

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

Jasco Pty Limited

Chemwatch Hazard Alert Code: 3

Chemwatch: 5672-03

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

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

Product Identifier

Product name	BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS
Chemical Name	Not Applicable
Synonyms	0121870 BYRON MATTE ACR PAINT 60ML SET 5 CLASSIC; 0121880 BYRON MATTE ACR PAINT 60ML SET 5 EARTH; 0121890 BYRON MATTE ACR PAINT 60ML SET 5 ANTIQUE; 0121900 BYRON MATTE ACR PAINT 60ML SET 5 PASTEL; 0121910 BYRON MATTE ACR PAINT 60ML SET 5 BLOOM; 0121920 BYRON MATTE ACR PAINT 60ML SET 5 TWILIGHT; 0121940 BYRON MATTE ACR PAINT 60ML SET 10 CLASSIC; 0121510 BYRON MATTE ACR PAINT 60ML SET 10 NEUTRAL; 0121520 BYRON MATTE ACR PAINT 60ML SET 20 CLASSIC
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains C.I. Pigment Blue 1)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

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

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

Emergency telephone number

Association / Organisation	Australian Poisons Centre	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone numbers	13 11 26 (24/7)	+61 1800 951 288
Other emergency telephone numbers	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

Poisons Schedule	Not Applicable
Classification ^[1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Germ Cell Mutagenicity Category 2, Carcinogenicity Category 1A, Hazardous to the Aquatic Environment Acute Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

Hazard pictogram(s)	
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Signal word	Danger
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Hazard statement(s)

H315	Causes skin irritation.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
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.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
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.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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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	30	<u>calcium carbonate</u>
25085-34-1	18.3	<u>styrene/ acrylic acid copolymer</u>
57-55-6	5	<u>propylene glycol</u>
7631-86-9	1	<u>silica amorphous</u>
25265-77-4	1	<u>2,2,4-trimethyl-1,3-pentanediol monoisobutyrate</u>
124-68-5	0.35	<u>monoisobutanolamine</u>
8020-83-5	0.3	<u>hydrocarbon oils</u>
9004-62-0	0.2	<u>hydroxyethylcellulose</u>

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CAS No	%[weight]	Name
84133-50-6	0.2	<u>alcohols C12-14 secondary ethoxylated</u>
52-51-7	0.05	<u>2-bromo-2-nitropropan-1,3-diol</u>
13463-67-7	0-6.1	<u>titanium dioxide</u>
1328-53-6	0-6.1	<u>C.I. Pigment Green 7</u>
2512-29-0	0-6.1	<u>C.I. Pigment Yellow 1</u>
6486-23-3	0-6.1	<u>C.I. Pigment Yellow 3</u>
1333-86-4	0-6.1	<u>carbon black</u>
5280-68-2	0-6.1	<u>C.I. Pigment Red 146</u>
57455-37-5	0-6.1	<u>C.I. Pigment Blue 29</u>
147-14-8	0-6.1	<u>C.I. Pigment Blue 15</u>
1325-87-7	0-6.1	<u>C.I. Pigment Blue 1</u>
64294-91-3	0-6.1	<u>C.I. Pigment Yellow 43</u>
20344-49-4	0-6.1	<u>ferric hydroxide</u>
1309-37-1	0-6.1	<u>red iron oxide</u>
6535-46-2	0-6.1	<u>C.I. Pigment Red 112</u>
6410-26-0	0-6.1	<u>C.I. Pigment Red 21</u>
3520-72-7	0-6.1	<u>C.I. Pigment Orange 13</u>
52357-70-7	0-6.1	<u>umber brown oxide</u>
6410-30-6	0-6.1	<u>C.I. Pigment Red 8</u>
980-26-7	0-6.1	<u>C.I. Pigment Red 122</u>
1317-61-9	0-6.1	<u>C.I. Pigment Black 11</u>
52357-70-7	0-6.1	<u>umber brown oxide</u>
7732-18-5	38	<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

