# KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB METALLIC

## Jasco Pty Limited

Chemwatch Hazard Alert Code: **3**

Chemwatch: **9659-82**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **28/10/2026**
Revision Date: **28/10/2026**
Print Date: **05/11/2026**
L.GHS.AUS.EN.E

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

### Product Identifier

| | |
|---|---|
| **Product name** | KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB METALLIC |
| **Chemical Name** | Not Applicable |
| **Synonyms** | METALLIC SILVER; METALLIC GOLD |
| **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** | Pen.<br>Use according to manufacturer's directions. |

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

| | |
|---|---|
| **Registered company name** | **Jasco Pty Limited** |
| **Address** | 1-5 Commercial Road Kingsgrove NSW 2208 Australia |
| **Telephone** | +61 2 9807 1555 |
| **Fax** | Not Available |
| **Website** | www.jasco.com.au |
| **Email** | sales@jasco.com.au |

### Emergency telephone number

| | | |
|---|---|---|
| **Association / Organisation** | **Australian Poisons Centre** | **CHEMWATCH EMERGENCY RESPONSE (24/7)** |
| **Emergency telephone number(s)** | **13 11 26 (24/7)** | **+61 1800 951 288 (ID#: 9659-82)** |
| **Other emergency telephone number(s)** | **Not Available** | **+61 3 9573 3188** |

## SECTION 2 Hazards identification

### Classification of the substance or mixture

| | |
|---|---|
| **Poisons Schedule** | Not Applicable |
| **Classification [1]** | Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Carcinogenicity Category 1B |
| ***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** | **Danger** |

### Hazard statement(s)

| | |
|---|---|
| **H315** | Causes skin irritation. |
| **H319** | Causes serious eye irritation. |
| **H350** | May cause cancer. |

### Precautionary statement(s) Prevention

| | |
|---|---|
| **P280** | Wear protective gloves, protective clothing, eye protection and face protection. |
| **P202** | Do not handle until all safety precautions have been read and understood. |
| **P264** | Wash all exposed external body areas thoroughly after handling. |

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**Precautionary statement(s) Response**

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

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

**Precautionary statement(s) Disposal**

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

No further product hazard information.

## SECTION 3 Composition / information on ingredients

**Substances**

See section below for composition of Mixtures

**Mixtures**

| CAS No | %[weight] | Name |
|---|---|---|
| 7732-18-5 | <=65 | water |
| 56-81-5 | 19.96 | glycerol |
| 57-55-6 | 10 | propylene glycol |
| 7429-90-5 | <=5 | aluminium |
| 2786-76-7 | 1.5 | C.I. Pigment Red 170 |
| 5567-15-7 | 1.5 | C.I. Pigment Yellow 83 |
| 52-51-7 | 0.02 | 2-bromo-2-nitropropan-1,3-diol |
| 55406-53-6 | 0.02 | 3-iodo-2-propynyl butyl carbamate |
| ***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**

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|---|---|
| **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 or hair contact occurs:<br>▸ Immediately flush body and clothes with large amounts of water, using safety shower if available.<br>▸ Quickly remove all contaminated clothing, including footwear.<br>▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.<br>▸ Transport to hospital, or doctor.<br>For thermal burns:<br>▸ Decontaminate area around burn.<br>▸ Consider the use of cold packs and topical antibiotics.<br>For first-degree burns (affecting top layer of skin)<br>▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides.<br>▸ Use compresses if running water is not available.<br>▸ Cover with sterile non-adhesive bandage or clean cloth.<br>▸ Do NOT apply butter or ointments; this may cause infection.<br>▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur.<br>For second-degree burns (affecting top two layers of skin)<br>▸ Cool the burn by immerse in cold running water for 10-15 minutes.<br>▸ Use compresses if running water is not available.<br>▸ Do NOT apply ice as this may lower body temperature and cause further damage.<br>▸ Do NOT break blisters or apply butter or ointments; this may cause infection.<br>▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape.<br>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):<br>▸ Lay the person flat.<br>▸ Elevate feet about 12 inches.<br>▸ Elevate burn area above heart level, if possible.<br>▸ Cover the person with coat or blanket.<br>▸ Seek medical assistance.<br>For third-degree burns<br>Seek immediate medical or emergency assistance.<br>In the mean time:<br>▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. |

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|---|---|
| | ▸ Separate burned toes and fingers with dry, sterile dressings.<br>▸ Do not soak burn in water or apply ointments or butter; this may cause infection.<br>▸ To prevent shock see above.<br>▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway.<br>▸ Have a person with a facial burn sit up.<br>▸ Check pulse and breathing to monitor for shock until emergency help arrives. |
| **Inhalation** | ▸ If fumes or combustion products are inhaled remove from contaminated area.<br>▸ 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** | ▸ Immediately give a glass of water.<br>▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. |

**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 substance or mixture**

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

**Advice for firefighters**

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|---|---|
| **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** | ▸ Combustible.<br>▸ Slight fire hazard when exposed to heat or flame.<br>▸ Heating may cause expansion or decomposition leading to violent rupture of containers.<br>▸ On combustion, may emit toxic fumes of carbon monoxide (CO).<br>▸ May emit acrid smoke.<br>▸ Mists containing combustible materials may be explosive.<br>Combustion products include:<br>▸ carbon dioxide ($CO_2$)<br>acrolein<br>hydrogen iodide<br>nitrogen oxides (NOx)<br>metal oxides<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**

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|---|---|
| **Minor Spills** | ▸ Remove all ignition sources.<br>▸ 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** | ▸ Clear area of personnel and move upwind.<br>▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ Wear full body protective clothing with breathing apparatus.<br>▸ Prevent, by all means available, spillage from entering drains or water courses.<br>▸ Consider evacuation (or protect in place).<br>▸ No smoking, naked lights or ignition sources.<br>▸ Increase ventilation. |

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|---|---|
| | ‣ Stop leak if safe to do so.<br>‣ Water spray or fog may be used to disperse / absorb vapour.<br>‣ Contain or absorb spill with sand, earth or vermiculite.<br>‣ Collect recoverable product into labelled containers for recycling.<br>‣ Collect solid residues and seal in labelled drums for disposal.<br>‣ Wash area and prevent runoff into drains.<br>‣ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.<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

