Safety Data Sheet according to WHS and ADG requirements

Issue Date: 20/09/23

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## SECTION 1 Identification of material and supplier

| Product Identifier | |
|---|---|
| Product Name | SC100 Epoxy Tint |
| Product code | AFS131AW, AFS132ABL, AFS133ANG, AFS134ALG, AFS135APG, AFS136AKG, AFS137AP |
| Chemical Name | Not Applicable |
| Synonyms | Not Available |
| Details of the supplier of the safety data | Australian Flooring Systems Pty Ltd |
| Proper shipping name | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid) |
| 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 | Resin based Epoxy Colourant / To be used with Epoxy Resins Only.<br>Use according to manufacturer's directions. |
| **Details of the manufacturer or supplier of the safety data sheet** | |
| Address | 60 Victoria Street Riverstone NSW 2765 |
| Telephone | 13 11 26(Poisons Information Centre) |
| Website | www.ausflooringsystems.com.au |
| Email | info@ausflooringsystems.com.au |
| Emergency telephone number | 13 11 26 |

## SECTION 2 HAZARDS IDENTIFICATION

| Classification of the substance or mixture | |
|---|---|
| Poisons Schedule | S5 |
| Classification [1] | Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 1A, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 4, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Germ Cell Mutagenicity Category 2, Carcinogenicity Category 1A, Hazardous to the Aquatic Environment Long-Term Hazard Category 2 |
| Legend: | 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI |
| **Label elements** | |
| Hazard pictogram(s) |  |

| | |
|---|---|
| Signal word | Danger |
| **Hazard statement(s)** | |
| AUH019 | May form explosive peroxides. |
| H304 | May be fatal if swallowed and enters airways. |
| H314 | Causes severe skin burns and eye damage |
| H317 | May cause an allergic skin reaction. |
| H332 | Harmful if inhaled. |
| H335 | May cause respiratory irritation. |
| H336 | May cause drowsiness or dizziness. |
| H341 | Suspected of causing genetic defects |
| H350 | May cause cancer |
| H411 | Toxic to aquatic life with long lasting effects |
| **Precautionary statement(s) Prevention** | |
| P201 | Obtain special instructions before use. |
| P260 | Do not breathe mist/vapours/spray. |
| P264 | Wash all exposed external body areas thoroughly after handling. |
| P271 | Use only outdoors or in a well-ventilated area. |
| P280 | Wear protective gloves, protective clothing, eye protection and face protection. |
| P273 | Avoid release to the environment. |
| P272 | Contaminated work clothing should not be allowed out of the workplace. |
| **Precautionary statement(s) Response** | |
| P301+P310 | IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider. |
| P301+P330+P331 | IF SWALLOWED: Rinse mouth. Do NOT induce vomiting. |
| P303+P361+P353 | IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower]. |
| P305+P351+P338 | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| P308+P313 | IF exposed or concerned: Get medical advice/ attention. |
| P302+P352 | IF ON SKIN: Wash with plenty of water and soap |
| P363 | Wash contaminated clothing before reuse |
| P333+P313 | If skin irritation or rash occurs: Get medical advice/attention. |
| P362+P364 | Take off contaminated clothing and wash it before reuse |
| P391 | Collect spillage. |
| P304+P340 | IF INHALED: Remove person to fresh air and keep comfortable for breathing. |
| **Precautionary statement(s) Storage** | |
| P405 | Store locked up |
| P403+P233 | Store in a well-ventilated place. Keep container tightly closed. |
| **Precautionary statement(s) Disposal** | |
| P501 | Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation. |

## SECTION 3: COMPOSITION / INFORMATION ON INGREDIENTS

**Substances** - See section below for composition of Mixtures

**Mixtures**

| CAS No | %[weight] | Name |
|---|---|---|
| 25068-38-6 | 20-60 | bisphenol A/ diglycidyl ether resin, liquid |
| 100-51-6 | 0-10 | benzyl alcohol |
| 13463-67-7 | 0-65 | titanium dioxide |
| 1333-86-4 | 0-25 | carbon black |
| 108-65-6 | 0-25 | propylene glycol monomethyl ether - mixture of isomers |

| | | |
|---|---|---|
| 64742-48-9 | 0-15 | naphtha petroleum, isoparaffin, hydrotreated |
| 1313-13-9 | <5 | manganese dioxide |
| 9046-01-9 | 0-5 | tridecanol ethoxylated, phosphated |
| 68412-54-4 | <5 | nonylphenol ethoxylate, branched |
| Not Available | Balance | Ingredients determined not to be hazardous |
| Legend: | 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available | |

## SECTION 4: FIRST AID MEASURES

### Description of first aid measures

| | |
|---|---|
| Eye Contact | If this product comes in contact with the eyes:<br>Immediately hold eyelids apart and flush the eye continuously with 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>Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.<br>Transport to hospital or doctor without delay.<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. |
| 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 | If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.<br>Observe the patient carefully.<br>Never give liquid to a person showing signs of being sleepy or with reduced awareness, i.e. becoming unconscious.<br>Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.<br>Transport to hospital or doctor without delay.<br>Avoid giving milk or oils.<br>Avoid giving alcohol.<br>If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus. |

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

Any material aspirated during vomiting may produce lung injury. Therefore, emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

Treat symptomatically

## SECTION 5 FIREFIGHTING MEASURES

### Extinguishing media

- Alcohol stable foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - large fires only.

| Special hazards arising from the substrate or mixture | |
|---|---|
| Fire Incompatibility | Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result |
| **Advice for firefighters** | |
| Fire Fighting | Alert Fire Brigade and tell them location and nature of hazard.<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 (CO2)<br>aldehydes<br>nitrogen oxides (NOx)<br>phosphorus oxides (POx)<br>sulfur oxides (SOx)<br>metal oxides<br>other pyrolysis products typical of burning organic material. |
| HAZCHEM | •3Z |

