

# POSTER PAINT TP31 Primary Color

## Jasco Pty Limited

Chemwatch Hazard Alert Code: 3

Chemwatch: 5671-47

Issue Date: 28/03/2024

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Print Date: 04/04/2024

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

L.GHS.AUS.EN

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

#### Product Identifier

<b>Product name</b>	POSTER PAINT TP31 Primary Color
<b>Chemical Name</b>	Not Applicable
<b>Synonyms</b>	0123130 SMUDGI POSTER PAINT 500ML WHT; 0123140 SMUDGI POSTER PAINT 500ML YELLOW; 0123150 SMUDGI POSTER PAINT 500ML ORANGE; 0123160 SMUDGI POSTER PAINT 500ML RED; 0123170 SMUDGI POSTER PAINT 500ML PINK; 0123180 SMUDGI POSTER PAINT 500ML PURPLE; 0123190 SMUDGI POSTER PAINT 500ML LIGHT BLUE; 0123210 SMUDGI POSTER PAINT 500ML DARK BLUE; 0123220 SMUDGI POSTER PAINT 500ML GREEN; 0123230 SMUDGI POSTER PAINT 500ML BLK
<b>Chemical formula</b>	Not Applicable
<b>Other means of identification</b>	Not Available

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

<b>Relevant identified uses</b>	Used for painting. Use according to manufacturer's directions.
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#### Details of the manufacturer or supplier of the safety data sheet

<b>Registered company name</b>	Jasco Pty Limited
<b>Address</b>	1-5 Commercial Road Kingsgrove NSW 2208 Australia
<b>Telephone</b>	+61 2 9807 1555
<b>Fax</b>	Not Available
<b>Website</b>	<a href="http://www.jasco.com.au">www.jasco.com.au</a>
<b>Email</b>	quickinfo@jasco.com.au

#### Emergency telephone number

<b>Association / Organisation</b>	Australian Poisons Centre	CHEMWATCH EMERGENCY RESPONSE (24/7)
<b>Emergency telephone numbers</b>	13 11 26 (24/7)	+61 1800 951 288
<b>Other emergency telephone numbers</b>	Not Available	+61 3 9573 3188


Once connected and if the message is not in your preferred language then please dial 01

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

<b>Poisons Schedule</b>	Not Applicable
<b>Classification <sup>[1]</sup></b>	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Germ Cell Mutagenicity Category 2, Carcinogenicity Category 1A, Hazardous to the Aquatic Environment Acute Hazard Category 3
<b>Legend:</b>	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)	
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Signal word	<b>Danger</b>
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**Hazard statement(s)**

H315	Causes skin irritation.
H318	Causes serious eye damage.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H402	Harmful to aquatic life.

**Precautionary statement(s) Prevention**

P201	Obtain special instructions before use.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

**Precautionary statement(s) Response**

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

**Precautionary statement(s) Storage**

P405	Store locked up.
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**Precautionary statement(s) Disposal**

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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**SECTION 3 Composition / information on ingredients****Substances**

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
471-34-1	4.2	<u>calcium carbonate</u>
25212-88-8	4	<u>methacrylic acid/ ethyl acrylate copolymer</u>
57-55-6	3.5	<u>propylene glycol</u>
124-68-5	0.4	<u>monoisobutanolamine</u>
9004-62-0	0.5	<u>hydroxyethylcellulose</u>
8020-83-5	0.3	<u>hydrocarbon oils</u>
52-51-7	0.05	<u>2-bromo-2-nitropropan-1,3-diol</u>
13463-67-7	0-2.25	<u>titanium dioxide</u>
1328-53-6	0-2.25	<u>C.I. Pigment Green 7</u>
2512-29-0	0-2.25	<u>C.I. Pigment Yellow 1</u>
6486-23-3	0-2.25	<u>C.I. Pigment Yellow 3</u>
1333-86-4	0-2.25	<u>C.I. Pigment Black 7</u>
6410-30-6	0-2.25	<u>C.I. Pigment Red 8</u>
147-14-8	0-2.25	<u>C.I. Pigment Blue 15</u>
6358-30-1	0-2.25	<u>C.I. Pigment Violet 23</u>

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CAS No	%[weight]	Name
6410-26-0	0-2.25	<u>C.I. Pigment Red 21</u>
3520-72-7	0-2.25	<u>C.I. Pigment Orange 13</u>
7732-18-5	84.8	<u>water</u>

**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

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Immediately hold eyelids apart and flush the eye continuously with running water.</li> <li>▶ 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.</li> <li>▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> </ul>

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

Treat symptomatically.

## SECTION 5 Firefighting measures

### Extinguishing media

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

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

In such an event consider:

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

### Special hazards arising from the substrate or mixture

<b>Fire Incompatibility</b>	None known.
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### Advice for firefighters

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves in the event of a fire.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water courses.</li> <li>▶ Use fire fighting procedures suitable for surrounding area.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Non combustible.</li> <li>▶ Not considered to be a significant fire risk.</li> <li>▶ Expansion or decomposition on heating may lead to violent rupture of containers.</li> </ul>

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	<ul style="list-style-type: none"> <li>▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).</li> <li>▶ May emit acrid smoke.</li> </ul> <p>carbon dioxide (CO<sub>2</sub>) hydrogen chloride phosgene nitrogen oxides (NO<sub>x</sub>) metal oxides other pyrolysis products typical of burning organic material.</p> <p>May emit poisonous fumes. May emit corrosive fumes.</p> <p><b>CARE:</b> Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.</p>
<b>HAZCHEM</b>	Not Applicable

### SECTION 6 Accidental release measures

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

See section 8

#### Environmental precautions

See section 12

#### Methods and material for containment and cleaning up

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

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

### SECTION 7 Handling and storage

#### Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ <b>DO NOT allow material to contact humans, exposed food or food utensils.</b></li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> </ul>

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- ▶ Store in a cool, dry, well-ventilated area.
- ▶ Store away from incompatible materials and foodstuff containers.
- ▶ Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

### Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Polyethylene or polypropylene container.</li> <li>▶ Packing as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
<b>Storage incompatibility</b>	None known

## 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	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	propylene glycol	Propane-1,2-diol total: (vapour & particulates)	150 ppm / 474 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol: particulates only	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	hydrocarbon oils	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
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	C.I. Pigment Black 7	Carbon black	3 mg/m3	Not Available	Not Available	Not Available

#### Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
calcium carbonate	45 mg/m3	210 mg/m3	1,300 mg/m3
propylene glycol	30 mg/m3	1,300 mg/m3	7,900 mg/m3
monoisobutanolamine	17 mg/m3	190 mg/m3	570 mg/m3
hydrocarbon oils	140 mg/m3	1,500 mg/m3	8,900 mg/m3
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3
C.I. Pigment Black 7	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
calcium carbonate	Not Available	Not Available
methacrylic acid/ ethyl acrylate copolymer	Not Available	Not Available
propylene glycol	Not Available	Not Available
monoisobutanolamine	Not Available	Not Available
hydroxyethylcellulose	Not Available	Not Available
hydrocarbon oils	2,500 mg/m3	Not Available
2-bromo-2-nitropropan-1,3-diol	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
C.I. Pigment Green 7	Not Available	Not Available
C.I. Pigment Yellow 1	Not Available	Not Available
C.I. Pigment Yellow 3	Not Available	Not Available
C.I. Pigment Black 7	1,750 mg/m3	Not Available
C.I. Pigment Red 8	Not Available	Not Available
C.I. Pigment Blue 15	Not Available	Not Available
C.I. Pigment Violet 23	Not Available	Not Available
C.I. Pigment Red 21	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
C.I. Pigment Orange 13	Not Available	Not Available
water	Not Available	Not Available

**Occupational Exposure Banding**

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
monoisobutanolamine	E	≤ 0.01 mg/m <sup>3</sup>
2-bromo-2-nitropropan-1,3-diol	E	≤ 0.01 mg/m <sup>3</sup>
C.I. Pigment Yellow 1	E	≤ 0.01 mg/m <sup>3</sup>
C.I. Pigment Yellow 3	E	≤ 0.01 mg/m <sup>3</sup>
C.I. Pigment Red 8	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m <sup>3</sup> )
C.I. Pigment Red 21	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m <sup>3</sup> )
C.I. Pigment Orange 13	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m <sup>3</sup> )

**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**

for 3,3'-dichlorobenzidine (DCB):

Various tumours developed after oral or subcutaneous administration of DCB to mice, rats, hamsters and dogs. Tumours have not yet been identified in persons exposed to the substance alone. The substance can be absorbed through the skin in dangerous quantities. Increases in temperature and relative humidity promote dermal absorption.

Toxicity and Irritation data for petroleum-based mineral oils are related to chemical components and vary as does the composition and source of the original crude. A small but definite risk of occupational skin cancer occurs in workers exposed to persistent skin contamination by oils over a period of years. This risk has been attributed to the presence of certain polycyclic aromatic hydrocarbons (PAH) (typified by benz[a]pyrene).

Petroleum oils which are solvent refined/extracted or severely hydrotreated, contain very low concentrations of both.

for mineral oils (excluding metal working fluids), pure, highly and severely refined:

Human exposure to oil mist alone has not been demonstrated to cause health effects except at levels above 5 mg/m<sup>3</sup> (this applies to particulates sampled by a method that does not collect vapour). It is not advisable to apply this standard to oils containing unknown concentrations and types of additive.

For calcium carbonate:

The TLV-TWA is thought to be protective against the significant risk of physical irritation associated with exposure.

Animals exposed by inhalation to 10 mg/m<sup>3</sup> titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

- The label on a package containing 1% or more of titanium oxide with aerodynamic diameter equal or below 10 microns shall bear the following statement: EUH211 "Warning! Hazardous respirable droplets may be formed when sprayed. Do NOT breathe spray or mist"

- The label on the packaging of solid mixtures containing 1% or more of titanium dioxide shall bear the following statement: EUH212 "Warning! Hazardous respirable dust may be formed when used. Do not breathe dust".


In addition, the label on the packaging of liquid and solid mixtures not intended for the general public and not classified as hazardous which are labelled EUH211 or EU212 shall bear statement EUH210: "Safety data sheet available on request."

Cellulose is considered a nuisance dust which has little adverse effect on lung and does not produce significant organic disease or toxic effects when appropriate controls are applied.

**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.						
	<table border="1" style="width: 100%;"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> </tbody> </table>	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.)
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.)						

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	<p>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</p> <p>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</p> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	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	<p>1-2.5 m/s (200-500 f/min.)</p> <p>2.5-10 m/s (500-2000 f/min.)</p>
Lower end of the range	Upper end of the range											
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents											
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3: Intermittent, low production.	3: High production, heavy use											
4: Large hood or large air mass in motion	4: Small hood-local control only											
<b>Individual protection measures, such as personal protective equipment</b>												
<b>Eye and face protection</b>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].</li> </ul>											
<b>Skin protection</b>	See Hand protection below											
<b>Hands/feet protection</b>	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul>											
<b>Body protection</b>	See Other protection below											
<b>Other protection</b>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eye wash unit.</li> </ul>											

**Recommended material(s)****GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

**"Forsberg Clothing Performance Index".**

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
BUTYL	C
NATURAL RUBBER	C
NEOPRENE	C
PE/EVAL/PE	C
PVA	C
VITON	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

**Respiratory protection**

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

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

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

Continued...

\* 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.

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

<b>Appearance</b>	Pasty.		
<b>Physical state</b>	Free-flowing Paste	<b>Relative density (Water = 1)</b>	Not Available
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Available	<b>Decomposition temperature (°C)</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	Not Applicable	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Applicable	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Not Available	<b>pH as a solution (1%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available

## SECTION 10 Stability and reactivity

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	Product is considered stable and hazardous polymerisation will not occur.
<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 Toxicological information

### Information on toxicological effects

<b>Inhaled</b>	<p>The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation, of the material, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.</p> <p>Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p>
<b>Ingestion</b>	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The</p>



vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products.]

Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain its availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively "inactivates" the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper.

Ingestion of propylene glycol produced reversible central nervous system depression in humans following ingestion of 60 ml. Symptoms included increased heart-rate (tachycardia), excessive sweating (diaphoresis) and grand mal seizures in a 15 month child who ingested large doses (7.5 ml/day for 8 days) as an ingredient of vitamin preparation.

Excessive repeated ingestions may cause hypoglycaemia (low levels of glucose in the blood stream) among susceptible individuals; this may result in muscular weakness, incoordination and mental confusion.

Very high doses given during feeding studies to rats and dogs produce central nervous system depression (although one-third of that produced by ethanol), haemolysis and insignificant kidney changes.

In humans propylene glycol is partly excreted unchanged in the urine and partly metabolised as lactic and pyruvic acid. Lactic acidosis may result.

**Skin Contact**

Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

The material may accentuate any pre-existing dermatitis condition

Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.

Open cuts, abraded or irritated skin should not be exposed to this material

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.

Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature.

**Eye**

When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.

Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea.

**Chronic**

On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans.

Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.

Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.

Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. 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.

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

There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

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.

Chronic poisoning from ionic bromides has historically resulted from medical use of bromides but not from exposure in the environment or workplace. In the absence of other signs of poisoning, there may be depression, hallucinations and schizophrenia-like psychosis. Bromides may also cause sedation, irritability, agitation, delirium, memory loss, confusion, disorientation, forgetfulness, inability to speak, difficulty speaking, weakness, fatigue, a spinning sensation, stupor, coma,

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decreased appetite, nausea, vomiting, an acne-like rash on the face (bronchoderma), legs and trunk, swelling of the bronchi and a profuse discharge from the nostrils. There may also be inco-ordination and very brisk reflexes. Correlation of nervous system symptoms with blood levels of bromide is inexact. Current day usage of bromides is generally limited to antihistamines such as brompheniramine, which is a covalent compound; ionic compounds are no longer regularly used due to their toxicity. In test animals, brominated vegetable oils (BVOs), historically used as emulsifiers in certain soda-based soft drinks, produced damage to the heart and kidneys in addition to increasing fat deposits in these organs. In extreme cases, BVOs caused testicular damage, stunted growth and produced lethargy and fatigue.