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|---|---|
| **Safe handling** | ‣ Avoid skin 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 come in direct contact with human skin or eyes.<br>‣ **DO NOT** allow material to come in contact with exposed food or food contact surfaces.<br>‣ Suitable PPE must be worn at all times.<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.<br>‣ **DO NOT** allow clothing wet with material to stay in contact with skin |
| **Other information** | ‣ Store in original containers.<br>‣ Keep containers securely sealed.<br>‣ No smoking, naked lights or ignition sources.<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** | ‣ Metal can or drum<br>‣ Packaging as recommended by manufacturer.<br>‣ Check all containers are clearly labelled and free from leaks. |
| **Storage incompatibility** | Glycerol:<br>‣ reacts violently with strong oxidisers, acetic anhydride, alkali metal hydrides, calcium hypochlorite, calcium oxychloride, chlorine, chromic anhydride, chromium oxides, ethylene oxide, hydrogen peroxide, phosphorous triiodide, potassium chlorate, potassium permanganate, potassium peroxide, silver perchlorate, sodium hydride, sodium peroxide, sodium triiodide, sodium tetrahydroborate,is incompatible with strong acids, caustics, aliphatic amines, isocyanates, uranium fluoride<br>‣ is able to polymerise above 145 C<br>‣ Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water.<br>Formaldehyde:<br>‣ is a strong reducing agent<br>‣ may polymerise in air unless properly inhibited (usually with methanol up to 15%) and stored at controlled temperatures<br>‣ will polymerize with active organic material such as phenol<br>‣ reacts violently with strong oxidisers, hydrogen peroxide, potassium permanganate, acrylonitrile, caustics (sodium hydroxide, yielding formic acid and flammable hydrogen), magnesium carbonate, nitromethane, nitrogen oxides (especially a elevated temperatures), peroxyformic acid<br>‣ is incompatible with strong acids (hydrochloric acid forms carcinogenic bis(chloromethyl)ether*), amines, ammonia, aniline, bisulfides, gelatin, iodine, magnesite, phenol, some monomers, tannins, salts of copper, iron, silver.<br>‣ acid catalysis can produce impurities: methylal, methyl formate<br>Aqueous solutions of formaldehyde:<br>‣ slowly oxidise in air to produce formic acid<br>‣ attack carbon steel<br>Concentrated solutions containing formaldehyde are:<br>‣ unstable, both oxidising slowly to form formic acid and polymerising; in dilute aqueous solutions formaldehyde appears as monomeric hydrate (methylene glycol) - the more concentrated the solution the more polyoxymethylene glycol occurs as oligomers and polymers (methanol and amine-containing compounds inhibit polymer formation)<br>‣ readily subject to polymerisation, at room temperature, in the presence of air and moisture, to form paraformaldehyde (8-100 units of formaldehyde), a solid mixture of linear polyoxymethylene glycols containing 90-99% formaldehyde; a cyclic trimer, trioxane ($CH_2O_3$), may also form<br>Flammable and/or toxic gases are generated by the combination of aldehydes with azo, diazo compounds, dithiocarbamates, nitrides, and strong reducing agents<br>*The empirical equation may be used to determine the concentration of bis(chloromethyl)ether (BCME) formed by reaction with HCl:<br>log(BCME)ppb = -2.25 + 0.67• log(HCHO) ppm + 0.77• log(HCl)ppm<br>Assume values for formaldehyde, in air, of 1 ppm and for HCl of 5 ppm, resulting BCME concentration, in air, would be 0.02 ppb.<br>None known |

## SECTION 8 Exposure controls / personal protection

### Control parameters

**Occupational Exposure Limits (OEL)**

**Continued...**

**INGREDIENT DATA**

| Source | Ingredient | Material name | TWA | STEL | Peak | Notes |
|---|---|---|---|---|---|---|
| Australia Exposure Standards | glycerol | Glycerin mist | 10 mg/m3 | Not Available | Not Available | (a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica. |
| Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits | glycerol | Glycerin mist | 10 mg/m3 | Not Available | Not Available | Containing no asbestos and < 1% crystalline silica. |
| 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 |
| Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits | propylene glycol | Propane-1,2-diol total (vapour and particulates) | 50 mg/m3 | Not Available | Not Available | Not Available |
| Australia Exposure Standards | aluminium | Aluminium (metal dust) | 10 mg/m3 | Not Available | Not Available | Not Available |
| Australia Exposure Standards | aluminium | Aluminium (welding fumes) (as Al) | 5 mg/m3 | Not Available | Not Available | Not Available |
| Australia Exposure Standards | aluminium | Aluminium, pyro powders (as Al) | 5 mg/m3 | Not Available | Not Available | Not Available |
| Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits | aluminium | Aluminium, soluble salts (as Al) | 2 mg/m3 | Not Available | Not Available | Not Available |
| Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits | aluminium | Aluminium, pyro powders (as Al) | 5 mg/m3 | Not Available | Not Available | Not Available |
| Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits | aluminium | Aluminium, alkyls (NOC) (as Al) | 2 mg/m3 | Not Available | Not Available | Not Available |
| Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits | aluminium | Aluminium (metal dust) | 10 mg/m3 | Not Available | Not Available | Not Available |
| Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits | aluminium | Aluminium (welding fumes) (as Al) | 1 mg/m3 | Not Available | Not Available | Not Available |

**MATERIAL DATA**

**WARNING:** This substance has been classified by the ACGIH as A2 Suspected Human Carcinogen.

**WARNING:** This substance is classified by the NOHSC as Category 2 Probable Human Carcinogen
For aluminium oxide and pyrophoric grades of aluminium:
Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA.
The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For aluminium oxide:
The experimental and clinical data indicate that aluminium oxide acts as an "inert" material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.
[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition
for 3,3'-dichlorobenzidine (DCB):
Various tumours developed after oral or subcutaneous administration of DCB to mice, rats, hamsters and dogs. Tumours have not yet been identified in persons exposed to the substance alone. The substance can be absorbed through the skin in dangerous quantities. Increases in temperature and relative humidity promote dermal absorption.

**Exposure controls**

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|---|---|
| **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.<br>The basic types of engineering controls are:<br>Process controls which involve changing the way a job activity or process is done to reduce the risk.<br>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.<br>Employers may need to use multiple types of controls to prevent employee overexposure.<br><br>▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.<br>▸ Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. |

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|---|---|
| | ‣ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.<br>‣ Open-vessel systems are prohibited.<br>‣ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.<br>‣ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.<br>‣ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.<br>‣ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).<br>‣ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.<br>‣ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed. |
| **Individual protection measures, such as personal protective equipment** |  |
| **Eye and face protection** | ‣ Safety glasses with side shields.<br>‣ Chemical goggles.[AS/NZS 1337.1, EN166 or national equivalent]<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** | ‣ Wear chemical protective gloves, e.g. PVC.<br>‣ Wear safety footwear or safety gumboots, e.g. Rubber<br>**NOTE:**<br>‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.<br>‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.<br>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.<br>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.<br>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.<br>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:<br>· frequency and duration of contact,<br>· chemical resistance of glove material,<br>· glove thickness and<br>· dexterity<br>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).<br>· 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.<br>· 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.<br>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.<br>· Contaminated gloves should be replaced.<br>As defined in ASTM F-739-96 in any application, gloves are rated as:<br>· Excellent when breakthrough time > 480 min<br>· Good when breakthrough time > 20 min<br>· Fair when breakthrough time < 20 min<br>· Poor when glove material degrades<br>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.<br>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.<br>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.<br>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:<br>· 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.<br>· 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<br>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. |
| **Body protection** | See Other protection below |
| **Other protection** | ‣ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent]<br>‣ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent]<br>‣ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely.<br>‣ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with |

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suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood.
- Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- 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:
KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB METALLIC

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

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
**NOTE**: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

**Ansell Glove Selection**

| **Glove** — *In order of recommendation* |
|---|
| AlphaTec® Solvex® 37-675 |
| MICROFLEX® 93-260 |
| AlphaTec® Solvex® 37-185 |
| AlphaTec® 38-612 |
| AlphaTec® 58-008 |
| AlphaTec® 58-530B |
| AlphaTec® 58-530W |
| AlphaTec® 58-735 |
| AlphaTec® 79-700 |
| DermaShield™ 73-711 |

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

### Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

| Required Minimum Protection Factor | Half-Face Respirator | Full-Face Respirator | Powered Air Respirator |
|---|---|---|---|
| up to 10 x ES | A-AUS P2 | - | A-PAPR-AUS / Class 1 P2 |
| up to 50 x ES | - | A-AUS / Class 1 P2 | - |
| up to 100 x ES | - | A-2 P2 | A-PAPR-2 P2 ^ |

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- 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

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| **Appearance** | Metallic liquid. |

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| **Physical state** | Liquid | **Relative density (Water = 1)** | 1.261 |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | 370 |
| **pH (as supplied)** | Not Available | **Decomposition temperature (°C)** | Not Available |
| **Melting point / freezing point (°C)** | 18.17 | **Viscosity (mm2/s)** | 1412 |
| **Initial boiling point and boiling range (°C)** | 290 | **Molecular weight (g/mol)** | Not Applicable |
| **Flash point (°C)** | 199 | **Taste** | Not Available |
| **Evaporation rate** | Not Available | **Explosive properties** | Not Available |
| **Flammability** | Not Applicable | **Oxidising properties** | Not Available |