## SECTION 6 ACCIDENTAL RELEASE MEASURES

**Personal precautions, protective equipment and emergency procedures**

See section 8

**Environmental precautions**

See section 12

**Methods and material for containment and cleaning up**

| | |
|---|---|
| Minor Spills | Environmental hazard - contain spillage.<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 | Environmental hazard - contain spillage.<br>Moderate hazard.<br>Clear area of personnel and move upwind.<br>Alert Fire Brigade and tell them location and nature of hazard.<br>Wear breathing apparatus plus protective gloves.<br>Prevent, by any means available, spillage from entering drains or water course.<br>No smoking, naked lights or ignition sources.<br>Increase ventilation.<br>Stop leak if safe to do so.<br>Contain spill with sand, earth or vermiculite.<br>Collect recoverable product into labelled containers for recycling.<br>Absorb remaining product with sand, earth or vermiculite.<br>Collect solid residues and seal in labelled drums for disposal.<br>Wash area and prevent runoff into drains.<br>If contamination of drains or waterways occurs, advise emergency services |

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

## SECTION 7 HANDLING AND STORAGE

**Precautions for safe handling**

| | |
|---|---|
| Safe handling | DO NOT allow clothing wet with material to stay in contact with skin<br>Avoid all personal contact, including inhalation.<br>Wear protective clothing when risk of exposure occurs.<br>Use in a well-ventilated area.<br>Prevent concentration in hollows and sumps.<br>DO NOT enter confined spaces until atmosphere has been checked.<br>Avoid smoking, naked lights or ignition sources.<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.<br>Use good occupational work practice. |

| | |
|---|---|
| | 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. |
| Other information | Store in original containers.<br>Keep containers securely sealed.<br>Store in a cool, dry, well-ventilated area.<br>Store away from incompatible materials and foodstuff containers.<br>Protect containers against physical damage and check regularly for leaks.<br>Observe manufacturer's storage and handling recommendations contained within this SDS |
| **Conditions for safe storage, including any incompatibilities** | |
| Suitable container | Lined metal can, lined metal pail/ can.<br>Plastic pail.<br>Polyliner drum.<br>Packing as recommended by manufacturer.<br>Check all containers are clearly labelled and free from leaks.<br>Metal can or drum<br>Packaging as recommended by manufacturer.<br>Check all containers are clearly labelled and free from leaks. |
| Storage incompatibility | Avoid reaction with amines, mercaptans, strong acids and oxidising agents<br>Avoid reaction with oxidising agents<br>Avoid strong acids, acid chlorides, acid anhydrides and chloroformates |

## SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

**Control parameters**

**Occupational Exposure Limits (OEL)**

**INGREDIENT DATA**

| Source | Ingredient | Material name | TWA | STEL | Peak | Notes |
|---|---|---|---|---|---|---|
| Australia Exposure Standards | titanium dioxide | Titanium dioxide | 10 mg/m3 | Not Available | Not Available | (a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica. |
| Australia Exposure Standards | carbon black | Carbon black | 3 mg/m3 | Not Available | Not Available | Not Available |
| Australia Exposure Standards | propylene glycol monomethyl ether - mixture of isomers | Propylene glycol monomethyl ether | 100 ppm / 369 mg/m3 | 553 mg/m3 /150 ppm | Not Available | Not Available |
| Australia Exposure Standards | propylene glycol monomethyl ether - mixture of isomers | -Methoxy-2-propanol acetate | 50 ppm / 274 mg/m3 | 548 mg/m3 /100 ppm | Not Available | Not Available |

| | | | | | | |
|---|---|---|---|---|---|---|
| Australia Exposure Standards | naphtha petroleum, isoparaffin, hydrotreated | Oil mist, refined Mineral | 5 mg/m3 | Not Available | Not Available | Not Available |
| Australia Exposure Standards | manganese dioxide | Manganese, dust & compounds (as Mn) | 1 mg/m3 | Not Available | Not Available | Not Available |

**Emergency Limits**

| Ingredient | TEEL-1 | TEEL-2 | TEEL-3 |
|---|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | 90 mg/m3 | 990 mg/m3 | 5,900 mg/m3 |
| benzyl alcohol | 30 ppm | 52 ppm | 740 ppm |
| titanium dioxide | 30 mg/m3 | 330 mg/m3 | 2,000 mg/m3 |
| carbon black | 9 mg/m3 | 99 mg/m3 | 590 mg/m3 |
| propylene glycol monomethyl ether - mixture of isomers | 100 ppm | 160 ppm | 660 ppm |
| propylene glycol monomethyl ether - mixture of isomers | Not Available | Not Available | Not Available |
| naphtha petroleum, isoparaffin, hydrotreated | 350 mg/m3 | 1,800 mg/m3 | 40,000 mg/m3 |
| naphtha petroleum, isoparaffin, hydrotreated | 1,100 mg/m3 | 1,800 mg/m3 | 40,000 mg/m3 |
| manganese dioxide | 4.7 mg/m3 | 7.9 mg/m3 | 690 mg/m3 |
| manganese dioxide | 4.2 mg/m3 | 6.9 mg/m3 | 41 mg/m3 |
| nonylphenol ethoxylate, branched | 30 mg/m3 | 330 mg/m3 | 2,000 mg/m3 |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | Not Available | Not Available |
| benzyl alcohol | Not Available | Not Available |
| titanium dioxide | 5,000 mg/m3 | Not Available |
| carbon black | 1,750 mg/m3 | Not Available |
| propylene glycol monomethyl ether - mixture of isomers | Not Available | Not Available |
| naphtha petroleum, isoparaffin, hydrotreated | 2,500 mg/m3 | Not Available |
| manganese dioxide | 500 mg/m3 | Not Available |
| tridecanol ethoxylated, phosphate | Not Available | Not Available |
| nonylphenol ethoxylate, branched | Not Available | Not Available |

**Occupational Exposure Banding**

| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | E | ≤ 0.1 ppm |
| benzyl alcohol | E | ≤ 0.1 ppm |
| tridecanol ethoxylated, phosphate | C | > 1 to ≤ 10 parts per million (ppm) |
| nonylphenol ethoxylate, branched | E | ≤ 0.1 ppm |

| | |
|---|---|
| Notes | Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health. |
| MATERIAL DATA | NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7).<br>Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.<br>European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP |

**Exposure controls**

Appropriate engineering Controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.
Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

| Type of Contaminant: | Air Speed: |
|---|---|
| solvent, vapours, degreasing etc., evaporating from tank (in still air). | 0.25-0.5 m/s (50-100 f/min.) |
| aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation) | 0.5-1 m/s (100-200 f/min.) |
| direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 f/min.) |
| grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion) | 2.5-10 m/s (500-2000 f/min.) |

Within each range the appropriate value depends on:

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

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

| | |
|---|---|
| Individual protection measures, such as personal protective equipment |  |
| Eye and face protection | Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories;<br>spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is<br>a danger of splashing, or if the material may be under pressure.<br>Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly<br>fitted.<br>Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these<br>afford face protection.<br>Alternatively a gas mask may replace splash goggles and face shields.<br>Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy<br>document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should<br>include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience.<br>Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the<br>event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should<br>be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers<br>have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] |
| Skin protection | See Hand protection below |
| Hands/feet protection | Elbow length PVC gloves<br>When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.<br>NOTE:<br>The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other<br>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<br>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 | Overalls.<br>P.V.C apron.<br>Barrier cream.<br>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:
AFS SC100 Colour Tint

| Material | CPI |
|---|---|
| BUTYL | C |
| BUTYL/NEOPRENE | C |
| HYPALON | C |
| NAT+NEOPR+NITRILE | C |
| NATURAL RUBBER | C |
| NATURAL+NEOPRENE | C |
| NEOPRENE | C |
| NEOPRENE/NATURAL | C |
| NITRILE | C |
| NITRILE+PVC | C |
| PE | C |
| PE/EVAL/PE | C |
| PVA | C |
| PVC | C |
| PVDC/PE/PVDC | C |
| TEFLON | C |
| VITON | C |
| VITON/BUTYL | C |

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion

**Respiratory protection**

Type AB-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 | AB-AUS P2 | - | AB-PAPR-AUS / Class 1 P2 |
| up to 50 x ES | - | AB-AUS / Class 1 P2 | - |
| up to 100 x ES | - | AB-2 P2 | AB-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

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

used

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### Information on basic physical and chemical properties

| | |
|---|---|
| Appearance | Coloured paint liquid with characteristic odour, immiscible in water. |

| | | | |
|---|---|---|---|
| Physical state | Liquid | Relative density (Water = 1) | Not Available |
| Odour | Characteristic | Partition coefficient n-octanol / water | Not Available |
| Odour threshold | Not Available | Auto-ignition temperature (°C) | Not Available |
| pH (as supplied) | Not Applicable | Decomposition temperature (°C) | Not Available |
| Melting point / freezing point (°C) | Not Available | Viscosity (cSt) | Not Available |
| Initial boiling point and boiling range (°C) | Not Available | Molecular weight (g/mol) | Not Applicable |
| Flash point (°C) | Not Available | Taste | Not Available |
| Evaporation rate | Not Available | Explosive properties | Not Available |
| Flammability | Not Available | Oxidising properties | Not Available |
| Upper Explosive Limit (%) | Not Applicable | Surface Tension (dyn/cm or mN/m) | Not Available |
| Lower Explosive Limit (%) | Not Applicable | Volatile Component (%vol) | Not Available |
| Vapour pressure (kPa) | Not Available | Gas group | Not Available |
| Solubility in water | Immiscible | pH as a solution (1%) | Not Applicable |
| Vapour density (Air = 1) | Not Available | VOC g/L | 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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| Inhaled | Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects.<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 inhalation.<br>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.<br>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.<br>Inhalation hazard is increased at higher temperatures.<br>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination |
| Ingestion | Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.<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 swallowing.<br>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.<br>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). |
| Skin Contact | 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>Skin contact with acidic corrosives may result in pain and burns; these may be deep with distinct edges and may heal slowly with the formation of scar tissue.<br>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.<br>Open cuts, abraded or irritated skin should not be exposed to this material. The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material |

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| | is unlikely to produce an irritant dermatitis as described in EC Directives .<br>The material may accentuate any pre-existing dermatitis condition.<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 | Direct eye contact with acid corrosives may produce pain, lachrymation, photophobia and burns. Mild burns of the epithelia generally recover rapidly and completely. Severe burns produce long-lasting and possible irreversible damage. The appearance of the burn may not be apparent for several weeks after the initial contact. The cornea may ultimately become deeply vascularised and opaque resulting in blindness.<br>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Irritation of the eyes may produce a heavy secretion of tears (lachrymation) |
| Chronic | Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.<br>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.<br>Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.<br>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.<br>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyperresponsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.<br>Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers<br>Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyperresponsive. |

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| | Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.<br>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>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.<br>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.<br>Based on epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans.<br>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.<br>Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.<br>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. |

| | TOXICITY | IRRITATION |
|---|---|---|
| AFS SC100 Epoxy Tint | Not Available | Not Available |

| | TOXICITY | IRRITATION |
|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | dermal (rat) LD50: >1200 mg/kg [2] | Eye (rabbit): 100mg - Mild |
| | Oral (Mouse) LD50; >500 mg/kg [2] | |

| | TOXICITY | IRRITATION |
|---|---|---|
| benzyl alcohol | Dermal (rabbit) LD50: 2000 mg/kg [2] | Eye (rabbit): 0.75 mg open SEVERE |
| | Inhalation (Rat) LC50: >4.178 mg/L4h [2] | Eye: adverse effect observed (irritating) [1] |
| | Oral (Rat) LD50: 1230 mg/kg [2] | Skin (man): 16 mg/48h-mild |
| | | Skin (rabbit):10 mg/24h open-mild |