Brominism (chronic bromine poisoning) produces slurred speech, apathy, headache, decreased memory, anorexia and drowsiness, psychosis resembling paranoid schizophrenia, and personality changes.

Several cases of foetal abnormalities have been described in mothers who took large doses of bromides during pregnancy. Reproductive effects caused by bromide (which crosses the placenta) include central nervous system depression, brominism, and bronchoderma (an acne-like rash) in the newborn.

POSTER PAINT TP31 Primary Color	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
calcium carbonate	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 0.75 mg/24h - SEVERE
	Inhalation (Rat) LC50: >3 mg/l4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
methacrylic acid/ ethyl acrylate copolymer	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
propylene glycol	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 11890 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg - mild
	Inhalation (Rat) LC50: >44.9 mg/l4h <sup>[1]</sup>	Eye (rabbit): 500 mg/24h - mild
	Oral (Rat) LD50: 20000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin(human):104 mg/3d Intermit Mod
		Skin(human):500 mg/7days mild
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
monoisobutanolamine	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup>	Not Available
	Oral (Mouse) LD50; 2150 mg/kg <sup>[2]</sup>	
hydroxyethylcellulose	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
hydrocarbon oils	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >5000 mg/kg <sup>[2]</sup>	Eye (rabbit) 100 mg/24H mild
	Oral (Rabbit) LD50; 2835 mg/kg <sup>[2]</sup>	Skin (rabbit) 500 mg/24H mild
2-bromo-2-nitropropan-1,3- diol	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: ~1600 mg/kg <sup>[1]</sup>	Eye (rabbit): 5 mg
	Inhalation (Rat) LC50: >0.12<1.14 mg/l4h <sup>[1]</sup>	Skin (human): 10 mg moderate
	Oral (Rat) LD50: 180 mg/kg <sup>[2]</sup>	Skin (rabbit): 500 mg/24h mild
		Skin (rabbit): 80 mg moderate
titanium dioxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (hamster) LD50: >=10000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation (Rat) LC50: >2.28 mg/l4h <sup>[1]</sup>	Skin (human): 0.3 mg /3D (int)-mild *
	Oral (Rat) LD50: >=2000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
C.I. Pigment Green 7	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation (Rat) LC50: >1.084<5.212 mg/l4h <sup>[1]</sup>	Not Available
	Oral (Mouse) LD50; 8400 mg/kg <sup>[2]</sup>	

Continued...

C.I. Pigment Yellow 1	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Oral (Rat) LD50: >5000 mg/kg <sup>[2]</sup>	Non-irritating/non-sensitising [Dominion]
C.I. Pigment Yellow 3	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Oral (Rat) LD50: >10000 mg/kg <sup>[2]</sup>	Not Available
C.I. Pigment Black 7	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup> Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
C.I. Pigment Red 8	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >5000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
C.I. Pigment Blue 15	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Inhalation (Rat) LC50: >1.084<5.212 mg/l4h <sup>[1]</sup> Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (human): non-irritant [Manuf. C.G.] Skin (human): non-irritant
C.I. Pigment Violet 23	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: 5000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin (rabbit): Non-irritating * * [Ravenswood] Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
C.I. Pigment Red 21	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
C.I. Pigment Orange 13	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Oral (Rat) LD50: >10000 mg/kg <sup>[2]</sup>	Not Available
water	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >90000 mg/kg <sup>[2]</sup>	Not Available
<b>Legend:</b>	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	

<b>CALCIUM CARBONATE</b>	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
<b>MONOISOBUTANOLAMINE</b>	For tris(hydroxymethyl)aminomethane (TRIS AMINO; CAS 77-88-1) and its surrogates 2-amino-2-methyl-1,3-propanediol (AMPD; CAS 115-69-5) and monoisobutanolamine (AMP; CAS 124-68-5) TRIS AMINO and the surrogate chemicals have displayed little if any toxicity to humans during their long history of use as human drugs and/or in personal care products and cosmetics. TRIS AMINO has found use as an IV drug for the management of acidosis in humans for many years and the toxicity of AMPD and AMP have been reviewed by the Cosmetic Ingredient Review Expert Panel which concluded that these materials are safe as used in cosmetic formulations up to 1% <b>Acute toxicity:</b> Mammalian toxicity studies have displayed similar results. The oral LD50 value for TRIS AMINO is 5500 mg/kg in the mouse, and its surrogates range from 2150 to greater than 5000 mg/kg in the rat and mouse. TRIS AMINO was non-irritating to eyes when a 40% aqueous solution was applied to the eyes of rabbits (pH 10.4 for 0.1M aqueous solution). In contrast, 95% AMP in water was severely irritating to the eyes, presumably due to the severely alkaline pH of the test solution used (pH 11.3 for 0.1M aqueous solution); however, more neutral cosmetic formulations containing lower concentrations of AMP are only minimally irritating. There is no sensitisation data available for TRIS AMINO; however, based on the following data, TRIS AMINO is not expected to be a sensitiser. Laboratory animal test samples of AMP did not cause allergic skin reactions when tested in guinea pigs following topical or intradermal administration. In patch tests with humans, AMP and cosmetic formulations containing either AMP or AMPD were negative for dermal sensitisation. <b>Repeated dose toxicity:</b> Repeated-dose mammalian toxicity studies conducted on TRIS AMINO and the two surrogate chemicals indicate that the compounds are generally well-tolerated at concentrations as high as 500 mg/kg/day via IV infusion

