# Butec OTM Pain Relief for Cattle and Sheep

## Troy Laboratories Pty Ltd

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5581-87**
Version No: **3.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **03/03/2025**
Print Date: **03/03/2025**
L.GHS.AUS.EN.E

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

### Product Identifier

| | |
|---|---|
| **Product name** | Butec OTM Pain Relief for Cattle and Sheep |
| **Chemical Name** | Not Applicable |
| **Synonyms** | ilium Buccalgesic OTM; APVMA number 69874; APVMA number 93151 |
| **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** | For the alleviation of pain associated with routine husbandry procedures in cattle and sheep. To be used as directed on product label. |

### Details of the manufacturer or supplier of the safety data sheet

| | |
|---|---|
| **Registered company name** | **Troy Laboratories Pty Ltd** |
| **Address** | 37 Glendenning Road Glendenning NSW 2761 Australia |
| **Telephone** | 02 8808 3600 |
| **Fax** | 02 9677 9300 |
| **Website** | www.Troylab.com.au |
| **Email** | admin@troylab.com.au |

### Emergency telephone number

| | |
|---|---|
| **Association / Organisation** | **Ixom Emergency Response Service** |
| **Emergency telephone number(s)** | 1800 033 111 (24 hours) |
| **Other emergency telephone number(s)** | Not Available |

## SECTION 2 Hazards identification

### Classification of the substance or mixture

| | |
|---|---|
| **Poisons Schedule** | S6 |
| **Classification [1]** | Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Carcinogenicity Category 2 |
| ***Legend:*** | ***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*** |

### Label elements

| | |
|---|---|
| **Hazard pictogram(s)** |  |

**Page 1 continued...**

| | |
|---|---|
| **Signal word** | **Warning** |

### Hazard statement(s)

| | |
|---|---|
| **H315** | Causes skin irritation. |
| **H317** | May cause an allergic skin reaction. |
| **H319** | Causes serious eye irritation. |
| **H351** | Suspected of causing cancer. |

### Precautionary statement(s) Prevention

| | |
|---|---|
| **P201** | Obtain special instructions before use. |
| **P280** | Wear protective gloves, protective clothing, eye protection and face protection. |
| **P261** | Avoid breathing mist/vapours/spray. |
| **P264** | Wash all exposed external body areas thoroughly after handling. |
| **P272** | Contaminated work clothing should not be allowed out of the workplace. |

### Precautionary statement(s) Response

| | |
|---|---|
| **P308+P313** | IF exposed or concerned: Get medical advice/ attention. |
| **P302+P352** | IF ON SKIN: Wash with plenty of water. |
| **P305+P351+P338** | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| **P333+P313** | If skin irritation or rash occurs: Get medical advice/attention. |
| **P337+P313** | If eye irritation persists: Get medical advice/attention. |
| **P362+P364** | Take off contaminated clothing and wash it before reuse. |

### Precautionary statement(s) Storage

| | |
|---|---|
| **P405** | Store locked up. |

### 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 | 30-60 | propylene glycol |
| 71125-38-7 | 1-10 | meloxicam |
| 100-51-6 | 1-10 | benzyl alcohol |
| 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** | If this product comes in contact with the eyes:<br>▸ Wash out immediately with fresh running water.<br>▸ 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.<br>▸ Seek medical attention without delay; if pain persists or recurs seek medical attention.<br>▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| **Skin Contact** | If skin contact occurs:<br>▸ Immediately remove all contaminated clothing, including footwear.<br>▸ Flush skin and hair with running water (and soap if available).<br>▸ Seek medical attention in event of irritation. |
| **Inhalation** | ▸ If fumes or combustion products are inhaled remove from contaminated area. |

**Continued...**

| | |
|---|---|
| | ▸ Lay patient down. Keep warm and rested.<br>▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.<br>▸ 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.<br>▸ Transport to hospital, or doctor, without delay. |
| **Ingestion** | ▸ For advice, contact a Poisons Information Centre or a doctor at once.<br>▸ Urgent hospital treatment is likely to be needed.<br>▸ If swallowed do **NOT** induce vomiting.<br>▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.<br>▸ Observe the patient carefully.<br>▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.<br>▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.<br>▸ Transport to hospital or doctor without delay. |

### Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

## SECTION 5 Firefighting measures

### Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.
Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:

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

### Special hazards arising from the substrate or mixture

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

### Advice for firefighters

| | |
|---|---|
| **Fire Fighting** | ▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ Wear full body protective clothing with breathing apparatus.<br>▸ Prevent, by any means available, spillage from entering drains or water course.<br>▸ Use water delivered as a fine spray to control fire and cool adjacent area.<br>▸ Avoid spraying water onto liquid pools.<br>▸ **DO NOT** approach containers suspected to be hot.<br>▸ Cool fire exposed containers with water spray from a protected location.<br>▸ If safe to do so, remove containers from path of fire. |
| **Fire/Explosion Hazard** | ▸ The material is not readily combustible under normal conditions.<br>▸ However, it will break down under fire conditions and the organic component may burn.<br>▸ Not considered to be a significant fire risk.<br>▸ Heat may cause expansion or decomposition with violent rupture of containers.<br>▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).<br>▸ May emit acrid smoke.<br><br>Decomposes on heating and produces toxic fumes of:<br>carbon dioxide ($CO_2$)<br>nitrogen oxides ($NO_x$)<br>other pyrolysis products typical of burning organic material.<br>May emit poisonous fumes.<br>May emit corrosive fumes. |
| **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** | ▸ Remove all ignition sources. |

**Continued...**

| | |
|---|---|
| | ▸ Clean up all spills immediately.<br>▸ Avoid breathing vapours and contact with skin and eyes.<br>▸ Control personal contact with the substance, by using protective equipment.<br>▸ Contain and absorb spill with sand, earth, inert material or vermiculite.<br>▸ Wipe up.<br>▸ Place in a suitable, labelled container for waste disposal. |
| **Major Spills** | Moderate hazard.<br>▸ Clear area of personnel and move upwind.<br>▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ Wear breathing apparatus plus protective gloves.<br>▸ Prevent, by any means available, spillage from entering drains or water course.<br>▸ No smoking, naked lights or ignition sources.<br>▸ Increase ventilation.<br>▸ Stop leak if safe to do so.<br>▸ Contain spill with sand, earth or vermiculite.<br>▸ Collect recoverable product into labelled containers for recycling.<br>▸ Absorb remaining product with sand, earth or vermiculite.<br>▸ Collect solid residues and seal in labelled drums for disposal.<br>▸ Wash area and prevent runoff into drains.<br>▸ 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** | ▸ Avoid all personal contact, including inhalation.<br>▸ Wear protective clothing when risk of exposure occurs.<br>▸ Use in a well-ventilated area.<br>▸ Prevent concentration in hollows and sumps.<br>▸ **DO NOT** enter confined spaces until atmosphere has been checked.<br>▸ **DO NOT** allow material to contact humans, exposed food or food utensils.<br>▸ Avoid contact with incompatible materials.<br>▸ When handling, **DO NOT** eat, drink or smoke.<br>▸ Keep containers securely sealed when not in use.<br>▸ Avoid physical damage to containers.<br>▸ Always wash hands with soap and water after handling.<br>▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use.<br>▸ Use good occupational work practice.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.<br>▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. |
| **Other information** | ▸ Store in original containers.<br>▸ Keep containers securely sealed.<br>▸ Store in a cool, dry, well-ventilated area.<br>▸ Store away from incompatible materials and foodstuff containers.<br>▸ Protect containers against physical damage and check regularly for leaks.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS. |

### Conditions for safe storage, including any incompatibilities

| | |
|---|---|
| **Suitable container** | ▸ Packaging as recommended by manufacturer.<br>▸ Check that containers are clearly labelled.<br>▸ Tamper-proof containers.<br>▸ Polyethylene or polypropylene containers.<br>▸ Metal drum with sealed plastic liner.<br>▸ Glass container is suitable for laboratory quantities<br>▸ Metal can or drum<br>▸ Packaging as recommended by manufacturer.<br>▸ Check all containers are clearly labelled and free from leaks. |
| **Storage incompatibility** | ▸ Avoid strong acids, bases.<br>▸ Avoid reaction with oxidising agents |