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor. <p>For thermal burns:</p> <ul style="list-style-type: none"> ▶ Decontaminate area around burn. ▶ Consider the use of cold packs and topical antibiotics. <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> ▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▶ Use compresses if running water is not available. ▶ Cover with sterile non-adhesive bandage or clean cloth. ▶ Do NOT apply butter or ointments; this may cause infection. ▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> ▶ Cool the burn by immerse in cold running water for 10-15 minutes. ▶ Use compresses if running water is not available. ▶ Do NOT apply ice as this may lower body temperature and cause further damage. ▶ Do NOT break blisters or apply butter or ointments; this may cause infection. ▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> ▶ Lay the person flat. ▶ Elevate feet about 12 inches. ▶ Elevate burn area above heart level, if possible. ▶ Cover the person with coat or blanket. ▶ Seek medical assistance. <p>For third-degree burns</p> <p>Seek immediate medical or emergency assistance.</p>

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	<p>In the mean time:</p> <ul style="list-style-type: none"> ▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▶ Separate burned toes and fingers with dry, sterile dressings. ▶ Do not soak burn in water or apply ointments or butter; this may cause infection. ▶ To prevent shock see above. ▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▶ Have a person with a facial burn sit up. ▶ Check pulse and breathing to monitor for shock until emergency help arrives.
<p>Inhalation</p>	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
<p>Ingestion</p>	<ul style="list-style-type: none"> ▶ IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY. ▶ For advice, contact a Poisons Information Centre or a doctor. ▶ Urgent hospital treatment is likely to be needed. ▶ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▶ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▶ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. <p>Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:</p> <ul style="list-style-type: none"> ▶ INDUCE vomiting with fingers down the back of the throat, ONLY IF CONSCIOUS. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. <p>NOTE: Wear a protective glove when inducing vomiting by mechanical means.</p>

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

<p>Fire Incompatibility</p>	<p>None known.</p>
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Advice for firefighters

<p>Fire Fighting</p>	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
<p>Fire/Explosion Hazard</p>	<ul style="list-style-type: none"> ▶ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▶ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. ▶ Non combustible. ▶ Not considered to be a significant fire risk. ▶ Expansion or decomposition on heating may lead to violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <p>carbon dioxide (CO2) hydrogen chloride</p>

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	<p>phosgene nitrogen oxides (NOx) silicon dioxide (SiO₂) metal oxides other pyrolysis products typical of burning organic material. CARE: 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>
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SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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

SECTION 7 Handling and storage

Precautions for safe handling

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

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▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks. ▶ Glass container is suitable for laboratory quantities
Storage incompatibility	▶ Avoid strong acids, bases.

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	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated) (respirable dust)	2 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
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	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	C.I. Pigment Blue 1	Molybdenum, soluble compounds (as Mo)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric hydroxide	Iron oxide fume (Fe ₂ O ₃) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric hydroxide	Rouge dust	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	red iron oxide	Rouge dust	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	red iron oxide	Iron oxide fume (Fe ₂ O ₃) (as Fe)	5 mg/m ³	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
calcium carbonate	45 mg/m ³	210 mg/m ³	1,300 mg/m ³
propylene glycol	30 mg/m ³	1,300 mg/m ³	7,900 mg/m ³
silica amorphous	18 mg/m ³	200 mg/m ³	1,200 mg/m ³
silica amorphous	18 mg/m ³	100 mg/m ³	630 mg/m ³
silica amorphous	120 mg/m ³	1,300 mg/m ³	7,900 mg/m ³
silica amorphous	45 mg/m ³	500 mg/m ³	3,000 mg/m ³
silica amorphous	18 mg/m ³	740 mg/m ³	4,500 mg/m ³
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	13 mg/m ³	140 mg/m ³	840 mg/m ³
monoisobutanolamine	17 mg/m ³	190 mg/m ³	570 mg/m ³
hydrocarbon oils	140 mg/m ³	1,500 mg/m ³	8,900 mg/m ³
titanium dioxide	30 mg/m ³	330 mg/m ³	2,000 mg/m ³
carbon black	9 mg/m ³	99 mg/m ³	590 mg/m ³
ferric hydroxide	30 mg/m ³	330 mg/m ³	2,000 mg/m ³
ferric hydroxide	15 mg/m ³	360 mg/m ³	2,200 mg/m ³
ferric hydroxide	24 mg/m ³	260 mg/m ³	1,600 mg/m ³
red iron oxide	15 mg/m ³	360 mg/m ³	2,200 mg/m ³
C.I. Pigment Black 11	21 mg/m ³	230 mg/m ³	1,400 mg/m ³