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| | | | |
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| **Upper Explosive Limit (%)** | 19 | **Surface Tension (dyn/cm or mN/m)** | Not Available |
| **Lower Explosive Limit (%)** | 2.7 | **Volatile Component (%vol)** | Not Available |
| **Vapour pressure (kPa)** | 0.003 | **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

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| **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 |

## SECTION 11 Toxicological information

### Information on toxicological effects

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| **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** | Based on available data, the classification criteria are not met. |
| **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. |

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| **Inhaled** | Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation.<br>The material is not classified as causing respiratory irritation under Regulation (EC) No 1272/2008 (CLP), as amended. Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be implemented in an occupational setting.<br>Not normally a hazard due to non-volatile nature of product |
| **Ingestion** | Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.<br>Overexposure to non-ring alcohols causes nervous system symptoms. These include headache, muscle weakness and inco-ordination, giddiness, confusion, delirium and coma.<br>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.<br>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.<br>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.<br>In humans propylene glycol is partly excreted unchanged in the urine and partly metabolised as lactic and pyruvic acid. Lactic acidosis may result.<br>The material is not expected to produce harmful effects following ingestion based on the available information. However, the material may still cause adverse health effects following ingestion, particularly in individuals with pre-existing organ damage (e.g. liver or kidney impairment). Gastrointestinal discomfort may occur and may result in nausea and vomiting. In an occupational setting, ingestion of incidental quantities should be avoided, and good hygiene practices should be followed to prevent ingestion. |
| **Skin Contact** | 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.<br><br>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.<br>The material may accentuate any pre-existing dermatitis condition |

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| | Skin contact is not expected to result in harmful health effects and the material is not classified as hazardous by the dermal route under Regulation (EC) No 1272/2008 (CLP), as amended. However, the material may cause harm following entry through wounds, lesions or abrasions.<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. |
| **Eye** | This material causes serious eye irritation. |
| **Chronic** | On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of:<br>- appropriate long-term animal studies<br>- other relevant information<br>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.<br>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.<br>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.<br>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.<br>Many azo dyes have been found to be carcinogenic in laboratory animals, affecting the liver, urinary bladder and intestines. Specific toxicity effects in humans have not been established but some dyes are known to be mutagenic.<br>The simplest azo dyes, which raise concern, have an exocyclic amino-group that is the key to any carcinogenicity for it is this group which undergoes biochemical N-oxidation and further reaction to reactive electrophiles. The DNA adducts formed by covalent binding through activated nitrogen have been identified. However not all azo compounds possess this activity and delicate alterations to structure vary the potential of carcinogenicity / acid, reduces or eliminates the effect. Complex azo dyes consisting of more than one azo (N=N) linkage may be metabolised to produce complexed carcinogenic aromatic amines such as benzidine<br>Benzidine and its metabolic derivatives have been detected in the urine of workers exposed to Direct azo dyes. An epidemiological study of silk dyers and painters with multiple exposures to benzidine based and other dyes indicate a strong association with bladder cancer.<br>Most organic azo dyes are potential skin sensitisers, the most important of which are para-phenylenediamine and its analogs. Water soluble azo dyes are more likely to cause clinical sensitisation than insoluble dyes. In addition to allergic eczematous contact dermatitis, color developing solutions have caused lichen planus like eruptions<br>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.<br>Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed<br>at periods up to 12 months following the last exposure.<br>When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.<br>The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.<br>Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.<br>There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.<br>When administered in the diet, 3,3'-dichlorobenzidine induced hepatomas in male mice, increased the incidences of granulocytic leukemia and Zymbal gland carcinomas in male rats and mammary adenocarcinomas in rats of both sexes, induced transitional cell carcinomas of the urinary bladder in hamsters and female dogs and hepatocellular carcinomas in female dogs.<br>Transplacental exposure increased the incidences of lymphoid leukemia in mice. In three retrospective epidemiological studies, no urinary bladder tumors were reported in men occupationally exposed to 3,3'-dichlorobenzidine<br>Propylene glycol is though, by some, to be a sensitising principal following the regular use of topical creams by eczema patients. A study of 866 persons using a formulation containing propylene glycol in a patch test indicated that propylene glycol caused primary irritation in 16% of exposed individuals probably caused by dehydration. Undiluted propylene glycol was tested on 1556 persons in a 24 hour patch test. 12.5% showed reactions which were largely toxic (70%) or allergic in nature (30%). Reaction responses reached their maximum on the second day or later. Reactions were seasonal in nature ranging from 17.8% in winter to 9.2% in other seasons. In a patch-test using 25 standard allergens conducted on 500 individuals, propylene glycol ranked fourth in sensitising response. 84 subjects were patch tested using 100% propylene glycol. as well as 2% and 5% in water. With undiluted material, 15% demonstrated a reaction, with 40% of the reactions being allergic in nature and 60% being irritant. In dilute solutions 5 of 248 subjects exhibited a reaction.<br>Undiluted propylene glycol tested on the skin of man produced no irritation under open conditions but when applied under occlusive conditions, for 2 weeks, it produced severe erythema, oedema and vesicles, probably due to sweat retention and weak primary irritation. Predictive contact skin sensitisation tests indicate that propylene glycol is an intermediate grade sensitiser with an index of 1% of tested subjects.<br>Groups of cats fed 5 gm/kg/day of propylene glycol for 14 weeks showed a significant dose-related increase in red blood cell Heinz body formation without any marked signs of haemolytic anaemia. The no-effect-level for cats without formation of Heinz bodies is 100-500 ml/kg. There is no evidence of anaemia or degenerative change. Groups of rats dosed orally with 0.5 or 10 mg/kg/day for 12 weeks had lowered food intake but no adverse effects on body weights. Erythrocytes were more fragile. Heinz bodies were not apparent. |

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| **KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB METALLIC** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |
| **water** | **TOXICITY** | **IRRITATION** |
| | Oral (Rat) LD50: >90000 mg/kg[2] | Not Available |

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|---|---|---|
| **glycerol** | **TOXICITY** | **IRRITATION** |
| | Dermal (Guinea Pig) LD50: 58500 mg/kg[1] | Eye (Rodent - rabbit): 500mg/24H - Mild |
| | Inhalation (Rat) LC50: >5.85 mg/L4h[1] | Eye: no adverse effect observed (not irritating)[1] |
| | Oral (Mouse) LD50; 4090 mg/kg[2] | Skin (Rodent - rabbit): 500mg/24H - Mild |
| | | Skin: no adverse effect observed (not irritating)[1] |
| **propylene glycol** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: 11890 mg/kg[2] | Eye (Rodent - rabbit): 100mg - Mild |
| | 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 - Moderate |
| | | Skin (Human - man): 10%/2D |
| | | Skin (Human - woman): 30%/96H - Mild |
| | | Skin (Human): 104mg/3D - Moderate |
| | | Skin (Human): 20% |
| | | Skin (Human): 500mg/7D - Mild |
| | | Skin: no adverse effect observed (not irritating)[1] |
| **aluminium** | **TOXICITY** | **IRRITATION** |
| | Inhalation (Rat) LC50: >2.3 mg/l4h[1] | Eye: no adverse effect observed (not irritating)[1] |
| | Oral (Rat) LD50: >2000 mg/kg[1] | Skin: no adverse effect observed (not irritating)[1] |
| **C.I. Pigment Red 170** | **TOXICITY** | **IRRITATION** |
| | dermal (rat) LD50: >2000 mg/kg[1] | Eye: no adverse effect observed (not irritating)[1] |
| | Inhalation (Rat) LC50: >1.58 mg/L4h[1] | Skin: no adverse effect observed (not irritating)[1] |
| | Oral (Rat) LD50: >10000 mg/kg[2] | |
| **C.I. Pigment Yellow 83** | **TOXICITY** | **IRRITATION** |
| | dermal (rat) LD50: >3000 mg/kg[1] | Eye: no adverse effect observed (not irritating)[1] |
| | Inhalation (Rat) LC50: >0.23 mg/L4h[1] | Skin (Rodent - rabbit): 500mg - Mild |
| | Oral (Mouse) LD50; >5000 mg/kg[2] | Skin: no adverse effect observed (not irritating)[1] |
| **2-bromo-2-nitropropan-1,3-diol** | **TOXICITY** | **IRRITATION** |
| | dermal (rat) LD50: ~1600 mg/kg[1] | Eye (Rodent - rabbit): 5mg |
| | Inhalation (Rat) LC50: >0.12<1.14 mg/l4h[1] | Eye: adverse effect observed (irreversible damage)[1] |
| | Oral (Rat) LD50: 180 mg/kg[2] | Skin (Human): 10mg - Moderate |
| | | Skin (Rodent - rabbit): 500mg/24H - Mild |
| | | Skin (Rodent - rabbit): 80mg - Moderate |
| | | Skin (Rodent - rat): 0.2% |
| | | Skin: adverse effect observed (irritating)[1] |
| **3-iodo-2-propynyl butyl carbamate** | **TOXICITY** | **IRRITATION** |
| | dermal (rat) LD50: >2000 mg/kg[2] | Eye: adverse effect observed (irreversible damage)[1] |
| | Inhalation (Rat) LC50: 0.63 mg/l4h[1] | Skin (Human): 0.3%/48H |
| | Oral (Rat) LD50: 1056 mg/kg[2] | 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*** |