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| | | Skin: no adverse effect observed (not irritating) [1] |
| titanium dioxide | TOXICITY | IRRITATION |
| | dermal (hamster) LD50: >=10000 mg/kg [2] | Eye: no adverse effect observed (not irritating) [1] |
| | Inhalation (Rat) LC50: >2.28 mg/l4h [1] | Skin (human): 0.3 mg /3D (int)-mild * |
| | Oral (Rat) LD50: >=2000 mg/kg [1] | Skin: no adverse effect observed (not irritating) [1] |
| carbon black | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: >2000 mg/kg [1] | Eye: no adverse effect observed (not irritating) [1] |
| | Oral (Rat) LD50: >2000 mg/kg [1] | Skin: no adverse effect observed (not irritating) [1] |
| propylene glycol monomethyl ether - mixture of isomers | TOXICITY | IRRITATION |
| | dermal (rat) LD50: >2000 mg/kg [1] | Eye (rabbit) 230 mg mild |
| | Oral (Rat) LD50: 3739 mg/kg [2] | Eye (rabbit) 500 mg/24 h. – mild |
| | | Eye: no adverse effect observed (not irritating) [1] |
| | | Skin (rabbit) 500 mg open – mild |
| | | Skin: no adverse effect observed (not irritating) [1] |
| naphtha petroleum, isoparaffin, hydrotreated | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: >1900 mg/kg [1] | Eye: no adverse effect observed (not irritating) [1] |
| | Inhalation (Rat) LC50: >4.42 mg/L4h [1] | Skin: adverse effect observed (irritating) [1] |
| | Oral (Rat) LD50: >4500 mg/kg [1] | |
| manganese dioxide | TOXICITY | IRRITATION |
| | Oral (Rat) LD50: >3478 mg/kg [2] | Eye: no adverse effect observed (not irritating) [1] |
| | | Skin: no adverse effect observed (not irritating) [1] |
| tridecanol ethoxylated, phosphate | TOXICITY | IRRITATION |
| | Oral (Rat) LD50: >2000 mg/kg [2] | Not Available |
| nonylphenol ethoxylate, branched | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: >2000 mg/kg [2] | Eye: Severe |
| | Oral (Rat) LD50: >2000 mg/kg [1] | Eye: no adverse effect observed (not irritating) [1] |
| | | Skin: Severe |
| | | Skin: no adverse effect observed (not irritating) [1] |
| Legend | 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances | |
| BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID | Foetoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity; NOEL (maternal 60 mg/kg<br>The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics.<br>Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities. | |

Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.
In vitro cell models were used to evaluate the ability of 22 bisphenols (BPs) to induce or inhibit estrogenic and androgenic activity. BPA, Bisphenol AF (BPAF), bisphenol Z (BPZ), bisphenol C (BPC), tetramethyl bisphenol A (TMBPA), bisphenol S (BPS), bisphenol E (BPE), 4,4-bisphenol F (4,4-BPF), bisphenol AP (BPAP), bisphenol B (BPB), tetrachlorobisphenol A (TCBPA), and benzylparaben (PHBB) induced estrogen receptor (ER)alpha and/or ERbeta-mediated activity. With the exception of BPS, TCBPA, and PHBB, these same BPs were also androgen receptor (AR) antagonists. Only 3 BPs were found to be ER antagonists. Bisphenol P (BPP) selectively inhibited ERbeta-mediated activity and 4-(4-phenylmethoxyphenyl) sulfonylphenol (BPS-MPE) and 2,4-bisphenol S (2,4-BPS) selectively inhibited ERalpha-mediated activity. None of the BPs induced AR-mediated activity.
The substance is classified by IARC as Group 3:
NOT classifiable as to its carcinogenicity to humans.
Evidence of carcinogenicity may be inadequate or limited in animal testing.
In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to
moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats,
dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The
no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE
(Same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg).
Reproductive and Developmental Toxicity: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1)
or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose,
but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg.
Carcinogenicity: IARC concluded that "there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in
experimental animals." Its overall evaluation was "Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to
humans (Group 3).
In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE
(Undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin
paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study,
BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the
skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally
exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of
tumours in the oral cavity (U.S. EPA, 1997).
Genotoxicity: In S. typhimurium strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg)

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| | failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg).<br>Immunotoxicity: Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8<br>injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs - Consumer exposure to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies supports a NOAEL of 50 mg/ kg/body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weigh/day (male rats) from the 2-year carcinogenicity<br>study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg<br>body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of<br>safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of<br>BADGE for use in articles intended to come into contact with foodstuffs. |
| BENZYL ALCOHOL | For benzyl alkyl alcohols:<br>Unlike benzylic alcohols, the beta-hydroxyl group of the members of this cluster is unlikely to undergo phase II metabolic activation. Instead, the beta-hydroxyl group is expected to contribute to detoxification via oxidation to hydrophilic acid. Despite structural similarity to carcinogenic ethyl benzene, only a marginal concern has been assigned to phenethyl alcohol due to limited mechanistic analogy.<br>For benzoates:<br>Acute toxicity: Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g., liver, kidney) were observed. However, with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol.<br>The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for<br>benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs<br>inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds.<br>Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate, but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate, but it is expected also to be only slightly irritating to the eye.<br>Sensitisation: The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally<br>very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact |

urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers.
Repeat dose toxicity: For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values
> 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed.
For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher
doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into
account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur.
Mutagenicity: All chemicals showed no mutagenic activity in in vitro Ames tests. Various results were obtained with other in vitro genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity in vivo. While some mixed and/or equivocal in vitro chromosomal/chromatid responses have been observed, no genotoxicity was observed in the in vivo cytogenetic, micronucleus, or other assays. The weight of the evidence of the in vitro and in vivo genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.
In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and
mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and
benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts.
Developmental toxicity: In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw).
For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses
(All by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1
mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g., increased mortality, reduced body weight and clinical toxicology.
Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed.
Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.
Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from
general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hay fever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to
ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the
mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via

the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.
Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and
benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.
Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of
the functional observational battery indicative of neurotoxicity in mice.
Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.
Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.
Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of
fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e., allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s)
in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the
acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic
contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or
hands. It may affect fitness for work and the quality of life of the individual.
Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g., by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention
of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention
(Avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.
Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand
eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema, perfumes are present in consumer products to which their hands are exposed. A significant relationship
between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact
allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe)
chronic hand eczema may not be clear.
Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.
Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an

eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.
Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g., citral are known.
Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this,
Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing.
This may be due to irritant effects or inadequate diagnostic procedures.
Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.
Pigmentary anomalies: The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that several fragrance ingredients were associated:
jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.
Photo-reactions Musk ambrette produced a considerable number of allergic photo contact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon.
Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for furocoumarins in fragrance products.
Phototoxic reactions still occur but are rare.
General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.
Fragrance allergens act as haptens, i.e., low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten is a chemical that itself is non- or low sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or as a prohapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.
Prohaptens Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.
In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation

processes increase the risk for cross-reactivity between fragrance substances. Cross reactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.
The human skin expresses enzyme systems that can metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point, they will be eliminated. However, many phase I products must undergo subsequent phase II transformations, i.e., conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.
QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well-established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha, beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g., it has been shown that autoxidation of the structural isomers' linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g., autoxidation) in relation to the metabolic activation
The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic).
This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
A member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption. metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances.
All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals.
The substances in this group:

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| | · Contain a benzene ring substituted with a reactive primary oxygenated functional group or can be hydrolysed to such a functional group<br>· The major pathway of metabolic detoxification involves hydrolysis and oxidation to yield the corresponding benzoic acid derivate which is excreted either as the free acid or the glycine conjugate<br>· They show a consistent pattern of toxicity in both short- and long- term studies and<br>· They exhibit no evidence of genotoxicity in standardised batteries of in vitro and in vivo assays.<br>The benzyl derivatives are rapidly absorbed through the gut, metabolised primarily in the liver, and excreted in the urine as glycine conjugates of benzoic acid derivatives.<br>In general, aromatic esters are hydrolysed in vivo through the catalytic activity of carboxylesterases, the most important of which are the A-esterases.<br>Hydrolysis of benzyl and benzoate esters to yield corresponding alcohols and carboxylic acids and hydrolysis of acetals to yield benzaldehyde and simple alcohols have been reported in several experiments.<br>The alcohols and aldehydes are rapidly oxidised to benzoic acid while benzoate esters are hydrolysed to benzoic acid.<br>Flavour and Extract Manufacturers Association (FEMA)<br>The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles.<br>The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity.<br>At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin.<br>The potential for eye irritation is minimal.<br>With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low.<br>NOAELs for maternal and developmental toxicity are far more than current human exposure levels.<br>No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity in vitro bacterial assays, and in vitro mammalian cell assays. All in vivo micronucleus assays were negative.<br>It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients.<br>The Research Institute for Fragrance Materials (RIFM) Expert Panel |
| TITANIUM DIOXIDE | * IUCLID<br>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man.<br>This concern is raised, generally, based on appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.<br>For titanium dioxide:<br>Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) Regarding inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral |

ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to
healthy skin of human volunteers revealed that titanium dioxide particles only penetrate the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.
Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function,
pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.
No data were available on genotoxic effects in titanium dioxide-exposed humans.
Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry
lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain.
Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols.
Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in
the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental
studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophagemediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium
dioxides are more slowly cleared than their fine counterparts.
Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury,
cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.
Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies
with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of
reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.
Animal carcinogenicity data
Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in
rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and
by intraperitoneal administration in male mice and female rats.
In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation
study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing
lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-

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| | dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.<br>Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.<br>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of<br>intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.<br>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may<br>produce conjunctivitis. |
| CARBON BLACK | Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported. |
| PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS | NOTE: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to<br>3000 ppm. Fetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.<br>for propylene glycol ethers (PGEs):<br>Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); Dipropylene glycol n-butyl ether (DPnB); Dipropylene glycol methyl ether acetate (DPMA); Tri propylene glycol methyl ether (TPM).<br>Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower<br>molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo<br>and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene<br>series, metabolism of the terminal hydroxyl group produces an alkoxy acetic acid. The reproductive and developmental toxicities<br>of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxy acetic and<br>ethoxy acetic acids.<br>Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxy acetic acid. The predominant alpha isomer of all the PGEs<br>(Thermodynamically favoured during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxy propionic acid. In<br>contrast beta-isomers can form the alkoxy propionic acids and these are linked to teratogenic effects (and possibly<br>haemolytic effects).<br>This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.<br>Because the alpha isomer cannot form an alkoxy propionic acid, this is the most likely reason for the lack of toxicity shown by the<br>PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test<br>data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or<br>Tri propylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of<br>any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the<br>primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the<br>body. |

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.
As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to non-irritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating,
None are skin sensitisers.
In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology).
LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).
Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB.
TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm).
In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.
One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity, the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two-generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.
In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported.

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| | Commercially available PGEs showed no teratogenicity.<br>The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. In vitro, negative results have<br>been seen in several assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic in vivo. In a 2-year bioassay on PM, there<br>were no statistically significant increases in tumours in rats and mice.<br>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to<br>irritants may produce conjunctivitis. |
| NAPHTHA PETROLEUM, ISOPARAFFIN, HYDROTREATED | Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the<br>absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the<br>carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo paraffins.<br>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In<br>many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon<br>absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and<br>asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products,<br>afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons<br>may traverse the mucosal epithelium unmetabolized and appear as solutes in lipoprotein particles in intestinal lymph, there is<br>evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte.<br>The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the<br>liver.<br>For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation.<br>Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans.<br>Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants).<br>Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as<br>lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the<br>foetus.<br>Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials.<br>Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable. |
| TRIDECANOL ETHOXYLATED, PHOSPHATED | Stephan SDS<br>for alkyl alcohol alkoxylate phosphate (AAAPD) surfactants (alkyl or alcohol ether phosphates): |