	<p>for TRIS AMINO and ingestion of up to 3200 ppm in the rodent diet (250-750 mg/kg/day for rats and mice, estimated). A number of human clinical trials of the IV infusion of TRIS AMINO have also been successfully conducted. In all studies, the only target tissue, when observed at all, has been the liver with AMP. Human clinical studies with Keterolac (a major component of which is TRIS AMINO) have suggested that patients with decreased liver function not be given the drug over extended treatment periods based upon changes in several clinical chemistry parameters. Ingestion of relatively high dosages of AMP has caused liver histopathological changes in rats and dogs. The most significant toxicological activity has been a foetotoxic effect of AMP when ingested at relatively high levels by pregnant rats. Subsequent dermal exposure to comparable dosages failed to elicit a developmental effect in rats. Overall, there have been no consistently-noted observations or treatment-related findings among the numerous repeated-dose mammalian toxicity studies that have been conducted over at last 50 years on these compounds that would indicate long-term significant toxicity of either compound at typical human exposure levels. Reflective of these findings is the fact that both TRIS AMINO and AMP display similar patterns of excretion from the body, being primarily eliminated unchanged via the urine over a relatively short period of time. Further, no evidence of either direct reactivity or metabolism to reactive species toward genetic material has been observed. <b>Genetic toxicity:</b> Studies conducted on the TRIS AMINO and the surrogate substances in the presence or absence of mammalian metabolic enzymes have all been negative.</p>
<b>HYDROCARBON OILS</b>	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<b>2-BROMO-2-NITROPROPAN-1,3-DIOL</b>	<p>The European Union has reclassified several formaldehyde-releasing agents (FRAs) such as methylenedimorpholine (MBM), oxazolidine (MBO) and hydroxypropylamine (HPT) as category 1B carcinogens. Previously, formaldehyde itself was classed as a carcinogen – but formaldehyde-releasing agents were not. This is no longer the case. Based on this regulation, formulations for which the maximum theoretical concentration of releasable formaldehyde is more than &gt; 1000 ppm (&gt;0.1%), have to be labelled as carcinogenic.</p> <p>Water mix metalworking fluids are subject to contamination by bacteria and fungi, and the control of this is an essential part of good fluid maintenance. The use of preservatives both within the formulation and tank-side treatment plays a significant contribution in the protection of potentially harmful microbes that could cause health problems for workers.</p> <p>A large proportion of bactericides on the market today are classed as formaldehyde releasing biocides which means that under specific conditions they release small amounts of formaldehyde – this is their mode of action in the presence of bacteria. Although they are effective as a biocide their use may become restricted or unfavourable due to potential changes in legislation.</p> <p>A decision by the ECHA (European Chemicals Agency) was made to re-classify formaldehyde as a category 1b H350 carcinogen and category 2 mutagen in June 2015.</p> <p>It has also been proposed by the ECHA Risk Assessment Committee (RAC) that formaldehyde release biocides should be classified the same as formaldehyde because formaldehyde is released when these substances come into contact under favorable conditions (i.e. interaction with microorganisms).</p> <p>Formaldehyde generators (releasers) are often used as preservatives (antimicrobials, biocides, microbiocides). Formaldehyde may be generated following hydrolysis. The most widely used antimicrobial compounds function by releasing formaldehyde once inside the microbe cell. Some release detectable levels of formaldehyde into the air space, above working solutions, especially when pH has dropped.</p> <p>Many countries are placing regulatory pressure on suppliers and users to replace formaldehyde generators.</p> <p>Formaldehyde generators are a diverse group of chemicals that can be recognised by a small, easily detachable formaldehyde moiety, prepared by reacting an amino alcohol with formaldehyde ("formaldehyde-condensates"), There is concern that when formaldehyde-releasing preservatives are present in a formulation that also includes amines, such as triethanolamine (TEA), diethanolamine (DEA), or monoethanolamine (MEA), nitrosamines can be formed; nitrosamines are carcinogenic substances that can potentially penetrate skin.</p> <p>One widely-discussed hypothesis states that formaldehyde-condensate biocides, such as triazines and oxazolidines, may cause an imbalance in the microbial flora of in-use metalworking fluids (MWFs). The hypothesis further asserts that this putative microbial imbalance favours the proliferation of certain nontuberculosis mycobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, in a small percentage of susceptible workers. Symptoms of HP include flu-like illness accompanied by chronic dyspnea, i.e., difficult or laboured respiration</p> <p>According to Annex VI of the Cosmetic Directive 76/768/EC, the maximum authorised concentration of free formaldehyde is 0.2% (2000 ppm). In addition, the provisions of Annex VI state that, <i>All finished products containing formaldehyde or substances in this Annex and which release formaldehyde must be labelled with the warning "contains formaldehyde" where the concentration of formaldehyde in the finished product exceeds 0.05%.</i> Formaldehyde-releasing preservatives have the ability to release formaldehyde in very small amounts over time. The use of formaldehyde-releasing preservatives ensures that the actual level of free formaldehyde in the products is always very low but at the same time sufficient to ensure absence of microbial growth. The formaldehyde reacts most rapidly with organic and inorganic anions, amino and sulfide groups and electron-rich groups to disrupt metabolic processes, eventually causing death of the organism.</p> <p>Chemical with the aliphatic nitro group (-C-NO<sub>2</sub>) have been added to a list of DNA-reactive subgroups recognised by the National Toxicological Program (NTP, U.S. Dept Health and Human Services) for possible carcinogenic activity.</p>
<b>TITANIUM DIOXIDE</b>	<p>* IUCLID</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<b>C.I. PIGMENT VIOLET 23</b>	<p>No carcinogenic effects observed during a 43 day test animal feeding study on Pigment Violet 23. [Manufacturer]</p>
<b>C.I. PIGMENT ORANGE 13</b>	<p>In vitro screening test for mutagenicity: negative</p> <p>For diarylide (disazo) pigments (3,3'-dichlorobenzidine-containing):</p> <p>The substances in this category do not present a hazard for human health due to their low hazard profile. Adequate screening-level data are available to characterise the human health hazard for the purposes of the OECD Cooperative Chemicals Assessment Programme.</p> <p>Diarylide pigments are synthesized by bis-diazotizing diamino-diphenyl derivatives, mainly 3,3'-dichlorobenzidine (DCB), and coupling with acetoacetylides or arylsubstituted pyrazolones</p> <p>Studies indicate that essentially there is no potential for uptake via the oral and dermal routes. However, following repeated oral exposure at high dose levels, there is some evidence that a very limited uptake of the compound (or its impurities) could</p>

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occur, based on observations of staining of the mucosal surfaces of internal organs (although the possibility of contamination during necropsy cannot be excluded). In an oral reproductive developmental screening study, staining of the pups could indicate a potential for limited placental transfer, again at a high dose level. Given that the Pigment Yellows are essentially not absorbed into the body, metabolism is not relevant. However, the presence of very low levels of 3,3'-dichlorobenzidine has been demonstrated in two studies using very sensitive techniques following oral administration of some yellow pigment compounds. It seems likely that this is due to the presence of a mono-azo impurity in some of the yellow pigment parent compounds, which is absorbed and subsequently metabolised. No DCB was found in the urine of experimental animals after exposure orally or via the lungs in long term studies. Following ingestion, the vast majority of the pigments are excreted unchanged in the faeces.

Many diarylide pigments are derived from DCB. Therefore, the diarylide pigments on DCB basis have been tested toxicologically very extensively. Diarylide pigments with their LD50 values above 2 000 mg/kg show no acute toxicity according to the EU classification criteria. They are not irritating to the skin or mucous membranes.

For acute dermal toxicity a single LD50 of >3,000 mg/kg bw is available for Pigment Yellow 13. No deaths or clinical signs of toxicity were observed following oral or dermal exposure. The inhalation LC50 available is >4,448 mg/m<sup>3</sup> for Pigment Yellow 13. Tachypnoea, dyspnoea, exophthalmos, ruffled fur and curved or ventral body position were observed, although all animals recovered and no gross abnormalities were observed at necropsy.