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| **GLYCEROL** | For glycerol:<br>**Acute toxicity:** Glycerol is of a low order of acute oral and dermal toxicity with LD50 values in excess of 4000 mg/kg bw. At very high dose levels, the signs of toxicity include tremor and hyperaemia of the gastro-intestinal -tract. Skin and eye irritation studies indicate that glycerol has low potential to irritate the skin and the eye. The available human and animal data, together with the very widespread potential for exposure and the absence of case reports of sensitisation, indicate that glycerol is not a skin sensitiser.<br>**Repeat dose toxicity:** Repeated oral exposure to glycerol does not induce adverse effects other than local irritation of the gastro-intestinal tract. The overall NOEL after prolonged treatment with glycerol is 10,000 mg/kg bw/day (20% in diet). At this dose level no systemic or local effects were observed. For inhalation exposure to aerosols, the NOAEC for local irritant effects to the upper respiratory tract is 165 mg/m3 and 662 mg/m3 for systemic effects.<br>**Genotoxicity:** Glycerol is free from structural alerts, which raise concern for mutagenicity. Glycerol does not induce gene mutations in bacterial strains, chromosomal effects in mammalian cells or primary DNA damage ***in vitro***. Results of a limited gene mutation test in mammalian cells were of uncertain biological relevance. ***In vivo***, glycerol produced no statistically significant effect in a chromosome aberrations and dominant lethal study. However, the limited details provided and the absence of a positive control, prevent any reliable conclusions to be drawn from the ***in vivo*** data. Overall, glycerol is not considered to possess genotoxic potential. |

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| | **Carcinogenicity:** The experimental data from a limited 2 year dietary study in the rat does not provide any basis for concerns in relation to carcinogenicity. Data from non-guideline studies designed to investigate tumour promotion activity in male mice suggest that oral administration of glycerol up to 20 weeks had a weak promotion effect on the incidence of tumour formation.<br>**Reproductive and developmental toxicity:** No effects on fertility and reproductive performance were observed in a two generation study with glycerol administered by gavage (NOAEL 2000 mg/kg bw/day). No maternal toxicity or teratogenic effects were seen in the rat, mouse or rabbit at the highest dose levels tested in a guideline comparable teratogenicity study (NOEL 1180 mg/kg bw/day). |
| **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.<br>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.<br>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).<br>Propylene glycol shows no evidence of being a carcinogen or of being genotoxic.<br>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.<br>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<br>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.<br>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.<br>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.<br>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. |
| **C.I. PIGMENT YELLOW 83** | For diarylide (disazo) pigments (3,3'-dichlorobenzidine-containing):<br>The substances in this category do not present a hazard for human health due to their low hazard profile. Adequate screening-level data are available to characterise the human health hazard for the purposes of the OECD Cooperative Chemicals Assessment Programme.<br>Diarylide pigments are synthesized by bis-diazotizing diamino-diphenyl derivatives, mainly 3,3'-dichlorobenzidine (DCB), and coupling with acetoacetarylides or arylsubstituted pyrazolones<br>Studies indicate that essentially there is no potential for uptake via the oral and dermal routes. However, following repeated oral exposure at high dose levels, there is some evidence that a very limited uptake of the compound (or its impurities) could occur, based on observations of staining of the mucosal surfaces of internal organs (although the possibility of contamination during necropsy cannot be excluded). In an oral reproductive developmental screening study, staining of the pups could indicate a potential for limited placental transfer, again at a high dose level. Given that the Pigment Yellows are essentially not absorbed into the body,metabolism is not relevant. However, the presence of very low levels of 3,3 -dichlorobenzidine has been demonstrated in two studies using very sensitive techniques following oral administration of some yellow pigment compounds. It seems likely that this is due to the presence of a mono-azo impurity in some of the yellow pigment parent compounds, which is absorbed and subsequently metabolised. No DCB was found in the urine of experimental animals after exposure orally or via the lungs in long term studies. Following ingestion, the vast majority of the pigments are excreted unchanged in the faeces.<br>Many diarylide pigments are derived from DCB. Therefore, the diarylide pigments on DCB basis have been tested toxicologically very extensively. Diarylide pigments with their LD50 values above 2 000 mg/kg show no acute toxicity according to the EU classification criteria. They are not irritating to the skin or mucous membranes.<br>For acute dermal toxicity a single LD50 of >3,000 mg/kg bw is available for Pigment Yellow 13. No deaths or clinical signs of toxicity were observed following oral or dermal exposure. The inhalation LC50 available is >4,448 mg/m3 for Pigment Yellow 13. Tachypnoea, dyspnoea, exophthalmos, ruffled fur and curved or ventral body position were observed, although all animals recovered and no gross abnormalities were observed at necropsy.<br>Based on the available data the pigments have a minimal to slight potential for eye irritation. There is no indication that they are sensitisers No adverse effects were seen after 4-7 weeks oral administration of Pigment Yellow 12 at 1000 mg/kg/day (NOAEL), the highest dose tested in a well conducted and reported test of repeated dose toxicity study. Furthermore, in the cases of Pigment Yellow 12 and 83, no toxicologically significant effects were observed in a range of chronic toxicity studies of lesser quality (in terms of reporting) in rats and mice at doses up to 6500 mg/kg/day. Based on the kinetics of the three pigments and the chemical similarities, it can be concluded that these findings can be extrapolated to most if not all diarylide pigments.<br>For the inhalation route the effects seen are related to the deposition of dust particles in the lungs, leading to Pigment Yellow 13 related effects even at the lowest exposure concentration of 54 mg/m3 (local LOAEL). Systemically no effects were observed at the highest concentration tested, 410 mg/m3 (systemic NOAEL).<br>All three pigments are not genotoxic in bacterial tests. Pigment Yellow 12 did not induce clastogenic effects in mammalian cells. Based on the chemical similarities between the three pigments, it is predicted that all three Yellow Pigments will not induce chromosomal changes in mammalian cells. There are no in vitro data to suggest that the pigments are genotoxic in vivo.<br>No increased tumour incidence after treatment with Pigment Yellow 12 and 83 were observed in several long-term studies in rats and mice (NOAEL (rat) > 630 mg/kg; NOAEL (mouse) > 1,960 mg/kg). Based on chemical similarity it can be concluded that the pigments are not carcinogenic.<br>It can be concluded that Pigment Yellow 12 does not have any adverse effects on reproductive parameters. There was no evidence of teratogenicity. The NOAEL for maternal and reproductive toxicity is >1,000 mg/kg bw. Supporting evidence is also available from the fact that no changes on the reproductive organs were observed in the studies of repeat dose toxicity and carcinogenicity study with Pigment Yellow 83. In view of the structural similarities and similar kinetics no effects on reproduction or development are expected from pigments of this class. |