## SECTION 8 Exposure controls / personal protection

### Control parameters

**Occupational Exposure Limits (OEL)**

**INGREDIENT DATA**

**Continued...**

| Source | Ingredient | Material name | TWA | STEL | Peak | Notes |
|---|---|---|---|---|---|---|
| Australia Exposure Standards | propylene glycol | Propane-1,2-diol: particulates only | 10 mg/m3 | Not Available | Not Available | Not Available |
| Australia Exposure Standards | propylene glycol | Propane-1,2-diol total: (vapour & particulates) | 150 ppm / 474 mg/m3 | Not Available | Not Available | Not Available |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| propylene glycol | Not Available | Not Available |
| meloxicam | Not Available | Not Available |
| benzyl alcohol | Not Available | Not Available |

**MATERIAL DATA**

## Exposure controls

**Appropriate engineering controls**

Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation.
HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours.
Barrier protection or laminar flow cabinets should be considered for laboratory scale handling.
A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg.
When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology.
Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies.
Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required.
Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved. Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. 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, 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 (released at low velocity into zone of active generation) | 0.5-1 m/s (100-200 f/min.) |
| direct spray, 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.) |

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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 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.
The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated.
The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of:
10; high efficiency particulate (HEPA) filters or cartridges
10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator.
25-50; a full face-piece negative pressure respirator with HEPA filters
50-100; tight-fitting, full face-piece HEPA PAPR
100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode.

**Individual protection measures, such as personal protective equipment**

**Continued...**

| | |
|---|---|
| **Eye and face protection** | When handling very small quantities of the material eye protection may not be required.<br>For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs:<br>▸ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]<br>▸ Face shield. Full face shield may be required for supplementary but never for primary protection of eyes.<br>▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59]. |
| **Skin protection** | See Hand protection below |
| **Hands/feet protection** | ▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference.<br>▸ Double gloving should be considered.<br>▸ PVC gloves.<br>▸ Change gloves frequently and when contaminated, punctured or torn.<br>▸ Wash hands immediately after removing gloves.<br>▸ Protective shoe covers. [AS/NZS 2210]<br>▸ Head covering. |
| **Body protection** | See Other protection below |
| **Other protection** | ▸ For quantities up to 500 grams a laboratory coat may be suitable.<br>▸ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs.<br>▸ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers.<br>▸ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection.<br>▸ Eye wash unit.<br>▸ Ensure there is ready access to an emergency shower.<br>▸ For Emergencies: Vinyl suit |

## 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:
Butec OTM Pain Relief for Cattle and Sheep

| Material | CPI |
|---|---|
| BUTYL | C |
| BUTYL/NEOPRENE | C |
| HYPALON | C |
| NATURAL RUBBER | C |
| NATURAL+NEOPRENE | C |
| NEOPRENE | C |
| NEOPRENE/NATURAL | C |
| NITRILE | C |
| NITRILE+PVC | 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.

### Ansell Glove Selection

## 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

**Continued...**

| **Glove** — *In order of recommendation* |
|---|
| AlphaTec® Solvex® 37-675 |
| MICROFLEX® 93-260 |
| TouchNTuff® 92-500 |
| TouchNTuff® 92-605 |
| TouchNTuff® 92-600 |
| TouchNTuff® 93-250 |
| TouchNTuff® 93-700 |
| AlphaTec® 15-554 |
| AlphaTec® Solvex® 37-185 |
| AlphaTec® 38-612 |

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

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

| | |
|---|---|
| **Appearance** | Clear blue viscous liquid with no odour; mixes with water. |