Based on the available data the pigments have a minimal to slight potential for eye irritation. There is no indication that they are sensitisers

No adverse effects were seen after 4-7 weeks oral administration of Pigment Yellow 12 at 1000 mg/kg/day (NOAEL), the highest dose tested in a well conducted and reported test of repeated dose toxicity study. Furthermore, in the cases of Pigment Yellow 12 and 83, no toxicologically significant effects were observed in a range of chronic toxicity studies of lesser quality (in terms of reporting) in rats and mice at doses up to 6500 mg/kg/day. Based on the kinetics of the three pigments and the chemical similarities, it can be concluded that these findings can be extrapolated to most if not all diarylide pigments.

For the inhalation route the effects seen are related to the deposition of dust particles in the lungs, leading to Pigment Yellow 13 related effects even at the lowest exposure concentration of 54 mg/m<sup>3</sup> (local LOAEL). Systemically no effects were observed at the highest concentration tested, 410 mg/m<sup>3</sup> (systemic NOAEL).

All three pigments are not genotoxic in bacterial tests. Pigment Yellow 12 did not induce clastogenic effects in mammalian cells. Based on the chemical similarities between the three pigments, it is predicted that all three Yellow Pigments will not induce chromosomal changes in mammalian cells. There are no in vitro data to suggest that the pigments are genotoxic in vivo.

No increased tumour incidence after treatment with Pigment Yellow 12 and 83 were observed in several long-term studies in rats and mice (NOAEL (rat) > 630 mg/kg; NOAEL (mouse) > 1,960 mg/kg). Based on chemical similarity it can be concluded that the pigments are not carcinogenic.

It can be concluded that Pigment Yellow 12 does not have any adverse effects on reproductive parameters. There was no evidence of teratogenicity. The NOAEL for maternal and reproductive toxicity is >1,000 mg/kg bw. Supporting evidence is also available from the fact that no changes on the reproductive organs were observed in the studies of repeat dose toxicity and carcinogenicity study with Pigment Yellow 83. In view of the structural similarities and similar kinetics no effects on reproduction or development are expected from pigments of this class.

In studies of the bioavailability of several representatives of this group of pigments, no carcinogenic cleavage product was released in detectable amounts after oral, inhalative or intratracheal application on rats.

One further study of the bioavailability of DCB (DCB haemoglobin adduct) has been performed with the diarylide pigments C.I. Pigment Yellow 13 and C.I. Pigment Yellow 17. In this study, no release of carcinogenic DCB from the pigments has been detected. This indicates the absence of metabolism to DCB under the test conditions.

In summary then, according to the known studies, diarylide pigments do not represent any health risk although risks might attach to contaminants introduced during synthesis.

Colourants for Food Contact Plastics - Aspects of Product Safety; Responsible Care initiative of the European Chemical Industry Council.

For 3,3'-dichlorobenzidine:

Various tumours developed after oral or subcutaneous administration of 3,3'-dichlorobenzidine to mice, rats, hamsters and dogs. Tumours have not yet been identified in persons exposed to the substance alone. The substance can be absorbed through the skin in dangerous quantities. Increases in temperature and relative humidity promote dermal absorption.

Upper respiratory infection and sore throat were listed among several principal reasons for visits to a company's medical clinic by workers handling 3,3'-dichlorobenzidine dihydrochloride. However, there is no conclusive evidence that these effects were due to inhalation of 3,3'-dichlorobenzidine dihydrochloride.

No adverse health effects were observed in male rats exposed by inhalation to 3,3'-dichlorobenzidine free base (23,700 mg/m<sup>3</sup>) 2 hours per day for 7 days. In another study, 10 rats were exposed to an unspecified concentration of 3,3'-dichlorobenzidine dihydrochloride dust particles for 1 hour and then observed for 14 days. Slight-to-moderate pulmonary congestion and one pulmonary abscess were observed upon necropsy. The effects observed in the study using the ionized (hydrochloride) form of 3,3'-dichlorobenzidine may have been due to the irritative properties of hydrochloric acid released from the salt in combination with particulate toxicity.

Gastrointestinal upset was one of the symptoms reported by employees who worked with 3,3'-dichlorobenzidine dihydrochloride. However, there is no conclusive evidence that the gastrointestinal effects, or other symptoms reported by employees, resulted specifically from inhalation of 3,3'-dichlorobenzidine dihydrochloride.

The only relevant information regarding neurological effects in humans exposed to 3,3'-dichlorobenzidine was found in an early study which reported that headache and dizziness were among several principal reasons why employees working with 3,3'-dichlorobenzidine in a chemical manufacturing plant visited the company medical clinic. However, there is no conclusive evidence that these symptoms were caused specifically by 3,3'-dichlorobenzidine since there was exposure to other chemicals as well. In a 3,3'-dichlorobenzidine carcinogenicity study, 1 of 6 dogs exhibited convulsions after 21, 28, or 42 months of oral treatment with 10.4 mg/kg/day over a period of 3.5 years

**Carcinogenicity:** Several epidemiological studies have investigated cancer incidences among workers occupationally exposed to 3,3'-dichlorobenzidine. Exposure may have been by both inhalation and dermal routes. Due, in part, to structure-activity considerations, epidemiological studies of potential cancer effects of occupational exposure to 3,3'-dichlorobenzidine have been particularly concerned with bladder tumors, since 3,3'-dichlorobenzidine is structurally similar to benzidine, a chemical which is known to be a human bladder carcinogen. No bladder tumors were found in a group of 35 workers who handled only 3,3'-dichlorobenzidine; in the same dyestuff plant, bladder tumors occurred in 3 out of 14 workers exposed to both benzidine and 3,3'-dichlorobenzidine. The investigator reported a total exposure time of 68,505 hours, equivalent to nearly 140 full-time working years. No cases of bladder tumors were found in an epidemiology study of 259 workers exposed

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to dry and sernidry 3,3 -dichlorobenzidine base and hydrochloride. Workers were exposed to an average of less than 16 years each to 3,3 -dichlorobenzidine, which means that an adequate exposure duration and/or the latent period following exposure may not have been reached for tumor expression.

In a retrospective epidemiological study of workers employed in a dye and pigment manufacturing plant that used 3,3 -dichlorobenzidine as chemical precursor, no bladder tumors were observed in a cohort of 207 workers, most of whom had been exposed for up to 15 years. Limitations of this study included using data from a very small and incomplete sample of workers; focusing solely on the occurrence of bladder tumors; and using data that may have been misleading and, at times, apparently inaccurate.

A statistically significant increased incidence of hepatomas was observed in male ICR/JCL mice exposed to 0.1% 3,3 -dichlorobenzidine in the diet (170 mg/kg/day) at 6 months (8 of 8 treated as opposed to 0 of 5 controls) and 12 months (18 of 18 treated as opposed to 2 of 2 1 controls). Hepatic tumors were observed in 4/1 8 strain D mice exposed to 11.2-1 1.9 mg 3,3 -dichlorobenzidine/kg/day in the diet for 10 months

No bladder carcinomas were observed in rats exposed to 0.03% 3,3 -dichlorobenzidine in the diet (27 mg/kg/day) for 4 or 40 weeks , nor were any mammary tumors observed in rats administered approximately 49 mg 3,3 -dichlorobenzidine dihydrochloride/kg/day by gavage once every 3 days over a 30-day period and sacrificed 8 months later. In a study in which rats were exposed to 10-20 mg 3,3 -dichlorobenzidine per day (120 mg/kg/day) in feed 6 days per week for 12 months, tumors were observed at a variety of sites, including the Zymbal gland (7 of 29 animals), mammary gland (7/29), bladder (3/29), hematopoietic system (3/29), skin (3/29), ileum (2/29), connective tissue (2/29), salivary gland (2/29), liver (1/29), and thyroid (1/29).