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|---|---|
| | In studies of the bioavailability of several representatives of this group of pigments, no carcinogenic cleavage product was released in detectable amounts after oral, inhalative or intratracheal application on rats.<br>One further study of the bioavailability of DCB (DCB haemoglobin adduct) has been performed with the diarylide pigments C.I. Pigment Yellow 13 and C.I. Pigment Yellow 17. In this study, no release of carcinogenic DCB from the pigments has been detected. This indicates the absence of metabolism to DCB under the test conditions.<br>In summary then, according to the known studies, diarylide pigments do not represent any health risk although risks might attach to contaminants introduced during synthesis.<br>Colourants for Food Contact Plastics - Aspects of Product Safety; Responsible Care initiative of the European Chemical Industry Council.<br>For 3,3'-dichlorobenzidine:<br>Various tumours developed after oral or subcutaneous administration of 3,3'-dichlorobenzidine to mice, rats, hamsters and dogs. Tumours have not yet been identified in persons exposed to the substance alone. The substance can be absorbed through the skin in dangerous quantities. Increases in temperature and relative humidity promote dermal absorption.<br>Upper respiratory infection and sore throat were listed among several principal reasons for visits to a company s medical clinic by workers handling 3,3 -dichlorobenzidine dihydrochloride However, there is no conclusive evidence that these effects were due to inhalation of 3,3 -dichlorobenzidine dihydrochloride.<br>No adverse health effects were observed in male rats exposed by inhalation to 3,3 - dichlorobenzidine free base (23,700 mg/m3) 2 hours per day for 7 days . In another study, 10 rats were exposed to an unspecified concentration of 3,3 -dichlorobenzidine dihydrochloride dust particles for 1 hour and then observed for 14 days. Slight-to-moderate pulmonary congestion and one pulmonary abscess were observed upon necropsy . The effects observed in the study using the ionized (hydrochloride) form of 3,3 -dichlorobenzidine may have been due to the irritative properties of hydrochloric acid released from the salt in combination with particulate toxicity.<br>Gastrointestinal upset was one of the symptoms reported by employees who worked with 3,3 -dichlorobenzidine dihydrochloride. However, there is no conclusive evidence that the gastrointestinal effects, or other symptoms reported by employees, resulted specifically from inhalation of 3,3 -dichlorobenzidine dihydrochloride.<br>The only relevant information regarding neurological effects in humans exposed to 3,3 -dichlorobenzidine was found in an early study which reported that headache and dizziness were among several principal reasons why employees working with 3,3 -dichlorobenzidine in a chemical manufacturing plant visited the company medical clinic. However, there is no conclusive evidence that these symptoms were caused specifically by 3,3 -dichlorobenzidine since there was exposure to other chemicals as well. In a 3,3 -dichlorobenzidine carcinogenicity study, 1 of 6 dogs exhibited convulsions after 21, 28, or 42 months of oral treatment with 10.4 mg/kg/day over a period of 3.5 years<br>**Carcinogenicity:** Several epidemiological studies have investigated cancer incidences among workers occupationally exposed to 3,3 -dichlorobenzidine . Exposure may have been by both inhalation and dermal routes. Due, in part, to structure-activity considerations, epidemiological studies of potential cancer effects of occupational exposure to 3,3 -dichlorobenzidine have been particularly concerned with bladder tumors, since 3,3 -dichlorobenzidine is structurally similar to benzidine, a chemical which is known to be a human bladder carcinogen. No bladder tumors were found in a group of 35 workers who handled only 3,3 -dichlorobenzidine; in the same dyestuff plant, bladder tumors occurred in 3 out of 14 workers exposed to both benzidine and 3,3 -dichlorobenzidine. The investigator reported a total exposure time of 68,505 hours, equivalent to nearly 140 full-time working years. No cases of bladder tumors were found in an epidemiology study of 259 workers exposed to dry and sernidry 3,3 -dichlorobenzidine base and hydrochloride. Workers were exposed to an average of less than 16 years each to 3,3 -dichlorobenzidine, which means that an adequate exposure duration and/or the latent period following exposure may not have been reached for tumor expression.<br>In a retrospective epidemiological study of workers employed in a dye and pigment manufacturing plant that used 3,3 -dichlorobenzidine as chemical precursor, no bladder tumors were observed in a cohort of 207 workers, most of whom had been exposed for up to 15 years. Limitations of this study included using data from a very small and incomplete sample of workers; focusing solely on the occurrence of bladder tumors; and using data that may have been misleading and, at times, apparently inaccurate.<br>A statistically significant increased incidence of hepatomas was observed in male ICR/JCL mice exposed to 0.1% 3,3 -dichlorobenzidine in the diet (170 mg/kg/day) at 6 months (8 of 8 treated as opposed to 0 of 5 controls) and 12 months (18 of 18 treated as opposed to 2 of 2 1 controls). Hepatic tumors were observed in 4/l 8 strain D mice exposed to 11.2-l 1.9 mg 3,3 -dichlorobenzidine/kg/day in the diet for 10 months<br>No bladder carcinomas were observed in rats exposed to 0.03% 3,3 -dichlorobenzidine in the diet<br>(27 mg/kg/day) for 4 or 40 weeks , nor were any mammary tumors observed in rats administered approximately 49 mg 3,3 -dichlorobenzidine dihydrochloride/kg/day by gavage once every 3 days over a 30-day period and sacrificed 8 months later.<br>In a study in which rats were exposed to 10-20 mg 3,3 -dichlorobenzidine per day (120 mg/kg/day) in feed 6 days per week for 12 months, tumors were observed at a variety of sites, including the Zymbal gland (7 of 29 animals), mammary gland (7/29), bladder (3/29), hematopoietic system (3/29), skin (3/29), ileum (2/29), connective tissue (2/29), salivary gland (2/29), liver (l/29), and thyroid (l/29).<br>In another rat study, 3,3 -dichlorobenzidine was administered to 50 male (70 mg/kg/day) and 50 female (80 mg/kg/day) Sprague-Dawley rats, in a standard diet for up to 16 months . In rats fed 3,3 -dichlorobenzidine in the diet for a total of 349 days (females) and 353 days (males), histopathological evaluations revealed mammary adenocarcinoma (16% incidence), malignant lymphoma (14%) granulocytic leukemia (20%), carcinoma of the Zymbal gland (18%) in males, and mammary adenocarcinoma (59%) in females. The authors noted that most of these tumors appeared to arise in the bone marrow and haematopoietic foci in the spleen and liver with subsequent metastasis to other organs.<br>**Haematological Effects**. Although haematological effects may not be sensitive indicators for 3,3 -dichlorobenzidine toxicity, haemoglobin adducts have been detected in female Wistar rats orally administered single 127 or 253 mg/kg doses of 3,3 -dichlorobenzidine or with repeated doses between 0.3 and 5.8 mg/kg/day . It was suggested that metabolically formed nitroso derivatives and the formation of a sulfinic acid amide with cysteine residues in haemoglobin may be the mechanism of adduct formation.<br>**Hepatic Effects**. Limited animal evidence suggests that chronic-duration oral exposure to 3,3 -dichlorobenzidine results in mild-to-moderate liver injury.<br>**Genotoxic effects:** Genotoxic effects have been reported in animals treated with 3,3 -dichlorobenzidine. A single dose of 3,3 -dichlorobenzidine (1,000 mg/kg) administered to male and pregnant female mice induced micronuclei in polychromatic erythrocytes in the bone marrow of the males and in the liver of the foetuses, but not in bone marrow of the dams.<br>In another study, an increase in unscheduled deoxyribonucleic acid synthesis (UDS) was observed in cultured liver cells from male mice previously pretreated orally with single doses of . 500 mg/kg 3,3 -dichlorobenzidine; no response was observed at a dose of .200 mg/kg. 3,3 -Dichlorobenzidine was also shown to bind extensively to tissue deoxyribonucleic acid (DNA) in rats and mice |
| **2-BROMO-2-NITROPROPAN-1,3-DIOL** | Formaldehyde generators (releasers) are often used as preservatives. The maximum authorised concentration of free formaldehyde is 0.2% and must be labelled with the warning sign "contains formaldehyde" where the concentration exceeds 0.05%.The use of formaldehyde-releasing preservatives ensures that the level of free formaldehyde in the products is always low but sufficient to inhibit microbial growth - it disrupts metabolism to cause death of the organism. However there is a concern that formaldehyde generators can produce amines capable of causing cancers (nitrosamines) when used in formulations containing amines.<br>Chemical with the aliphatic nitro group (-C-NO2) have been added to a list of DNA-reactive subgroups recognised by the National Toxicological Program (NTP, U.S. Dept Health and Human Services) for possible carcinogenic activity. |
| **3-IODO-2-PROPYNYL BUTYL CARBAMATE** | 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>for carbamates:<br>Carbamates are effective insecticides by virtue of their ability to inhibit acetylcholinesterase (AChE) (EC 3.1.1.7) in the nervous system. They can also inhibit other esterases. The carbamylation of the enzyme is unstable, and the regeneration of AChE is relatively rapid compared with that from a phosphorylated enzyme. Thus, carbamate pesticides are less dangerous with regard to human exposure than |