Acute toxicity: This group of surfactants exhibits similar effects to the alcohol ether sulphates (AAASDs) (typically sodium lauryl
ether sulphate - SLES - CAS RN 68891-38-3).
They are likely to be skin/ eye irritants (R36/38) in their undiluted forms but not acutely toxic. The reported oral LD50 values were higher than 1600 mg/kg for the alkyl ether phosphates family described by CAS RN: 9046-01-9. No effects were found at any concentration tested dermally.
Commercial products may contain excess phosphoric acid and may produce serious eye irritation (R41) or may even be classified as corrosive, acidic substances.
Subchronic toxicity: Data for sulphate derivatives has been identified in the public domain. Subchronic 21-day repeat dose
dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxymethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity.
No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2(CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day
repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than
approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy,
increased liver weight, and related increases in haematological endpoints related to liver enzyme induction.
SLES was evaluated for effects on the reproduction and prenatal/postnatal development of the rat when administered orally via
the drinking water through two successive generations. Based on this study an overall no-observed-adverse-effect level (NOAEL)
for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The
NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulphate derivatives.
Genotoxicity: Alcohol ether phosphates are unlikely to be genotoxic by analogy with their alcohol ether sulphate equivalents.
Carcinogenicity: Chronic dietary studies conducted with rats on sulphate derivatives showed no incidence of cancer and no
effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).]
Reproductive and developmental toxicity: Studies with sulphate derivatives showed little to no toxicity in dams or pups with the
NOEL in a developmental toxicity study in rats with SLES at the limit dose of 1000 mg/kg/day and a reproductive NOAEL of 0.3%
in drinking water (equivalent to 300 mg/kg/day), the highest dose tested in a two-generation reproduction study.
In studies with phosphate derivatives, the reproductive/ developmental NOAEL for an OECD 422 study with CAS 681340-47-2
was 800 mg/kg/day, the highest dose tested, and for CAS RN 78330-24-2 the NOEL was 200 mg/kg/day.
A NOAEL of 200 mg/kg/day was selected as the toxicological endpoint for he chronic risk assessment for phosphate derivatives by the US EPA.
Both alcohol ether sulphates and phosphates have been evaluated in acute, subchronic, developmental and reproductive studies capable of detecting effects on endocrine mediated events. The results of these studies did not give any indication of a
treatment-related effect on the oestrogen receptor or endocrine system.
Metabolic fate: For compounds of comparable C16 carbon chain, the metabolites of the lower molecular weight ethoxylated
(POE n=3) alcohol ether sulphate surfactants are readily absorbed and excreted primarily in the urine whereas the C16 surfactants
with increased ethoxylation (POE n=9) are poorly absorbed and excreted primarily in the faeces There was also no evidence of

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| | hydrolysis of the sulphate group from C16 POE n= 3 and C16 POE n=9 or of metabolism of the ethoxylate portion of the molecule.<br>With C11 POE n=3 and C12 POE n=3 metabolic studies in rats confirmed that the alkyl chain is extensively metabolised by betaor omega oxidation leaving the ethoxysulfate, which is excreted directly.<br>By analogy alcohol ether phosphate esters may initially undergo metabolism to generate the corresponding alkyl alcohol<br>alkoxylate and POE (or POE/POP - polyoxypropylene) phosphate glycol; the dephosphorylated metabolite should be hydrolysed to the POE (or POE/POP) polyalkoxylate glycols and linear branched saturated and unsaturated alkyl alcohol metabolites. The resultant alkyl alcohol metabolites would be oxidised in fatty acid oxidation pathways. The polyalkoxylate glycols may either be conjugated and excreted unchanged or hydrolysed/ oxidised to various degraded metabolites before bring conjugated and<br>excreted<br>Sensitising potential: Polyether's, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards<br>air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol<br>(Penta ethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is no sensitizing but that many of the<br>investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.<br>Based on the lower irritancy, non-ionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to<br>these compounds by patch testing.<br>for acid mists, aerosols, vapours<br>Data from assays for genotoxic activity in vitro suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of<br>the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric<br>epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events in vivo in the<br>respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH in vivo differ from exposures in vitro in that, in vivo, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than in vitro. |
| NONYLPHENOL<br>ETHOXYLATE, BRANCHED | For nonylphenol and its compounds:<br>Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens.<br>Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and<br>humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens.<br>Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor), Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogenous hormone for binding with the estrogen<br>receptors ERalpha and ERbeta.<br>Effects in pregnant women. |

Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These “low doses” ranged from 10-13-10-9 M, which is lower than what is generally found in the environment. Nonylphenol has also been shown to affect cytokine signalling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumour necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications.

Effects on metabolism

Nonylphenol has been shown to act as an obesity enhancing chemical or obesogenic, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behaviour by interfering with leptin signalling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behaviour. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signalling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells).

Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signalling in the liver of adult male rats.

Cancer

Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to

produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple
studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in
terms of oral and dermal toxicity.
Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin,
carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity
is thought to be substantially lower than that of nonylphenol ethoxylates.
Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the
ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (Penta ethylene
glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to
air.
Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is no sensitizing but that many of the
investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.
Based on the lower irritancy, non-ionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing
Overall, alcohol alkoxylates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers,
e.g., methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and
immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow.
Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005, US
EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol
butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.
Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the
chemicals with an average alkyl chain length C >=6 may also contain shorter alkyl chains C <6. It is not practical to quantify the
proportion of shorter C <6 chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The
available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa).
therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.
Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:
EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41
EO > 15-20 gives Harmful (Xn) with R22-41
>20 EO is not classified (CESIO 2000)
Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).
AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC.
In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the
gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO2). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO2)). The metabolism of
C12 AE yields PEG, carboxylic acids, and CO2 as metabolites. The LD50 values after oral administration to rats range from
about 1-15 g/kg body weight indicating a low to moderate acute toxicity.
The ability of non-ionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling
mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the
alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of
the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Non-ionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by non-ionic surfactants, and proteins with poor solubility are not solubilized by non-ionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro
demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic, or carcinogenic. No adverse
reproductive or developmental effects were observed. Most available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes except for liver hypertrophy (indicative of an adaptive response to
metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of
90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into
account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Considering the conservatism in the
exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.
AEs are not contact sensitisers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs
depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products
are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of
the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.
In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause

concern regarding consumer use. For high boiling ethylene glycol ethers (typically Tri ethylene- and tetra ethylene glycol ethers):
Skin absorption: Available skin absorption data for Tri ethylene glycol ether (TGBE), Tri ethylene glycol methyl ether (TGME), and
Tri ethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm2/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The
rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/ cm2/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a
decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the
diethylene glycol series is larger than that of the diethylene glycol to Tri ethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on
absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetra ethylene glycol methyl; ether (TetraME) and tetra ethylene glycol butyl ether (Tetra BE) are expected to be less permeable to skin than TGME and TGBE,
the differences in permeation between these molecules may only be slight.
Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is
oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected in vivo. The principal metabolite of TGME
is believed to be 2-[2-(2-methoxyethoxy) ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of
glycol ethers.
The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur
Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure.
Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and
heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and
piloerection before death.
Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.
Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity.
In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema
were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one
rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypo spermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes
in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs
for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.
A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study,

significantly increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500
mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or
haemolysed blood in the stomach These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats.
In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and
was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but
no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity.
Mutagenicity: Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.
Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed,
several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower
molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition,
results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater that the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).
Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.
for nonylphenol:
Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day.
Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of

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| | the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.<br>Nonylphenol was not mutagenic to Salmonella typhimurium, TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA, with or without an exogeneous metabolic activation system.<br>Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system. |
| BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & BENZYL ALCOHOL | The following information refers to contact allergens as a group and may not be specific to this product.<br>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g., contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. |
| TITANIUM DIOXIDE & PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS & TRIDECANOL ETHOXYLATED, PHOSPHATED | 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.<br>The disorder is characterized by difficulty breathing, cough and mucus production. |
| TITANIUM DIOXIDE & CARBON BLACK & PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS & NAPHTHA PETROLEUM, ISOPARAFFIN, HYDROTREATED & MANGANESE DIOXIDE | No significant acute toxicological data identified in literature search. |
| TITANIUM DIOXIDE & PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS | The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic).<br>This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. |

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| TITANIUM DIOXIDE & CARBON BLACK | WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans |
| TRIDECANOL ETHOXYLATED, PHOSPHATED & NONYLPHENOL ETHOXYLATE, BRANCHED | Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is no sensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture. Based on the lower irritancy, non-ionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69 Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners. PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxymethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalysed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalysed by magnesium-, aluminium-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used. Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology http://doi.org/10.5487/TR.2015.31.2.105 |

| | | | |
|---|---|---|---|
| Acute Toxicity | / | Carcinogenicity | / |
| Skin Irritation/Corrosion | / | Reproductivity | X |
| Serious Eye Damage/Irritation | / | STOT - Single Exposure | / |
| Respiratory or Skin sensitisation | / | STOT - Repeated Exposure | X |

| | | | |
|---|---|---|---|
| Mutagenicity | / | Aspiration Hazard | / |

Legend X - Data either not available or does not fill the criteria for classification
/ - **Data** available to make classification

## SECTION 12 ECOLOGICAL INFORMATION

**Toxicity**

| | | | | | |
|---|---|---|---|---|---|
| AFS SC100 Epoxy Tint | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | Not Available | Not Available | Not Available | Not Available | Not Available |
| bisphenol A/ diglycidyl ether resin, liquid | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | EC50(ECx | 24h | Crustacea | 3mg/l | Not Available |
| | LC50 | 96h | Fish | 2.4mg/l | Not Available |
| | EC50 | 48h | Crustacea | ~2mg/l | 2 |
| benzyl alcohol | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | LC50 | 96h | Fish | 10mg/l | 4 |
| | EC50 | 72h | Algae or other aquatic plants | 500mg/l | 2 |
| | EC50 | 48h | Crustacea | 230mg/l | 2 |
| | NOEC(ECx) | 336h | Fish | 5.1mg/l | 2 |
| | EC50 | 96h | Algae or other aquatic plants | 76.828mg/l | 2 |
| titanium dioxide | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | BCF | 1008h | Fish | <1.1-9.6 | 7 |
| | LC50 | 96h | Fish | 1.85-3.06mg/l | 4 |
| | EC50 | 72h | Algae or other aquatic plants | 3.75-7.58mg/l | 4 |
| | EC50 | 48h | Crustacea | 1.9mg/l | 2 |
| | EC50 | 96h | Algae or other aquatic plants | 179.05mg/l | 2 |
| | NOEC(ECx) | 504h | Crustacea | 0.02mg/l | 4 |
| carbon black | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | LC50 | 96h | Fish | >100mg/l | 2 |
| | EC50 | 72h | Algae or other aquatic plants | >0.2mg/l | 2 |
| | EC50 | 48h | Crustacea | 33.076-41.968mg/l | 4 |
| | NOEC(ECx) | 24h | Crustacea | 3200mg/l | 1 |
| propylene glycol monomethyl ether - mixture of isomers | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | LC50 | 96h | Fish | 100mg/l | 1 |
| | EC50 | 72h | Algae or other aquatic plants | >1000mg/l | 2 |
| | EC50 | 48h | Crustacea | 373mg/l | 2 |
| | NOEC(ECx) | 336h | Fish | 47.5mg/l | 2 |
| | EC50 | 96h | Algae or other aquatic plants | >1000mg/l | 2 |
| naphtha petroleum, isoparaffin, hydrotreated | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | EC50(ECx) | 48h | Crustacea | >0.002mg/l | 2 |
| | EC50 | 96h | Algae or other aquatic plants | 64mg/l | 2 |
| | EC50 | 48h | Crustacea | >0.002mg/l | 2 |
| manganese dioxide | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | NOEC(ECx) | 1560h | Fish | 0.55mg/l | 2 |
| tridecanol ethoxylated, phosphated | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | Not | Not | Not | Not | Not |

| | Available | Available | Available | Available | Available |
|---|---|---|---|---|---|
| nonylphenol ethoxylate, branched | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | NOEC(ECx) | 2184h | Fish | 0.006mg/l | 2 |
| | LC50 | 96h | Fish | 0.136mg/l | 2 |
| | EC50 | 72h | Algae or other aquatic plants | >3mg/l | 2 |
| Legend | Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) -Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data | | | | |