Ingredient	Original IDLH	Revised IDLH
calcium carbonate	Not Available	Not Available
styrene/ acrylic acid copolymer	Not Available	Not Available
propylene glycol	Not Available	Not Available
silica amorphous	3,000 mg/m ³	Not Available
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Not Available	Not Available
monoisobutanolamine	Not Available	Not Available
hydrocarbon oils	2,500 mg/m ³	Not Available
hydroxyethylcellulose	Not Available	Not Available
alcohols C12-14 secondary ethoxylated	Not Available	Not Available
2-bromo-2-nitropropan-1,3-diol	Not Available	Not Available
titanium dioxide	5,000 mg/m ³	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
carbon black	1,750 mg/m ³	Not Available
C.I. Pigment Red 146	Not Available	Not Available
C.I. Pigment Blue 29	Not Available	Not Available
C.I. Pigment Blue 15	Not Available	Not Available
C.I. Pigment Blue 1	1,000 mg/m ³	Not Available
C.I. Pigment Yellow 43	Not Available	Not Available
ferric hydroxide	2,500 mg/m ³	Not Available
red iron oxide	2,500 mg/m ³	Not Available
C.I. Pigment Red 112	Not Available	Not Available
C.I. Pigment Red 21	Not Available	Not Available
C.I. Pigment Orange 13	Not Available	Not Available
umber brown oxide	Not Available	Not Available
C.I. Pigment Red 8	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
C.I. Pigment Red 122	Not Available	Not Available
C.I. Pigment Black 11	Not Available	Not Available
umber brown oxide	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 ³
alcohols C12-14 secondary ethoxylated	E	≤ 0.1 ppm
2-bromo-2-nitropropan-1,3-diol	E	≤ 0.01 mg/m ³
C.I. Pigment Yellow 1	E	≤ 0.01 mg/m ³
C.I. Pigment Yellow 3	E	≤ 0.01 mg/m ³
C.I. Pigment Red 21	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m ³)
C.I. Pigment Orange 13	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m ³)
umber brown oxide	E	≤ 0.01 mg/m ³
C.I. Pigment Red 8	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m ³)
C.I. Pigment Black 11	E	≤ 0.01 mg/m ³
umber brown oxide	E	≤ 0.01 mg/m ³
Notes:	<i>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.</i>	

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.

Airborne particulate or vapour must be kept to levels as low as is practicably achievable given access to modern engineering controls and monitoring hardware. Biologically active compounds may produce idiosyncratic effects which are entirely unpredictable on the basis of literature searches and prior clinical experience (both recent and past).

NOTE: This substance has been classified by the ACGIH as A4 **NOT** classifiable as causing Cancer in humans

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/m³ (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.

The concentration of respirable dust for application of this limit is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative lognormal function with a median aerodynamic volume of 4.0 µm (+-) 0.3 µm and with a geometric standard deviation of 1.5 µm (+-) 0.1 µm, i.e.. less than 5 µm.

The TLV-TWA is thought to be sufficiently low to prevent changes in pre-employment chest X-ray findings in exposed employees, in some cases following decades of exposure. The limit is thought to be protective against disabling pneumoconiosis.

For amorphous crystalline silica (precipitated silicic acid):

Amorphous crystalline silica shows little potential for producing adverse effects on the lung and exposure standards should reflect a particulate of low intrinsic toxicity. Mixtures of amorphous silicas/ diatomaceous earth and crystalline silica should be monitored as if they comprise only the crystalline forms.

The dusts from precipitated silica and silica gel produce little adverse effect on pulmonary functions and are not known to produce significant disease or toxic effect.

IARC has classified silica, amorphous as Group 3: **NOT** classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

For iron oxide (ferric oxide):

Inhalation of iron oxide dust or fume may produce a benign pneumoconiosis (siderosis). The TLV-TWA is recommended to minimise the potential for development of X-ray changes in the lung on long-term exposure. These changes are not considered to be associated with any physical impairment of lung function, although more sophisticated physiological testing, including measurement of the lung's mechanical properties and expiratory lung flow is required to reach firm and final conclusions.