| | | | |
|---|---|---|---|
| **Physical state** | Liquid | **Relative density (Water = 1)** | 1.035 |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | Not Applicable |
| **pH (as supplied)** | Not Available | **Decomposition temperature (°C)** | Not Available |
| **Melting point / freezing point (°C)** | ~0 | **Viscosity (cSt)** | Not Available |
| **Initial boiling point and boiling range (°C)** | ~100 | **Molecular weight (g/mol)** | Not Applicable |
| **Flash point (°C)** | Not Applicable | **Taste** | Not Available |
| **Evaporation rate** | Not Available | **Explosive properties** | Not Available |
| **Flammability** | Not Applicable | **Oxidising properties** | Not Available |
| **Upper Explosive Limit (%)** | Not Applicable | **Surface Tension (dyn/cm or mN/m)** | Not Available |
| **Lower Explosive Limit (%)** | Not Applicable | **Volatile Component (%vol)** | Not Available |
| **Vapour pressure (kPa)** | 2.37 @20C | **Gas group** | Not Available |
| **Solubility in water** | Miscible | **pH as a solution (1%)** | Not Available |
| **Vapour density (Air = 1)** | Not Available | **VOC g/L** | Not Available |
| **Heat of Combustion (kJ/g)** | Not Available | **Ignition Distance (cm)** | Not Available |
| **Flame Height (cm)** | Not Available | **Flame Duration (s)** | Not Available |
| **Enclosed Space Ignition Time Equivalent (s/m3)** | Not Available | **Enclosed Space Ignition Deflagration Density (g/m3)** | Not Available |

## SECTION 10 Stability and reactivity

| | |
|---|---|
| **Reactivity** | See section 7 |
| **Chemical stability** | ▸ Unstable in the presence of incompatible materials.<br>▸ Product is considered stable.<br>▸ 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 |

**Continued...**

## SECTION 11 Toxicological information

### Information on toxicological effects

| | |
|---|---|
| **a) Acute Toxicity** | Based on available data, the classification criteria are not met. |
| **b) Skin Irritation/Corrosion** | There is sufficient evidence to classify this material as skin corrosive or irritating. |
| **c) Serious Eye Damage/Irritation** | There is sufficient evidence to classify this material as eye damaging or irritating |
| **d) Respiratory or Skin sensitisation** | There is sufficient evidence to classify this material as sensitising to skin or the respiratory system |
| **e) Mutagenicity** | Based on available data, the classification criteria are not met. |
| **f) Carcinogenicity** | There is sufficient evidence to classify this material as carcinogenic |
| **g) Reproductivity** | Based on available data, the classification criteria are not met. |
| **h) STOT - Single Exposure** | Based on available data, the classification criteria are not met. |
| **i) STOT - Repeated Exposure** | Based on available data, the classification criteria are not met. |
| **j) Aspiration Hazard** | Based on available data, the classification criteria are not met. |

| | |
|---|---|
| **Inhaled** | Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.<br>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.<br>Inhalation hazard is increased at higher temperatures. |
| **Ingestion** | Accidental ingestion of the material may be damaging to the health of the individual. |
| **Skin Contact** | The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either<br>▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or<br>▸ produces significant, but moderate, 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.<br>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.<br>Open cuts, abraded or irritated skin should not be exposed to this material<br>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.<br>Limited evidence suggests that repeated exposure may cause skin cracking, flaking or drying following normal handling and use. |
| **Eye** | Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.<br>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. |
| **Chronic** | Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.<br>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.<br>Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers<br>Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.<br>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.<br><br>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. |

| | | |
|---|---|---|
| **Butec OTM Pain Relief for Cattle and Sheep** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |
| **propylene glycol** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: 11890 mg/kg[2] | Eye (Rodent - rabbit): 100mg - Mild |

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| | Inhalation (Rat) LC50: >44.9 mg/l4h[1] | Eye (Rodent - rabbit): 500mg/24H - Mild |
| | Oral (Rat) LD50: 20000 mg/kg[2] | Eye: no adverse effect observed (not irritating)[1] |
| | | Skin (Human - child): 30%/96H(continuous) - Moderate |
| | | Skin (Human - man): 10%/2D |
| | | Skin (Human - woman): 30%/96H - Mild |
| | | Skin (Human): 104mg/3D (intermittent) - Moderate |
| | | Skin (Human): 20% |
| | | Skin (Human): 500mg/7D - Mild |
| | | Skin: no adverse effect observed (not irritating)[1] |

| **meloxicam** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Oral (Rabbit) LD50; 320 mg/kg[2] | Not Available |

| **benzyl alcohol** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Dermal (rabbit) LD50: 2000 mg/kg[2] | Eye (Rodent - rat): 0.1mL |
| | Inhalation (Rat) LC50: >4.178 mg/L4h[2] | Eye: adverse effect observed (irritating)[1] |
| | Oral (Rat) LD50: 1230 mg/kg[2] | Skin (Human - man): 16mg/48H - Mild |
| | | Skin (Human): 1%/2D |
| | | Skin (Mammal - pig): 100% - Moderate |
| | | Skin (Rodent - rabbit): 100mg/24H - Moderate |
| | | Skin: no adverse effect observed (not irritating)[1] |