In another rat study, 3,3 -dichlorobenzidine was administered to 50 male (70 mg/kg/day) and 50 female (80 mg/kg/day) Sprague-Dawley rats, in a standard diet for up to 16 months . In rats fed 3,3 -dichlorobenzidine in the diet for a total of 349 days (females) and 353 days (males), histopathological evaluations revealed mammary adenocarcinoma (16% incidence), malignant lymphoma (14%) granulocytic leukemia (20%), carcinoma of the Zymbal gland (18%) in males, and mammary adenocarcinoma (59%) in females. The authors noted that most of these tumors appeared to arise in the bone marrow and haematopoietic foci in the spleen and liver with subsequent metastasis to other organs.

**Haematological Effects.** Although haematological effects may not be sensitive indicators for 3,3 -dichlorobenzidine toxicity, haemoglobin adducts have been detected in female Wistar rats orally administered single 127 or 253 mg/kg doses of 3,3 -dichlorobenzidine or with repeated doses between 0.3 and 5.8 mg/kg/day . It was suggested that metabolically formed nitroso derivatives and the formation of a sulfinic acid amide with cysteine residues in haemoglobin may be the mechanism of adduct formation.

**Hepatic Effects.** Limited animal evidence suggests that chronic-duration oral exposure to 3,3 -dichlorobenzidine results in mild-to-moderate liver injury.

**Genotoxic effects:** Genotoxic effects have been reported in animals treated with 3,3 -dichlorobenzidine. A single dose of 3,3 -dichlorobenzidine (1,000 mg/kg) administered to male and pregnant female mice induced micronuclei in polychromatic erythrocytes in the bone marrow of the males and in the liver of the foetuses, but not in bone marrow of the dams.

In another study, an increase in unscheduled deoxyribonucleic acid synthesis (UDS) was observed in cultured liver cells from male mice previously pretreated orally with single doses of . 500 mg/kg 3,3 -dichlorobenzidine; no response was observed at a dose of .200 mg/kg. 3,3 -Dichlorobenzidine was also shown to bind extensively to tissue deoxyribonucleic acid (DNA) in rats and mice

POSTER PAINT TP31 Primary Color & TITANIUM DIOXIDE

Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.

For titanium dioxide:

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.) With regard to 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 into 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 macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide 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

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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-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.

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.

In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of 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.

**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

**POSTER PAINT TP31 Primary Color & METHACRYLIC ACID/ETHYL ACRYLATE COPOLYMER & HYDROXYETHYLCELLULOSE & TITANIUM DIOXIDE & C.I. PIGMENT GREEN 7 & C.I. PIGMENT BLACK 7 & C.I. PIGMENT RED 21 & WATER**

No significant acute toxicological data identified in literature search.

**POSTER PAINT TP31 Primary Color & PROPYLENE GLYCOL**

The acute oral toxicity of propylene glycol is very low, and large quantities are required to cause perceptible health damage in humans. Serious toxicity generally occurs only at plasma concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time. It would be nearly impossible to reach toxic levels by consuming foods or supplements, which contain at most 1 g/kg of PG. Cases of propylene glycol poisoning are usually related to either inappropriate intravenous administration or accidental ingestion of large quantities by children. The potential for long-term oral toxicity is also low. Because of its low chronic oral toxicity, propylene glycol was classified by the U. S. Food and Drug Administration as "generally recognized as safe" (GRAS) for use as a direct food additive.

Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce slight transient conjunctivitis (the eye recovers after the exposure is removed). Exposure to mists may cause eye irritation, as well as upper respiratory tract irritation. Inhalation of the propylene glycol vapours appears to present no significant hazard in ordinary applications. However, limited human experience indicates that inhalation of propylene glycol mists could be irritating to some individuals. It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations.

Propylene glycol is metabolised in the human body into pyruvic acid (a normal part of the glucose-metabolism process, readily converted to energy), acetic acid (handled by ethanol-metabolism), lactic acid (a normal acid generally abundant during digestion), and propionaldehyde (a potentially hazardous substance).

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

Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but that they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis to propylene glycol may be greater than 2% in patients with eczema.

One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic reactions, such as rhinitis or hives in children.

Another study suggested that the concentrations of PGEs (counted as the sum of propylene glycol and glycol ethers) in indoor air, particularly bedroom air, is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema, and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use of water-based paints and water-based system cleansers.

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

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

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

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

**POSTER PAINT TP31 Primary Color & HYDROCARBON**

The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives;

**OILS**

The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:

- The adverse effects of these materials are associated with undesirable components, and
- The levels of the undesirable components are inversely related to the degree of processing;
- Distillate base oils receiving the same degree or extent of processing will have similar toxicities;
- The potential toxicity of *residual base oils* is independent of the degree of processing the oil receives.
- The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing.

The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential.

Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.

Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing.

Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).

Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class (Other Lubricant Base Oils).

Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class (Other Lubricant Base Oils))

Germ cell mutagenicity: The tests performed within the "in vivo" studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class (Other Lubricant Base Oils)).

Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction.

STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOEL value of 1000 mg/kg. NOEL for inhalation, local effects > 280 mg/m<sup>3</sup> and for systemic effects NOEL > 980 mg/m<sup>3</sup>.

Sub-chronic toxicity

90-day study Dermal: NOEL > 2000 mg/kg (CONCAWE studies).

Repeat dose toxicity:

Oral

NOEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.

Inhalation

The NOEL for lung changes associated with oil deposition in the lungs was 220 mg/m<sup>3</sup>. As no systemic toxicity was observed, the overall NOEL for systemic effects was > 980 mg/m<sup>3</sup>.

Dermal

In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOEL for the test material is less than 30 mg/kg/day.

Toxicity to reproduction:

Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOEL for this study is =1000 mg/kg/day and no LOEL was determined.

Developmental toxicity, teratogenicity:

Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.

The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic

Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.



## Highly and Severely Refined Distillate Base Oils

**Acute toxicity:** Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to > 4 mg/l. When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating"

Testing in guinea pigs for sensitization has been negative

**Repeat dose toxicity:** Several studies have been conducted with these oils. The weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil s toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.

- ▶ The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,
- ▶ The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and
- ▶ The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.

**Reproductive and developmental toxicity:** A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study s authors.

A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed foetuses were found among three litters The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.

**Genotoxicity:**

*In vitro* (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.

*In vivo* (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.

**Carcinogenicity:** Highly & severely refined base oils are not carcinogens, when given either orally or dermally.

- ▶ NOTE: Detailed analysis of the molecular structure, by various Authorities/ Agencies and in other cases by Chemwatch, indicates that the azo colourant can split off carcinogenic arylamines.