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/m³ 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

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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	<p>Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation. HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours. Barrier protection or laminar flow cabinets should be considered for laboratory scale handling. A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg. When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology. Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies. Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required. Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved. Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>	
	Type of Contaminant:	Air Speed:
	solvent, vapours, etc. evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min.)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
	3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only	
<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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p> <p>The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated.</p> <p>The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of:</p> <p>10; high efficiency particulate (HEPA) filters or cartridges 10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator. 25-50; a full face-piece negative pressure respirator with HEPA filters 50-100; tight-fitting, full face-piece HEPA PAPR 100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode.</p>		
Individual protection measures, such as personal protective equipment		
Eye and face protection	<p>When handling very small quantities of the material eye protection may not be required. For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs:</p> <ul style="list-style-type: none"> Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] Face shield. Full face shield may be required for supplementary but never for primary protection of eyes. 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. 	

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	Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Elbow length PVC gloves <p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. ▶ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▶ Double gloving should be considered. ▶ PVC gloves. ▶ Change gloves frequently and when contaminated, punctured or torn. ▶ Wash hands immediately after removing gloves. ▶ Protective shoe covers. [AS/NZS 2210] ▶ Head covering.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ For quantities up to 500 grams a laboratory coat may be suitable. ▶ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ▶ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ▶ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ▶ Eye wash unit. ▶ Ensure there is ready access to an emergency shower. ▶ For Emergencies: Vinyl suit

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the: **"Forsberg Clothing Performance Index"**.
The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:
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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. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 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)

- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
- The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
- Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
- Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
- Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)

· Use approved positive flow mask if significant quantities of dust becomes airborne.

· Try to avoid creating dust conditions.

Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.

Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.

Filtration rate: Filters at least 99.95% of airborne particles

Suitable for:

· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.

· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.

· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS

· Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos

Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

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

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

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<p style="text-align: center;">Inhaled</p>	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.</p> <p>Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.</p> <p>Manganese fume is toxic and produces nervous system effects characterised by tiredness. Acute poisoning is rare although acute inflammation of the lungs may occur. A chemical pneumonia may also result from frequent exposure. 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> <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>
<p style="text-align: center;">Ingestion</p>	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.</p> <p>Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality</p> <p>Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.</p> <p>Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.</p>
<p style="text-align: center;">Skin Contact</p>	<p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Skin contact with the material may produce severe damage to the health of the individual; systemic effects may result following absorption and these may be fatal.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>

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	<p>Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</p> <p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.</p>
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.
Chronic	<p>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.</p> <p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>When administered in the diet, 3,3'-dichlorobenzidine induced hepatomas in male mice, increased the incidences of granulocytic leukemia and Zymbal gland carcinomas in male rats and mammary adenocarcinomas in rats of both sexes, induced transitional cell carcinomas of the urinary bladder in hamsters and female dogs and hepatocellular carcinomas in female dogs.</p> <p>Transplacental exposure increased the incidences of lymphoid leukemia in mice. In three retrospective epidemiological studies, no urinary bladder tumors were reported in men occupationally exposed to 3,3'-dichlorobenzidine</p> <p>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.</p>