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organophosphorus pesticides. The ratio between the dose required to produce death and the dose required to produce minimum symptoms of poisoning is substantially larger for carbamate compounds than for organophosphorus compounds. A dose-effect relationship exists between the dose, the severity of symptoms, and the degree of cholinesterase (ChE) inhibition. Because most carbamates have a low volatility, inhalation studies are mainly carried out using a dust or mist. In these studies, the toxicity is highly dependent on the size of the particles or droplets and, therefore, difficult to evaluate. The acute dermal toxicity of carbamates is generally low to moderate.
From controlled human studies, it is clear that poisoning symptoms can be seen a few minutes after exposure, and can last for a few hours. Thereafter, recovery starts and within hours, the symptoms disappear, and the ChE activity in erythrocytes and plasma returns to normal, because the carbamate is rather rapidly metabolised and the metabolites excreted. The appearance of these metabolites in the urine may be used for biological monitoring. Apart from the symptoms indicative of ChE poisoning, other signs and symptoms induced by certain carbamates have been described, such as skin and eye irritation, hyperpigmentation, and influence on the function of testes (slight increase of sperm abnormalities). These signs and symptoms were found in a few studies and should be confirmed before it can be stated that they were induced by carbamates. Epidemiological studies with persons primarily exposed to carbamates are not available.
Carbamates produce slight to moderate skin and eye irritation, depending on the vehicle used, duration of contact, and on whether the substance is applied to the abraded or intact skin. From the available data, it cannot be excluded that some of the carbamates will have a slight to moderate sensitization potential. Short- and long-term toxicity studies have been carried out. Some carbamates are very toxic and others are less toxic in long- term studies. From these studies, it is evident that, apart from the anticholinesterase activity, the following changes can be found: an influence on the haemopoietic system, an influence on the functioning of, and, at higher dosages, degeneration of, the liver and kidneys, and degeneration of testes. These abnormalities in the different organ systems depend on the animal strain and on the chemical structure of the carbamate. A clear influence on the nervous system, functional as well as histological, was found, particularly in non-laboratory animals such as pigs.
A considerable number of reproduction and teratogenicity studies have been carried out with different carbamates and various animal species. Different types of abnormalities were found, i.e., increase in mortality, disturbance of the endocrine system, and effects on the hypophysis and its gonadotrophic function. These effects were mainly seen at high dose levels. Generally, the fetal effects included an increase in mortality, decreased weight gain in the first weeks after birth, and induction of early embryonic death. All these effects can be summarized as embryotoxic effects. Certain carbamates also induce teratogenic effects, mainly at high dose levels applied by stomach tube. When the same dose level was administered with the diet, no effects were seen.
Some carbamates induce mutagenic effects, others are negative. In general, the methyl carbamates are negative in mammalian tests, while compounds such as carbendazim, benomyl, and the 2 thiophanate derivatives showed a positive effect with very high dose levels in certain systems. The benzimidazole moiety may act as a base analogue for DNA and as a spindle poison. They are antimitotic agents and cause mitotic arrest, mitotic delay, and a low incidence of chromosome damage. Sometimes, the results are contradictory or cannot be reproduced, but positive results for point mutation and chromosome aberrations are well documented. These benzimidazole derivatives can be considered as weak mutagenic compounds.
Carcinogenicity studies with benzimidazole derivatives showed either positive or equivocal results. Added to the fact that certain mutagenicity studies also give positive results, it cannot be excluded that these compounds may have carcinogenic or promotor properties.
Carbamate pesticides may be converted to ***N*** -nitroso compounds. This was demonstrated in a great number of ***in vivo*** nitrosation studies in which high levels of the carbamates were administered to animals in combination with high levels of nitrite. These ***N*** - nitroso compounds have to be considered as mutagenic and carcinogenic. However, the amount of nitroso compounds that can be expected to result from dietary intake of carbamate pesticide residues is negligible in comparison with nitroso-precursors that occur naturally in food and drinking-water.
The metabolic fate of carbamates is basically the same in plants, insects, and mammals. Carbamates are usually easily absorbed through the skin, mucous membranes, and respiratory and gastrointestinal tracts, but there are exceptions. Generally, the metabolites are less toxic than the parent compounds. However, in certain cases, the metabolites are just as toxic or even more toxic than the parent carbamate. In most mammals, the metabolites are mainly excreted rather rapidly in the urine. The dog seems to be different in this respect. Accumulation takes place in certain cases, but is of minor importance because of the rapid metabolism. The first step in the metabolism of carbamates is hydrolysis to carbamic acid, which decomposes to carbon dioxide (CO2) and the corresponding amine. The rate of hydrolysis by esterases is faster in mammals than in plants and insects.
The organs in which residues have been reported are the liver, kidneys, brain, fat, and muscle. The half-life in the rat is of the order of 3 - 8 h. From the limited data available, it seems that the excretion of carbamates via urine is also rapid in man, and that the metabolic pathways in man are the same as those in the rat
for 3-iodo-2-propynyl butyl carbamate (IPBC):
**Acute toxicity:** Acceptable acute toxicity studies with IPBC indicate low toxicity except eye irritation. In a primary eye irritation study in rabbi. IPBC technical was severely irritating to the eyes of white rabbits, with corneal opacity and corneal vascularization reported in unwashed eyes by day 21 post-treatment. The technical grade of IPBC was slightly irritating to the skin of white rabbits. In a dermal sensitization study in Guinea pigs
IPBC technical, at a concentration of 0.32%, produced no evidence of sensitization in male and female Guinea pigs.
**Subchronic toxicity:** In a subchronic oral toxicity study, male and female Sprague-Dawley rats received IPBC technical by gavage for 13 weeks at doses of 0, 20, 50, and 125 mg/kg/day. At the 125 mg/kg/day dose level, body weight gain was decreased by 19% in male rats for weeks 1-13 of the study, and by 12% in female rats over the same period. Absolute liver weight was increased by 20% in male rats at the 125 mg/kg/day dose, and by 31% in female rats at this dose level. Liver to body weight ratio was significantly increased by approximately 31% in both male and female rats at the 125 mg/kg/day dose level, while kidney to body weight ratio in female rats was increased 18% at the 125 mg/kg/day dose level. The systemic NOEL was considered to be 20 mg/kg/day, while the systemic LEL was considered to be 50 mg/kg/day, based on increased liver to body weight ratio.
In a subchronic dermal toxicity study, male and female Sprague-Dawley rats (10/sex/dose) received dermal doses of 50, 200, and 500 mg/kg/day IPBC technical grade (97.5%) to the shaved skin for five days a week, six hours per day. At the 500 mg/kg/day dose, decreased body weight (4-6%) and weight gain (11%) were observed in male rats, but not in female rats. In female rats, significant increases in haemoglobin, haematocrit, and eosinophils were observed at the 500 mg/kg/day dose level. Reticulocytes as a percentage of red cells were decreased in the 50 and 200 mg/kg/day dose groups but not at the 500 mg/kg/day dose level. Females in this study showed inhibition of plasma cholinesterase at 500 mg/k/day test article, which may have been the result of either direct liver toxicity or inhibition of cholinesterase itself. Based upon the results of this study, the systemic NOEL is 200 mg/kg/day, the systemic LEL is 500 mg/kg/day for male and female rats.
**Carcinogenicity:** In a 2-year chronic toxicity/carcinogenicity study, technical grade IPBC (98.68% ai) was administered to male and female Sprague Dawley rats (50/sex/group) at dose levels of 0, 20, 40, and 80 mg/kg/day. There were no statistically significant increases in tumor incidences in male rats. The incidence of mammary gland fibroadenoma and combined fibroadenoma/carcinoma in female rats was significantly increased at the 20 mg/kg/day dose level but there was no dose-related trend.
**Developmental and reproductive toxicity:** The developmental toxicity of IPBC was assessed in pregnant Sprague-Dawley rats on gestation days six through 15 by oral administration of the test chemical at doses of 0, 20, 50, and 125 mg/kg/day. Maternal toxicity as reduced body weight gain during dosing was observed at the 125 mg/kg/day dose level. Developmental toxicity consisted of an increased incidence of skeletal abnormalities at the 125 mg/kg/day dose level. The maternal toxicity NOEL was determined to be 50 mg/kg/day, and the maternal toxicity LEL was determined to be 125 mg/kg/day, based on reduced body weight gain. The developmental toxicity NOEL was determined to be 50 mg/kg/day, and the developmental toxicity LEL was determined to be 125 mg/kg/day, based on incompletely ossified frontal skull bones and pelvic girdles.
A 2-generation reproductive toxicity study was conducted in male and female Sprague- Dawley rats. IPBC technical was administered over two generations at doses of 0, 120, 300, and 750 ppm (0, 6, 15, and 37.5 mg/kg/day). Reduced body weight and food consumption was observed for P1 and F1 males during the premating period at the 37.5 mg/kg/day dose. A decreased mean live birth index was reported for P1 and F1 generations without an effect on viability and development of pups. No adverse effects on reproductive indices or mating performance were observed at any dose level. The parental toxicity NOEL was determined to be 15 mg/kg/day, and the parental toxicity LEL was determined to be 37.5 mg/kg/day, based on decreased body weight and food consumption during premating for P1 and F1 males, and decreased mean live birth index for the P1 and F1 generations. The reproductive toxicity Noel was determined to be 37.5 mg/kg/day, and the reproductive toxicity LEL was determined to be >37.5 mg/kg/day.
**Mutagenicity:** In a mutagenicity study, IPBC technical was tested for the ability to cause mutations in ***Salmonella typhimurium*** strains TA 1535, TA 1537, TA 1538, TA 98, and TA 100. In the five strains used, IPBC was found to be non-mutagenic in the presence or absence of