The azo linkage is considered the most labile portion of an azo dye. The linkage easily undergoes enzymatic breakdown, but thermal or photochemical breakdown may also take place. The breakdown results in cleavage of the molecule and in release of the component amines. Water solubility determines the ultimate degradation pathways of the dyes. For example the azo linkage of many azo pigments is, due to very low solubility in water, not available for intracellular enzymatic breakdown but may be susceptible to endogenous micro-organisms found in the bladder or in the gut.

After cleavage of the azo linkage by bacteria, the component aromatic amines are absorbed in the intestine and excreted in the urine. Twenty-two of the component amines are recognised as potential human carcinogens, and/or several of them have shown carcinogenic potential on experimental animals. Sulfonation of the dye reduces the toxicity by enhancement of the excretion.

The component amines which may be released from azo dyes are mostly aromatic amines (compounds where an amine group or amine-generating group(s) are connected to an aryl moiety). In general, aromatic amines known as carcinogenic may be grouped into five groups

- ▶ Anilines, e.g. o-toluidine.
- ▶ Extended anilines, e.g. benzidine.
- ▶ Fused ring amines, e.g. 2-naphthylamine.
- ▶ Aminoazo and other azo compounds, e.g. 4-(phenylazo)aniline.
- ▶ Heterocyclic amines.

The aromatic amines containing moieties of anilines, extended anilines and fused ring amines are components of the majority of the industrially important azo dyes.

Reductive fission of the azo group, either by intestinal bacteria or by azo reductases of the liver and extra-hepatic tissues can cause benzidine-based aromatic amines to be released. Such breakdown products have been detected in animal experiments as well as in man (urine). Mutagenicity, which has been observed with numerous azo colourants in in vitro test systems, and the carcinogenicity in animal experiments are attributed to the release of amines and their subsequent metabolic activation.

There are now epidemiological indications that occupational exposure to benzidine-based azo colourants can increase the incidence of bladder carcinoma.

The acute toxicity of azo dyes is low.. However, potential health effects are recognised.

Despite a very broad field of application and exposure, sensitising properties of azo dyes have been identified in relatively few reports. Red azoic dyes have been linked to allergic contact dermatitis in heavily exposed workers. Furthermore, textiles coloured with disperse azo dyes have caused allergic dermatitis in a few cases.

**POSTER PAINT TP31 Primary  
Color & C.I. PIGMENT  
YELLOW 3 & C.I. PIGMENT  
RED 8**

**CALCIUM CARBONATE & 2-  
BROMO-2-NITROPROPAN-  
1,3-DIOL & TITANIUM  
DIOXIDE**

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

**CALCIUM CARBONATE &  
PROPYLENE GLYCOL & 2-**

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.

## POSTER PAINT TP31 Primary Color

<b>BROMO-2-NITROPROPAN-1,3-DIOL</b>	Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
<b>HYDROCARBON OILS &amp; TITANIUM DIOXIDE</b>	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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

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

## SECTION 12 Ecological information

## Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
<b>POSTER PAINT TP31 Primary Color</b>	Not Available	Not Available	Not Available	Not Available	Not Available
<b>calcium carbonate</b>	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	NOEC(ECx)	1h	Fish	4-320mg/l	4
	LC50	96h	Fish	>165200mg/L	4
<b>methacrylic acid/ ethyl acrylate copolymer</b>	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
<b>propylene glycol</b>	EC50	48h	Crustacea	>114.4mg/L	4
	EC50	96h	Algae or other aquatic plants	19000mg/l	2
	EC50	72h	Algae or other aquatic plants	19300mg/l	2
	NOEC(ECx)	336h	Algae or other aquatic plants	<5300mg/l	1
	LC50	96h	Fish	710mg/l	4
<b>monoisobutanolamine</b>	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	>103mg/l	2
	EC50	48h	Crustacea	193mg/l	1
	EC50	72h	Algae or other aquatic plants	>103mg/l	2
	EC0(ECx)	48h	Crustacea	100mg/l	1
<b>hydroxyethylcellulose</b>	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
<b>hydrocarbon oils</b>	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
<b>2-bromo-2-nitropropan-1,3-diol</b>	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	1.1-3.52mg/L	4
	EC50	96h	Algae or other aquatic plants	0.02-0.025mg/L	4
	EC50	72h	Algae or other aquatic plants	0.026mg/l	2
	EC10(ECx)	72h	Algae or other aquatic plants	0.013mg/l	2

Continued...

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	LC50	96h	Fish	10.274-14.454mg/L	4
titanium dioxide	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	48h	Crustacea	1.9mg/l	2
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	LC50	96h	Fish	1.85-3.06mg/l	4
C.I. Pigment Green 7	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	BCF	1008h	Fish	0.51-4.8	7
	EC50	48h	Crustacea	153.6mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	504h	Crustacea	>=1mg/l	2
	LC50	96h	Fish	>100mg/l	2
C.I. Pigment Yellow 1	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>1mg/l	2
	NOEC(ECx)	504h	Crustacea	1mg/l	2
C.I. Pigment Yellow 3	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	504h	Crustacea	1mg/l	2
	LC50	96h	Fish	>1mg/l	2
C.I. Pigment Black 7	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	33.076-41.968mg/l	4
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	NOEC(ECx)	24h	Crustacea	3200mg/l	1
	LC50	96h	Fish	>100mg/l	2
C.I. Pigment Red 8	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Pigment Blue 15	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	BCF	1008h	Fish	<0.33-11	7
	EC50	48h	Crustacea	>500mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50(ECx)	504h	Crustacea	>1mg/l	2
	LC50	96h	Fish	>100mg/l	2
C.I. Pigment Violet 23	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC0(ECx)	48h	Crustacea	>=100mg/l	2
C.I. Pigment Red 21	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Pigment Orange 13	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	BCF	1008h	Fish	0.75-5.6	7

Continued...