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

**Persistence and degradability**

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | HIGH | HIGH |
| benzyl alcohol | LOW | LOW |
| titanium dioxide | HIGH | HIGH |
| propylene glycol monomethyl ether - mixture of isomers | LOW (Half-life = 56 days) | LOW (Half-life = 1.7 days) |

**Bioaccumulative potential**

| Ingredient | Bioaccumulation |
|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | OW (LogKOW = 2.6835) |
| benzyl alcohol | LOW (LogKOW = 1.1) |
| titanium dioxide | LOW (BCF = 10) |
| propylene glycol monomethyl ether - mixture of isomers | LOW (BCF = 2) |

**Mobility in soil**

| Ingredient | Mobility |
|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | LOW (KOC = 51.43) |
| benzyl alcohol | LOW (KOC = 15.66) |
| titanium dioxide | LOW (KOC = 23.74) |
| propylene glycol monomethyl ether - mixture of isomers | HIGH (KOC = 1) |

## 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 cannot 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) |

| | |
|---|---|
| | 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 |  |
| HAZCHEM | •3Z |
| **Land transport (ADG)** | |
| UN number or ID number | 3082 |
| UN proper shipping name | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid) |
| Transport hazard class(es) | Class \| 9<br>Subsidiary Risk \| Not applicable |
| Packing group | III |
| Environmental hazard | Environmentally hazardous |
| Special precautions for User | Special provisions \| 274 331 335 375 AU01<br>Limited quantity \| 5L |

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

| Air transport (ICAO-IATA / DGR) | |
|---|---|
| UN number | 3082 |
| UN proper shipping name | Environmentally hazardous substance, liquid, n.o.s. (contains bisphenol A/ diglycidyl ether resin, liquid) |
| Transport hazard class(es) | ICAO/IATA Class \| 9<br>ICAO / IATA Subrisk \| Not Applicable<br>ERG Code \| 9L |

| | | |
|---|---|---|
| Packing group | III | |
| Environmental hazard | Environmentally hazardous | |
| Special precautions for User | Special provisions | A97 A158 A197 A215 |
| | Cargo Only Packing Instructions | 964 |
| | Cargo Only Maximum Qty / Pack | 450 L |
| | Passenger and Cargo Packing Instructions | 964 |
| | Passenger and Cargo Maximum Qty / Pack | 450 L |
| | Passenger and Cargo Limited Quantity Packing Instructions | Y964 |
| | Passenger and Cargo Limited Maximum Qty / Pack | 30 kg G |

**Sea transport (IMDG-Code / GGVSee)**

| | | |
|---|---|---|
| UN number | 3082 | |
| UN proper shipping name | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains bisphenol A/ diglycidyl ether resin, liquid) | |
| Transport hazard class(es) | IMDG Class | 9 |
| | IMDG Subrisk | Not Applicable |
| Packing group | III | |
| Environmental hazard | Marine Pollutant | |
| Special precautions for user | EMS Number | F-A, S-F |
| | Special provisions | 274 335 969 |
| | Limited Quantities | 5 L |

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

Not Applicable

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

| Product name | Group |
|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | Not Available |
| benzyl alcohol | Not Available |
| titanium dioxide | Not Available |
| carbon black | Not Available |
| propylene glycol monomethyl ether - mixture of isomers | Not Available |
| naphtha petroleum, isoparaffin, hydrotreated | Not Available |
| manganese dioxide | Not Available |
| tridecanol ethoxylated, phosphate | Not Available |
| nonylphenol ethoxylate, branched | Not Available |

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

| Product name | Ship Type |
|---|---|
| bisphenol A/ diglycidyl ether resin, liquid | Not Available |
| benzyl alcohol | Not Available |
| titanium dioxide | Not Available |
| carbon black | Not Available |
| propylene glycol monomethyl ether - mixture of isomers | Not Available |
| naphtha petroleum, isoparaffin, hydrotreated | Not Available |
| manganese dioxide | Not Available |
| tridecanol ethoxylated, phosphate | Not Available |
| nonylphenol ethoxylate, | Not Available |

branched

## SECTION 15 REGULATORY INFORMATION

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

**bisphenol A/ diglycidyl ether resin, liquid is found on the following regulatory lists**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL)
Values for Manufactured Nanomaterials (MNMS)

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

Australian Inventory of Industrial Chemicals (AIIC)

**titanium dioxide is found on the following regulatory lists**

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 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL)
Values for Manufactured Nanomaterials (MNMS)

**carbon black is found on the following regulatory lists**

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 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL)
Values for Manufactured Nanomaterials (MNMS)

**propylene glycol monomethyl ether - mixture of isomers is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

**naphtha petroleum, isoparaffin, hydrotreated is found on the following regulatory lists**

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 - Not Classified as Carcinogenic

**manganese dioxide 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)

**tridecanol ethoxylated, phosphate is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**nonylphenol ethoxylate, branched is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

**National Inventory Status**

| National Inventory | Status |
|---|---|
| Australia - AIIC / Australia Non-Industrial Use | Yes |
| Canada – DSL | Yes |
| Canada – NDSL | No (bisphenol A/ diglycidyl ether resin, liquid; benzyl alcohol; carbon black; naphtha petroleum, isoparaffin, hydrotreated; manganese dioxide; tridecanol ethoxylated, phosphate; nonylphenol ethoxylate, branched) |
| China - IECSC | Yes |
| Europe - EINEC / ELINCS / NLP | No (tridecanol ethoxylated, phosphate) |
| Japan – ENCS | Yes |
| Korea – KECI | Yes |
| New Zealand – NZIoC | Yes |
| Philippines – PICCS | Yes |

| | |
|---|---|
| USA – TSCA | Yes |
| Taiwan – TCSI | Yes |
| Mexico – INSQ | No (tridecanol ethoxylated, phosphated) |
| Vietnam – NCI | Yes |
| Russia – FBEPH | Yes |
| 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 | 22/09/23 |
| Initial Date | 22/09/23 |

### Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch.
Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

### Definitions and abbreviations

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

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