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| ***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 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 eostrogen cream may notice that brand name creams made with propylene glycol often create extreme, uncomfortable burning along the vulva and perianal area. Additionally, some electronic cigarette users who inhale propylene glycol vapor may experience dryness of the throat or shortness of breath . As an alternative, some suppliers will put Vegetable Glycerin in the "e-liquid" for those who are allergic (or have bad reactions) to propylene glycol.
Adverse responses to intravenous administration of drugs which use PG as an excipient have been seen in a number of people, particularly with large dosages thereof. Responses may include "hypotension, bradycardia... QRS and T abnormalities on the ECG, arrhythmia, cardiac arrest, serum hyperosmolality, lactic acidosis, and haemolysis". A high percentage (12% to 42%) of directly-injected propylene glycol is eliminated/secreted in urine unaltered depending on dosage, with the remainder appearing in its glucuronide-form. The speed of renal filtration decreases as dosage increases, which may be due to propylene glycol's mild anesthetic / CNS-depressant -properties as an alcohol. In one case, intravenous administration of propylene glycol-suspended nitroglycerin to an elderly man may have induced coma and acidosis.

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| | 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)<br>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. |
| **MELOXICAM** | Carcinogenicity: No carcinogenic effect of meloxicam was observed in rats given oral doses up to 0.8 mg/kg/day (approximately 0.4-fold the human dose at 15 mg/day for a 50 kg adult based on body surface area conversion) for 104 weeks or in mice given oral doses up to 8.0 mg/kg/day (approximately 2.2-fold the human dose, as noted above) for 99 weeks. Reproductive Toxicity: Meloxicam did not impair male and female fertility in rats at oral doses up to 9 and 5 mg/kg/day, respectively (4.9-fold and 2.5-fold the human dose, as noted above). However, an increased incidence of embryolethality at oral doses >/= 1 mg/kg/day (0.5-fold the human dose, as noted above) was observed in rats when dams were given meloxicam 2 weeks prior to mating and during early embryonic development. Teratogenicity: Pregnancy Category C: Meloxicam caused an increased incidence of septal defect of the heart, a rare event, at an oral dose of 60 mg/kg/day (64.5-fold the human dose at 15 mg/day for a 50 kg adult based on body surface area conversion) and embryolethality at oral doses >/= 5 mg/kg/day (5.4-fold the human dose, as noted above) when rabbits were treated throughout organogenesis. Meloxicam was not teratogenic in rats up to an oral dose of 4 mg/kg/day (approximately 2.2-fold the human dose, as noted above) throughout organogenesis. An increased incidence of stillbirths was observed when rats were given oral doses >/= 1 mg/kg/day throughout organogenesis. Meloxicam crosses the placental barrier. There are no adequate and well-controlled studies in pregnant women. Mutagenicity: Meloxicam was not mutagenic in an Ames assay, or clastogenic in a chromosome aberration assay with human lymphocytes and an in vivo micronucleus test in mouse bone marrow. * Apotex SDS<br>Accumulated studies have proved that non-steroidal anti-inflammatory drugs (NSAIDs) which block inflammation by their actions on arachidonic acid (AA) metabolism have a potential role in cancer chemotherapy and chemoprevention.<br>There is a general acceptance that NSAIDs induce colon cancer in humans. One suggested reason is that the balance between COX and lipoxygenase (LOX) activity determines tumorigenesis critically. Under low COX activity, arachidonic acid released from cell membranes in response to external stimuli is preferentially metabolized by LOX enzymes. The oxygenated lipids (metabolites) produced by LOXs initiate subsequent biological reactions, activate cellular signaling mechanisms through specific cell surface receptors, or are further metabolized into potent lipid mediators.<br>There is evidence that a 15-LOX metabolite 13S-HPODE (13S-hydroperoxyoctadecaenoic acid) generated from linoleic acid induces apoptosis in colon cancer.<br>Ingestion of aspirin or other NSAIDs may elicit respiratory, nasal, and gastrointestinal symptoms, as well as dermal changes in a subset of patients with asthma. The sensitivity to cyclooxygenase (COX) inhibitors has led to the hypothesis that NSAIDs may be causing upregulation of the 5-lipoxygenase pathway and its attendant products, the leukotrienes, in these patients. It has been shown increase in urinary leukotriene E 4 (LTE4) after aspirin ingestion or inhalation of lysine-aspirin in aspirin-sensitive patients with asthma. It has also been demonstrated that pharmacologic blockade at the level of the cysteinyl leukotriene receptor(s) can blunt the bronchospastic response to aspirin. Cysteinyl leukotrienes are potent bronchoconstrictors, induce mucus secretion, and increase vascular permeability. Importantly, inhibition of 5-lipoxygenase blocks not only the respiratory but also the gastrointestinal and dermal reactions to aspirin in aspirin-sensitive patients with asthma. Although these results establish the importance of 5-lipooxygenase products in mediating reactions to aspirin, the cellular source and mechanism of release of these mediators remain unclear.<br>Mast cells, which are a known source of leukotrienes, are activated in the nasal response to aspirin as demonstrated by the detection of nasal tryptase after aspirin challenge. Tryptase is an enzyme specific to mast cells and is an indicator of mast cell activation. Cysteinyl leukotrienes and histamine, which can be produced by mast cells, were detected as well. The occurrence of nasal symptoms, as well as activation of mast cells, in response to aspirin was blocked by zileuton, an inhibitor of 5-lipoxygenase. This confirms that 5-lipoxygenase products are critical to the development of aspirin-induced asthma (ASA-induced) reactions in the nose. It also suggests that 5-lipoxygenase products may have a role in the activation of mast cells during this reaction. |
| **BENZYL ALCOHOL** | The following information refers to contact allergens as a group and may not be specific to this product.<br>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.<br>Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.<br>Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.<br>Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.<br>Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.<br>Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.<br>Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the |