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	NOEC(ECx)	504h	Crustacea	1mg/l	2
	LC50	96h	Fish	>500mg/l	2
<b>water</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	Not Available	Not Available	Not Available	Not Available	Not Available
<b>Legend:</b>	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				

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

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

## Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene glycol	LOW	LOW
monoisobutanolamine	LOW	LOW
hydroxyethylcellulose	LOW	LOW
2-bromo-2-nitropropan-1,3-diol	LOW	LOW
titanium dioxide	HIGH	HIGH
C.I. Pigment Yellow 1	HIGH	HIGH
C.I. Pigment Yellow 3	HIGH	HIGH
C.I. Pigment Blue 15	HIGH	HIGH
water	LOW	LOW

## Bioaccumulative potential

Ingredient	Bioaccumulation
propylene glycol	LOW (BCF = 1)
monoisobutanolamine	LOW (BCF = 330)
hydroxyethylcellulose	LOW (LogKOW = -8.995)
2-bromo-2-nitropropan-1,3-diol	LOW (LogKOW = -0.6408)
titanium dioxide	LOW (BCF = 10)
C.I. Pigment Green 7	LOW (BCF = 74)
C.I. Pigment Yellow 1	MEDIUM (LogKOW = 3.9388)
C.I. Pigment Yellow 3	MEDIUM (LogKOW = 4.1171)
C.I. Pigment Blue 15	LOW (BCF = 11)
C.I. Pigment Orange 13	LOW (BCF = 5.6)

## Mobility in soil

Ingredient	Mobility
propylene glycol	HIGH (Log KOC = 1)
monoisobutanolamine	MEDIUM (Log KOC = 2.196)
hydroxyethylcellulose	LOW (Log KOC = 10)
2-bromo-2-nitropropan-1,3-diol	HIGH (Log KOC = 1)
titanium dioxide	LOW (Log KOC = 23.74)
C.I. Pigment Yellow 1	LOW (Log KOC = 278.5)
C.I. Pigment Yellow 3	LOW (Log KOC = 460.5)
C.I. Pigment Blue 15	LOW (Log KOC = 1000000000)

## SECTION 13 Disposal considerations

## Waste treatment methods

## Product / Packaging disposal

- ▶ Containers may still present a chemical hazard/ danger when empty.
- ▶ Return to supplier for reuse/ recycling if possible.

Continued...

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Otherwise:

- ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.
- ▶ **DO NOT allow wash water from cleaning or process equipment to enter drains.**
- ▶ It may be necessary to collect all wash water for treatment before disposal.
- ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- ▶ Where in doubt contact the responsible authority.
- ▶ Recycle wherever possible or consult manufacturer for recycling options.
- ▶ Consult State Land Waste Authority for disposal.
- ▶ Bury or incinerate residue at an approved site.
- ▶ Recycle containers if possible, or dispose of in an authorised landfill.

**SECTION 14 Transport information**

**Labels Required**

<b>Marine Pollutant</b>	NO
<b>HAZCHEM</b>	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
calcium carbonate	Not Available
methacrylic acid/ ethyl acrylate copolymer	Not Available
propylene glycol	Not Available
monoisobutanolamine	Not Available
hydroxyethylcellulose	Not Available
hydrocarbon oils	Not Available
2-bromo-2-nitropropan-1,3-diol	Not Available
titanium dioxide	Not Available
C.I. Pigment Green 7	Not Available
C.I. Pigment Yellow 1	Not Available
C.I. Pigment Yellow 3	Not Available
C.I. Pigment Black 7	Not Available
C.I. Pigment Red 8	Not Available
C.I. Pigment Blue 15	Not Available
C.I. Pigment Violet 23	Not Available
C.I. Pigment Red 21	Not Available
C.I. Pigment Orange 13	Not Available
water	Not Available

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

Product name	Ship Type
calcium carbonate	Not Available
methacrylic acid/ ethyl acrylate copolymer	Not Available
propylene glycol	Not Available
monoisobutanolamine	Not Available
hydroxyethylcellulose	Not Available
hydrocarbon oils	Not Available

Product name	Ship Type
2-bromo-2-nitropropan-1,3-diol	Not Available
titanium dioxide	Not Available
C.I. Pigment Green 7	Not Available
C.I. Pigment Yellow 1	Not Available
C.I. Pigment Yellow 3	Not Available
C.I. Pigment Black 7	Not Available
C.I. Pigment Red 8	Not Available
C.I. Pigment Blue 15	Not Available
C.I. Pigment Violet 23	Not Available
C.I. Pigment Red 21	Not Available
C.I. Pigment Orange 13	Not Available
water	Not Available

## SECTION 15 Regulatory information

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

#### calcium carbonate 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)

#### methacrylic acid/ ethyl acrylate copolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

#### propylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

#### monoisobutanolamine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

#### hydroxyethylcellulose is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

#### hydrocarbon oils 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 1: Carcinogenic to humans

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

#### 2-bromo-2-nitropropan-1,3-diol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

#### 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)

#### C.I. Pigment Green 7 is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

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

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

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

#### C.I. Pigment Yellow 1 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

**C.I. Pigment Yellow 3 is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**C.I. Pigment Black 7 is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

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)

**C.I. Pigment Red 8 is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**C.I. Pigment Blue 15 is found on the following regulatory lists**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

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

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

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**C.I. Pigment Violet 23 is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**C.I. Pigment Red 21 is found on the following regulatory lists**

Not Applicable

**C.I. Pigment Orange 13 is found on the following regulatory lists**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**water is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**Additional Regulatory Information**

Not Applicable

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (C.I. Pigment Red 21)
Canada - DSL	No (C.I. Pigment Red 21)
Canada - NDSL	No (methacrylic acid/ ethyl acrylate copolymer; propylene glycol; monoisobutanolamine; hydroxyethylcellulose; hydrocarbon oils; 2-bromo-2-nitropropan-1,3-diol; C.I. Pigment Green 7; C.I. Pigment Yellow 1; C.I. Pigment Yellow 3; C.I. Pigment Black 7; C.I. Pigment Red 8; C.I. Pigment Blue 15; C.I. Pigment Violet 23; C.I. Pigment Orange 13; water)
China - IECSC	No (C.I. Pigment Red 21)
Europe - EINEC / ELINCS / NLP	No (methacrylic acid/ ethyl acrylate copolymer; hydroxyethylcellulose; hydrocarbon oils)
Japan - ENCS	Yes
Korea - KECI	No (hydrocarbon oils; C.I. Pigment Red 21)
New Zealand - NZIoC	No (C.I. Pigment Red 21)
Philippines - PICCS	No (C.I. Pigment Red 21)
USA - TSCA	No (hydrocarbon oils)
Taiwan - TCSI	No (C.I. Pigment Red 21)
Mexico - INSQ	No (hydrocarbon oils; C.I. Pigment Green 7; C.I. Pigment Yellow 3; C.I. Pigment Red 8; C.I. Pigment Red 21)
Vietnam - NCI	Yes
Russia - FBEPH	No (methacrylic acid/ ethyl acrylate copolymer; hydrocarbon oils; C.I. Pigment Red 8; C.I. Pigment Red 21)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory

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National Inventory	Status
	No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

## SECTION 16 Other information

Revision Date	28/03/2024
Initial Date	28/03/2024

## SDS Version Summary

Version	Date of Update	Sections Updated
2.1	28/03/2024	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, Firefighting measures - Fire Fighter (extinguishing media), Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), Firefighting measures - Fire Fighter (fire incompatibility), First Aid measures - First Aid (eye), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Stability and reactivity - Instability Condition, Exposure controls / personal protection - Personal Protection (other), Exposure controls / personal protection - Personal Protection (eye), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Handling and storage - Storage (suitable container), Toxicological information - Toxicity and Irritation (Other), Transport information - Transport, Identification of the substance / mixture and of the company / undertaking - Use

## Other information

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

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

## Definitions and abbreviations

- PC - TWA: Permissible Concentration-Time Weighted Average
- PC - STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit,
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
  
- 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
- KECL: 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



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- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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