**Continued...**

| | |
|---|---|
| | metabolic activation at the concentrations tested, 1-1000 jig/plate . In a micronucleus assay in mice, IPBC at doses of 200, 600, and 2000 mg/kg did not induce any significant increase of the PCE containing micronuclei from the treated mice when compared to that of the vehicle control mice. In two independent unscheduled DNA synthesis (UDS) assays in primary rat hepatocytes, eight doses of IPBC ranging from 3.0 to 13.5 ug/ml did not cause an appreciable increase in mean net nuclear grain counts. Doses >13.5 ug/ml were cytotoxic, supporting the conclusion that IPBC induced cytotoxicity but no genotoxicity in this assay.<br>**Metabolism:** Based on the metabolite identification data, a scheme for metabolism of IPBC was proposed. According to this scheme, IPBC undergoes reductive dehalogenation followed by dealkylation to form the URM-9 and URM-10 metabolites. In addition, de-carboxylation following reductive dehalogenation yields carbon dioxide. Various other metabolites formed from dehalogenation are glucuronidated and constitute minor metabolites of IPBC.. |
| **KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB METALLIC & WATER & ALUMINIUM** | No significant acute toxicological data identified in literature search. |
| **GLYCEROL & 2-BROMO-2-NITROPROPAN-1,3-DIOL** | Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production. |
| **PROPYLENE GLYCOL & 2-BROMO-2-NITROPROPAN-1,3-DIOL** | The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. |

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

**KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB METALLIC**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**water**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**glycerol**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC0(ECx) | 24h | Crustacea | >500mg/l | 1 |
| LC50 | 96h | Fish | >11mg/L | 2 |

**propylene glycol**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 72h | Algae or other aquatic plants | 19300mg/l | 2 |
| EC50 | 48h | Crustacea | >114.4mg/L | 4 |
| EC50 | 96h | Algae or other aquatic plants | 19000mg/l | 2 |
| NOEC(ECx) | 336h | Algae or other aquatic plants | <5300mg/l | 1 |
| LC50 | 96h | Fish | 710mg/L | 4 |

**aluminium**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 72h | Algae or other aquatic plants | 0.017mg/L | 2 |
| EC50 | 48h | Crustacea | 0.736mg/L | 2 |
| NOEC(ECx) | 72h | Algae or other aquatic plants | >100mg/l | 1 |
| EC50 | 96h | Algae or other aquatic plants | 0.005mg/L | 2 |
| LC50 | 96h | Fish | 0.078-0.108mg/l | 2 |

**C.I. Pigment Red 170**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 72h | Algae or other aquatic plants | >1mg/l | 2 |
| EC50 | 48h | Crustacea | >100mg/l | 2 |
| ErC50 | 72h | Algae or other aquatic plants | 17mg/l | 2 |
| NOEC(ECx) | 72h | Algae or other aquatic plants | 1mg/l | 2 |
| LC50 | 96h | Fish | >100mg/l | 2 |

**C.I. Pigment Yellow 83**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|

**Continued...**

| | | | | | |
|---|---|---|---|---|---|
| | EC50 | 72h | Algae or other aquatic plants | >100mg/l | 2 |
| | EC50 | 48h | Crustacea | >100mg/l | 2 |
| | NOEC(ECx) | 24h | Fish | >=0.1mg/l | 2 |
| | LC50 | 96h | Fish | >0.1mg/l | 2 |

| **2-bromo-2-nitropropan-1,3-diol** | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
|---|---|---|---|---|---|
| | EC50 | 72h | Algae or other aquatic plants | 0.026mg/l | 2 |
| | EC50 | 48h | Crustacea | 1.1-3.52mg/L | 4 |
| | EC50 | 96h | Algae or other aquatic plants | 0.02-0.025mg/L | 4 |
| | EC10(ECx) | 72h | Algae or other aquatic plants | 0.013mg/l | 2 |
| | LC50 | 96h | Fish | 10.274-14.454mg/L | 4 |

| **3-iodo-2-propynyl butyl carbamate** | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
|---|---|---|---|---|---|
| | EC50 | 72h | Algae or other aquatic plants | 0.022mg/L | 2 |
| | EC50 | 48h | Crustacea | 0.04mg/L | 5 |
| | NOEC(ECx) | 0.5h | Fish | <0.001mg/L | 4 |
| | LC50 | 96h | Fish | 0.05-0.089mg/L | 4 |