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS	TOXICITY	IRRITATION
	Not Available	Not Available
calcium carbonate	<p>dermal (rat) LD50: >2000 mg/kg^[1]</p> <p>Inhalation (Rat) LC50: >3 mg/l4h^[1]</p> <p>Oral (Rat) LD50: >2000 mg/kg^[1]</p>	<p>Eye (rabbit): 0.75 mg/24h - SEVERE</p> <p>Eye: no adverse effect observed (not irritating)^[1]</p> <p>Skin (rabbit): 500 mg/24h-moderate</p> <p>Skin: no adverse effect observed (not irritating)^[1]</p>
styrene/ acrylic acid copolymer	Not Available	Not Available
propylene glycol	<p>Dermal (rabbit) LD50: 11890 mg/kg^[2]</p> <p>Inhalation (Rat) LC50: >44.9 mg/l4h^[1]</p> <p>Oral (Rat) LD50: 20000 mg/kg^[2]</p>	<p>Eye (rabbit): 100 mg - mild</p> <p>Eye (rabbit): 500 mg/24h - mild</p> <p>Eye: no adverse effect observed (not irritating)^[1]</p> <p>Skin(human):104 mg/3d Intermit Mod</p> <p>Skin(human):500 mg/7days mild</p> <p>Skin: no adverse effect observed (not irritating)^[1]</p>
silica amorphous	<p>dermal (rat) LD50: >2000 mg/kg^[1]</p> <p>Inhalation (Rat) LC50: >0.09<0.84 mg/l4h^[1]</p> <p>Oral (Rat) LD50: >1000 mg/kg^[1]</p>	<p>Eye (rabbit): non-irritating ** [Grace]</p> <p>Eye: no adverse effect observed (not irritating)^[1]</p> <p>Skin (rabbit): non-irritating *</p> <p>Skin: no adverse effect observed (not irritating)^[1]</p>
2,2,4-trimethyl-1,3-pentenediol monoisobutyrate	<p>dermal (guinea pig) LD50: >19 mg/kg^[2]</p> <p>Oral (Rat) LD50: >3200 mg/kg^[2]</p>	<p>Eye: no adverse effect observed (not irritating)^[1]</p> <p>Eyes - Moderate irritant *</p>

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		Skin - Slight irritant *
		Skin (rabbit): mild ***
		Skin: no adverse effect observed (not irritating) ^[1]
monoisobutanolamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1] Oral (Mouse) LD50; 2150 mg/kg ^[2]	Not Available
hydrocarbon oils	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2] Oral (Rabbit) LD50; 2835 mg/kg ^[2]	Eye (rabbit) 100 mg/24H mild Skin (rabbit) 500 mg/24H mild
hydroxyethylcellulose	TOXICITY	IRRITATION
	Not Available	Not Available
alcohols C12-14 secondary ethoxylated	TOXICITY	IRRITATION
	Not Available	Not Available
2-bromo-2-nitropropan-1,3-diol	TOXICITY	IRRITATION
	dermal (rat) LD50: ~1600 mg/kg ^[1] Inhalation (Rat) LC50: >0.12<1.14 mg/l4h ^[1]	Eye (rabbit): 5 mg Skin (human): 10 mg moderate
	Oral (Rat) LD50: 180 mg/kg ^[2]	Skin (rabbit): 500 mg/24h mild Skin (rabbit): 80 mg moderate
titanium dioxide	TOXICITY	IRRITATION
	dermal (hamster) LD50: >=10000 mg/kg ^[2] Inhalation (Rat) LC50: >2.28 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin (human): 0.3 mg /3D (int)-mild *
	Oral (Rat) LD50: >=2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
C.I. Pigment Green 7	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: >1.084<5.212 mg/l4h ^[1] Oral (Mouse) LD50; 8400 mg/kg ^[2]	Not Available
C.I. Pigment Yellow 1	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50: >5000 mg/kg ^[2]	Non-irritating/non-sensitising [Dominion]
C.I. Pigment Yellow 3	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50: >10000 mg/kg ^[2]	Not Available
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
C.I. Pigment Red 146	TOXICITY	IRRITATION
	Oral (Rat) LD50: 10000 mg/kg ^[2]	Not Available
C.I. Pigment Blue 29	TOXICITY	IRRITATION
	Oral (Rat) LD50: >10000 mg/kg ^[2]	Not Available
C.I. Pigment Blue 15	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Inhalation (Rat) LC50: >1.084<5.212 mg/l4h ^[1]	Eye (human): non-irritant [Manuf. C.G.] Skin (human): non-irritant
	Oral (Rat) LD50: >2000 mg/kg ^[1]	

