------------|------------|
| Revision Date | 10/26/2021 |
| Initial Date | 10/22/2021 |

Other information

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

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
 PC—STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit.
 IDLH: Immediately Dangerous to Life or Health Concentrations
 ES: Exposure Standard
 OSF: Odour Safety Factor
 NOAEL :No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index
 AIIC: Australian Inventory of Industrial Chemicals
 DSL: Domestic Substances List
 NDSL: Non-Domestic Substances List
 IECSC: Inventory of Existing Chemical Substance in China
 EINECS: European INventory of Existing Commercial chemical Substances
 ELINCS: European List of Notified Chemical Substances
 NLP: No-Longer Polymers
 ENCS: Existing and New Chemical Substances Inventory
 KECI: Korea Existing Chemicals Inventory
 NZIoC: New Zealand Inventory of Chemicals
 PICCS: Philippine Inventory of Chemicals and Chemical Substances
 TSCA: Toxic Substances Control Act
 TCSI: Taiwan Chemical Substance Inventory
 INSQ: Inventario Nacional de Sustancias Químicas
 NCI: National Chemical Inventory
 FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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