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allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

**Hands:** Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

**Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

**Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic.

**Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

**Pigmentary anomalies:** The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

**Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

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

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or as a prohapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.

**Prohaptens**

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.

In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of

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xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.
**QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation
CYP1A2 is a member of the cytochrome P450 super family, is one of the best characterized. It is responsible for the metabolism of commonly drugs belonging to classes such as antidepressants, antipsychotics, mood stabilizers, beta blockers and sedative/hypnotics CYP1A2 also metabolises a number of procarcinogens (such as those in cigarettes). Cigarette smoking may lead to three fold increase in 1A2 activity, which explains why smokers require higher doses of beta blockers than than non-smokers
Drugs that inhibit CYP1A2 will predictably increase the plasma concentrations of the medications or decrease in clearance of substrates. Drugs such as ciprofloxacin, fluvoxamine, verapamil cimetidine , caffeine and isoniazid are inhibitors of CYP1A2 enzyme. Vegetables such as grape fruit juice, cumic and turmeric are inhibitors of the CYP1A2 enzyme which may leads to increase plasma concentration of psychotropics
Inhibition of NF-kB in vivo can be detrimental. NF-kB controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF-kB activity.
The same functions that make NF-kB attractive for developing inhibitors for treating disease also play a role in homeostasis,and disruption of the NF-kB pathway during development or in adults leads to unfavorable and potentially unhealthy consequences. NF-kB plays a role in multiple homeostatic cellular processes including response to stimuli,cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such a p38 and JNK. In addition to mediating proinflammatory responses, NF-kB may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF-kB can have adverse consequences such as immune suppression and tissue damage.
Understanding the consequences of lack of NF-kB activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF-kB resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF-kB activation including IKK gamma (NEMO), a subunit of the IKK complex, and IkBalpha.The IKK gamma mutations result in a defective IKK complex and the IkBalpha mutation results in an IkBalpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF-kB activation and ectodermal dysplasia with immunodeficiency.In general patients with these genetic defects have multiple immunological defects including impaired innate immunity,impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF-kB pathway will help prepare for potential adverse effects of pharmacologic NF-kB inhibitors
The requirement for NF-kB in the development and maintenance of the immune system is well documented. NF-kB is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF-kB or the IKKbeta gene results in death during fetal development primarily due to massive liver apoptosis
Fetal liver stem cells from p65 or IKKbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF-kB for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF-kB activity. Lymphocyte depletion with chemical or genetic inhibition of NF-kB have implications for therapeutic potential use in humans. The double-sided nature of NF-kB inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations.
In addition to controlling lymphocyte development, NF-kB plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF-kB which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF-kB through phosphorylation of CARMA1 by PKC theta and PKC beta respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF-kB plays a role in T-cell response to costimulatory signals. Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs).TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF-kB leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF-kB during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF-kB is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF-kB activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation.Given the diverse roles NF-kB plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF-kB pathway are susceptible to parasitic and bacterial infection.
The role of NF-kB in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF-kB deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF-kB targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator.Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF-kB activation.
The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles.
The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity.
At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin.
The potential for eye irritation is minimal.
With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low.