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

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
For glycerol
log Kow : -2.66- -2.47
BOD 5: 0.617-0.87,31-51%
COD : 1.16,82-95%
ThOD : 1.217-1.56
Completely biodegradable.
Environmental fate:
Based on the relevant physical-chemical properties and the fact that glycerol is readily biodegradable, glycerol will partition primarily to water.
Biodegradability: Glycerol is considered to be readily biodegradable in the aquatic environment. Pre-adapted microorganisms can degrade glycerol rapidly under both aerobic and anaerobic conditions.
Bioaccumulation: Based on Log Kow -1.76, glycerol will have a low bioaccumulation potential and is not expected to bioaccumulate.
Photodegradation: The calculated half-life for the photo-oxidation (reaction with hydroxyl radicals) of glycerol in air is 6.8 hours (EPIWIN vs 3.04).
Stability in Water: Glycerol does not contain functional groups that are expected to react with water.
Transport between Environmental Compartments: From the EQC model (Mackay level III), it can be deduced that 100% of glycerol will end up in the water phase. Negligible amounts will be distributed towards soil, air and sediment
Ecotoxicity:
Fish LC50: >5000 mg/l
Algae IC50: >2900 mg/l
Bacteria EC50: .10000 mg/l (Pseudomonas putida)
The weight of evidence indicates that glycerol is of low toxicity to aquatic organisms and this conclusion is supported by QSAR predictions. The lowest LC50 for fish is a 24-h LC50 of >5000 mg/l for Carassius auratus (Goldfish) and for aquatic invertebrates, a 24 h EC50 of >10000 mg/l for Daphnia magna is the lowest EC50. Several tests on algae are available, which suggest very low toxicity to a range of species, however their validity is uncertain. A QSAR prediction for the 96h EC50 to algae was 78000 mg/l. No toxicity towards the microorganism Pseudomonas putida was observed at 10000 mg/l after exposure for 16 hours. No long-term aquatic toxicity data is available. Screening studies are available on frog and carp embryos which indicate some effects on growth and hatching rates respectively at very high concentrations of glycerol, >7000 mg/l. However, their ecological relevance is not clear.

For 2-bromo-2-nitropropan-1,3-diol (Bronopol)
**Environmental fate:**
One hydrolysis study indicates that bronopol appears to hydrolyse slowly at acidic or neutral pH conditions. Bronopol decomposes in aqueous solution on exposure to light. Increases in temperature increase decomposition.
**Ecotoxicity:**
Bird LD50: mallard duck 510 mg/kg
Bird dietary LC50: quail 4488 ppm
Daphnia magna EC50 (48 h): 1.4 mg/l
Fish LC50: trout 41.5 ppm
**DO NOT** discharge into sewer or waterways.

### Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| water | LOW | LOW |
| glycerol | LOW | LOW |
| propylene glycol | LOW | LOW |
| C.I. Pigment Yellow 83 | HIGH | HIGH |
| 2-bromo-2-nitropropan-1,3-diol | LOW | LOW |
| 3-iodo-2-propynyl butyl carbamate | HIGH | HIGH |

### Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| water | LOW (LogKOW = -1.38) |
| glycerol | LOW (LogKOW = -1.76) |

**Continued...**

| Ingredient | Bioaccumulation |
|---|---|
| propylene glycol | LOW (BCF = 1) |
| aluminium | LOW (LogKOW = 0.33) |
| C.I. Pigment Yellow 83 | LOW (LogKOW = 8.6648) |
| 2-bromo-2-nitropropan-1,3-diol | LOW (LogKOW = -0.6408) |
| 3-iodo-2-propynyl butyl carbamate | LOW (LogKOW = 2.4542) |

**Mobility in soil**

| Ingredient | Mobility |
|---|---|
| glycerol | HIGH (Log KOC = 1) |
| propylene glycol | HIGH (Log KOC = 1) |
| C.I. Pigment Yellow 83 | LOW (Log KOC = 1126000) |
| 2-bromo-2-nitropropan-1,3-diol | HIGH (Log KOC = 1) |
| 3-iodo-2-propynyl butyl carbamate | LOW (Log KOC = 365.3) |

## SECTION 13 Disposal considerations

**Waste treatment methods**

| | |
|---|---|
| **Product / Packaging disposal** | ▸ 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>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.<br>A Hierarchy of Controls seems to be common - the user should investigate:<br>▸ Reduction<br>▸ Reuse<br>▸ Recycling<br>▸ Disposal (if all else fails)<br>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.<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.<br>▸ 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. |

## 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 |
|---|---|
| water | Not Applicable |
| glycerol | Not Applicable |
| propylene glycol | Not Applicable |
| aluminium | Not Applicable |
| C.I. Pigment Red 170 | Not Applicable |
| C.I. Pigment Yellow 83 | Not Applicable |
| 2-bromo-2-nitropropan-1,3-diol | Not Applicable |

**Continued...**

| Product name | Group |
|---|---|
| 3-iodo-2-propynyl butyl carbamate | Not Applicable |

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

| Product name | Ship Type |
|---|---|
| water | Not Applicable |
| glycerol | Not Applicable |
| propylene glycol | Not Applicable |
| aluminium | Not Applicable |
| C.I. Pigment Red 170 | Not Applicable |
| C.I. Pigment Yellow 83 | Not Applicable |
| 2-bromo-2-nitropropan-1,3-diol | Not Applicable |
| 3-iodo-2-propynyl butyl carbamate | Not Applicable |

## SECTION 15 Regulatory information

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

**water is found on the following regulatory lists**
Australian Inventory of Industrial Chemicals (AIIC)

**glycerol is found on the following regulatory lists**
Australian Inventory of Industrial Chemicals (AIIC)

**propylene glycol is found on the following regulatory lists**
Australian Inventory of Industrial Chemicals (AIIC)

**aluminium is found on the following regulatory lists**
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**C.I. Pigment Red 170 is found on the following regulatory lists**
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**C.I. Pigment Yellow 83 is found on the following regulatory lists**
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans

**2-bromo-2-nitropropan-1,3-diol is found on the following regulatory lists**
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

**3-iodo-2-propynyl butyl carbamate 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
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**Additional Regulatory Information**
Not Applicable

**National Inventory Status**

| National Inventory | Status |
|---|---|
| Australia - AIIC / Australia Non-Industrial Use | Yes |
| Canada - DSL | Yes |
| Canada - NDSL | No (water; glycerol; propylene glycol; aluminium; C.I. Pigment Red 170; C.I. Pigment Yellow 83; 2-bromo-2-nitropropan-1,3-diol; 3-iodo-2-propynyl butyl carbamate) |
| China - IECSC | Yes |
| Colombia - Colombia National Industrial Chemicals Inventory | No (water; glycerol; propylene glycol; aluminium; C.I. Pigment Red 170; C.I. Pigment Yellow 83; 2-bromo-2-nitropropan-1,3-diol; 3-iodo-2-propynyl butyl carbamate) |
| Europe - EINEC / ELINCS / NLP | Yes |
| Japan - ENCS | No (aluminium) |
| Korea - KECI | Yes |

**Continued...**

| National Inventory | Status |
|---|---|
| New Zealand - NZIoC | Yes |
| Philippines - PICCS | Yes |
| USA - TSCA | All chemical substances in this product have been designated as TSCA Inventory 'Active' |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (C.I. Pigment Yellow 83) |
| Vietnam - NCI | Yes |
| Russia - FBEPH | Yes |
| UAE - Control List (Banned/Restricted Substances) | No (water; glycerol; propylene glycol; C.I. Pigment Red 170; C.I. Pigment Yellow 83; 2-bromo-2-nitropropan-1,3-diol; 3-iodo-2-propynyl butyl carbamate) |
| ***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** | 28/10/2026 |
| **Initial Date** | 28/10/2026 |

**SDS Version Summary**

| Version | Date of Update | Sections Updated |
|---|---|---|
| 2.1 | 28/10/2026 | Identification of the substance / mixture and of the company / undertaking - Synonyms |

**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
- 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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