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| | NOAELs for maternal and developmental toxicity are far in excess of current human exposure levels.<br>No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity in vitro bacterial assays, and in vitro mammalian cell assays. All in vivo micronucleus assays were negative.<br>It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients<br>The Research Institute for Fragrance Materials (RIFM) Expert Panel<br>A member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption. metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances.<br>All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals.<br>The substances in this group:<br>· contain a benzene ring substituted with a reactive primary oxygenated functional group or can be hydrolysed to such a functional group<br>· the major pathway of metabolic detoxification involves hydrolysis and oxidation to yield the corresponding benzoic acid derivate which is excreted either as the free acid or the glycine conjugate<br>· they show a consistent pattern of toxicity in both short- and long- term studies and<br>· they exhibit no evidence of genotoxicity in standardised batteries of in vitro and in vivo assays.<br>The benzyl derivatives are rapidly absorbed through the gut, metabolised primarily in the liver, and excreted in the urine as glycine conjugates of benzoic acid derivatives.<br>In general, aromatic esters are hydrolysed in vivo through the catalytic activity of carboxylesterases, the most important of which are the A-esterases. Hydrolysis of benzyl and benzoate esters to yield corresponding alcohols and carboxylic acids and hydrolysis of acetals to yield benzaldehyde and simple alcohols have been reported in several experiments.<br>The alcohols and aldehydes are rapidly oxidised to benzoic acid while benzoate esters are hydrolysed to benzoic acid.<br>Flavor and Extract Manufacturers Association (FEMA)<br>For benzyl alkyl alcohols:<br>Unlike benzylic alcohols, the beta-hydroxyl group of the members of this cluster is unlikely to undergo phase II metabolic activation. Instead, the beta-hydroxyl group is expected to contribute to detoxification via oxidation to hydrophilic acid. Despite structural similarity to carcinogenic ethyl benzene, only a marginal concern has been assigned to phenethyl alcohol due to limited mechanistic analogy.<br>For benzoates:<br>**Acute toxicity:** Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol.<br>The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds.<br>Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye.<br>**Sensitisation:** The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers.<br>**Repeat dose toxicity:** For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed.<br>For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur.<br>**Mutagenicity:** All chemicals showed no mutagenic activity in ***in vitro*** Ames tests. Various results were obtained with other ***in vitro*** genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity ***in vivo.*** While some mixed and/or equivocal ***in vitro*** chromosomal/chromatid responses have been observed, no genotoxicity was observed in the ***in vivo*** cytogenetic, micronucleus, or other assays. The weight of the evidence of the ***in vitro*** and ***in vivo*** genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.<br>In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts.<br>**Developmental toxicity:** In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed. |
| **PROPYLENE GLYCOL & BENZYL ALCOHOL** | 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. |

**Continued...**

| | | | |
|---|---|---|---|
| **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

| | | | | | |
|---|---|---|---|---|---|
| **Butec OTM Pain Relief for Cattle and Sheep** | **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** |
|---|---|---|---|---|---|
| | EC50 | 96h | Algae or other aquatic plants | 19000mg/l | 2 |
| | EC50 | 72h | Algae or other aquatic plants | 19300mg/l | 2 |
| | EC50 | 48h | Crustacea | >114.4mg/L | 4 |
| | NOEC(ECx) | 336h | Algae or other aquatic plants | <5300mg/l | 1 |
| | LC50 | 96h | Fish | 710mg/L | 4 |

| **meloxicam** | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
|---|---|---|---|---|---|
| | NOEC(ECx) | 144h | Fish | 0.1mg/L | 4 |

| **benzyl alcohol** | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
|---|---|---|---|---|---|
| | EC50 | 96h | Algae or other aquatic plants | 76.828mg/l | 2 |
| | EC50 | 72h | Algae or other aquatic plants | 500mg/l | 2 |
| | NOEC(ECx) | 336h | Fish | 5.1mg/l | 2 |
| | EC50 | 48h | Crustacea | 230mg/l | 2 |
| | LC50 | 96h | Fish | 10mg/l | 2 |

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

**DO NOT** discharge into sewer or waterways.

### Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| propylene glycol | LOW | LOW |
| benzyl alcohol | LOW | LOW |

### Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| propylene glycol | LOW (BCF = 1) |
| benzyl alcohol | LOW (LogKOW = 1.1) |

### Mobility in soil

| Ingredient | Mobility |
|---|---|
| propylene glycol | HIGH (Log KOC = 1) |
| benzyl alcohol | LOW (Log KOC = 15.66) |

## SECTION 13 Disposal considerations

### Waste treatment methods

**Continued...**

| | |
|---|---|
| **Product / Packaging disposal** | ▸ Recycle wherever possible or consult manufacturer for recycling options.<br>▸ Consult State Land Waste Authority for disposal.<br>▸ Bury or incinerate residue at an approved site.<br>▸ Recycle containers if possible, or dispose of in an authorised landfill.<br>▸ Containers may still present a chemical hazard/ danger when empty.<br>▸ Return to supplier for reuse/ recycling if possible.<br>Otherwise:<br>▸ 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.<br>▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product.<br>▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains.<br>▸ It may be necessary to collect all wash water for treatment before disposal.<br>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.<br>▸ Where in doubt contact the responsible authority. |

## SECTION 14 Transport information

### Labels Required

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

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

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

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

### 14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

| Product name | Group |
|---|---|
| propylene glycol | Not Available |
| meloxicam | Not Available |
| benzyl alcohol | Not Available |

### 14.7.3. Transport in bulk in accordance with the IGC Code

| Product name | Ship Type |
|---|---|
| propylene glycol | Not Available |
| meloxicam | Not Available |
| benzyl alcohol | 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)

**meloxicam 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 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
FEI Equine Prohibited Substances List - Controlled Medication
FEI Equine Prohibited Substances List (EPSL)

**benzyl alcohol is found on the following regulatory lists**

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

### Additional Regulatory Information

**Continued...**

Not Applicable

### National Inventory Status

| National Inventory | Status |
|---|---|
| Australia - AIIC / Australia Non-Industrial Use | No (meloxicam) |
| Canada - DSL | No (meloxicam) |
| Canada - NDSL | No (propylene glycol; meloxicam; benzyl alcohol) |
| China - IECSC | No (meloxicam) |
| Europe - EINEC / ELINCS / NLP | No (meloxicam) |
| Japan - ENCS | No (meloxicam) |
| Korea - KECI | No (meloxicam) |
| New Zealand - NZIoC | Yes |
| Philippines - PICCS | No (meloxicam) |
| USA - TSCA | TSCA Inventory 'Active' substance(s) (propylene glycol; benzyl alcohol); No (meloxicam) |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | Yes |
| Vietnam - NCI | Yes |
| Russia - FBEPH | No (meloxicam) |
| ***Legend:*** | ***Yes = All CAS declared ingredients are on the inventory***<br>***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** | 03/03/2025 |
| **Initial Date** | 23/01/2023 |

### Other information

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

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

### Definitions and abbreviations

- PC—TWA: Permissible Concentration-Time Weighted Average
- PC—STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China

**Continued...**

- 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