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C.I. Pigment Blue 1	TOXICITY	IRRITATION
	Oral (Rat) LD50: >5000 mg/kg ^[2]	Not Available
C.I. Pigment Yellow 43	TOXICITY	IRRITATION
	Not Available	Not Available
ferric hydroxide	TOXICITY	IRRITATION
	Oral (Rat) LD50: >10000 mg/kg ^[2]	Not Available
red iron oxide	TOXICITY	IRRITATION
	Oral (Rat) LD50: >5000 mg/kg ^[2]	Eye (rabbit): non-irritant Skin (rabbit): non-irritant 24h
C.I. Pigment Red 112	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1] Oral (Rat) LD50: >5000 mg/kg ^[2]	Not Available
C.I. Pigment Red 21	TOXICITY	IRRITATION
	Not Available	Not Available
C.I. Pigment Orange 13	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50: >10000 mg/kg ^[2]	Not Available
umber brown oxide	TOXICITY	IRRITATION
	Oral (Rat) LD50: >10000 mg/kg ^[2]	Not Available
C.I. Pigment Red 8	TOXICITY	IRRITATION
	Oral (Rat) LD50: >5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
C.I. Pigment Red 122	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2] Oral (Rat) LD50: >2000 mg/kg ^[1]	Not Available
C.I. Pigment Black 11	TOXICITY	IRRITATION
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Not Available
umber brown oxide	TOXICITY	IRRITATION
	Oral (Rat) LD50: >10000 mg/kg ^[2]	Eyes (rabbit) 50 mg Non-irrit. [Hodgsons] Skin (rabbit) 500mg Non-irrit.
water	TOXICITY	IRRITATION
	Oral (Rat) LD50: >90000 mg/kg ^[2]	Not Available

Legend:

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

CALCIUM CARBONATE	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
SILICA AMORPHOUS	Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]
2,2,4-TRIMETHYL-1,3-PENTANEDIOL MONOISOBUTYRATE	Not a skin sensitiser (guinea pig, Magnusson-Kligman) *** Ames Test: negative *** Micronucleus, mouse: negative *** Not mutagenic *** No effects on fertility or foetal development seen in the rat *** [SWIFT] ** [Eastman] *** [Perstop]
MONOISOBUTANOLAMINE	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

Continued...

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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%

Acute toxicity: 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 sensitizer. 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.

Repeated dose toxicity: 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 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 least 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. **Genetic toxicity:** Studies conducted on the TRIS AMINO and the surrogate substances in the presence or absence of mammalian metabolic enzymes have all been negative.

ALCOHOLS C12-14
SECONDARY ETHOXYLATED

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

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

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

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

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

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

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

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

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

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

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

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

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene

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glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

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

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products.

However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing.

Overall, alcohol alkoxylates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length $C \geq 6$ may also contain shorter alkyl chains $C < 6$. It is not practical to quantify the proportion of shorter $C < 6$ chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.

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

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

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

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

>20 EO is not classified (CESIO 2000)

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

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

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

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

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

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

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

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

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of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

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

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

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

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

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

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

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

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

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

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

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

2-BROMO-2-NITROPROPAN-1,3-DIOL

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 > 1000 ppm (>0.1%), have to be labelled as carcinogenic.

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.

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.

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.

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).

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

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	<p>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. 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,</p> <p><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></p> <p>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-NO₂) have been added to a list of DNA-reactive subgroups recognised by the National Toxicological Program (NTP, U.S. Dept Health and Human Services) for possible carcinogenic activity.</p>
TITANIUM DIOXIDE	<p>* IUCLID</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m ³ /6h/90D-I Nil reported
C.I. PIGMENT BLUE 29	NOTE: 90 day (chronic), teratological and mutagenicity tests here all provided negative results. Animal tests have also demonstrated no skin irritation or sensitization. [IC]
C.I. PIGMENT RED 112	Mutagenicity: In vitro screening tests have reported negative results.
C.I. PIGMENT ORANGE 13	In vitro screening test for mutagenicity: negative
C.I. PIGMENT RED 122	<p>The utility of acridines and acridones as chemotherapeutics is due to their chemical and biological stability and their capability of effective binding to DNA or RNA, resulting in the disorder of the biological functions in living cells. The mechanism of their intercalation into DNA is based on p-stacking interaction with base pairs of double-stranded nucleic acids. The heterocyclic, polyaromatic flat structure of acridine fits effectively into the gap between two chains of polynucleotides, and the intercalation of the acridine moiety disturbs their crucial role in cell division. The ability of acridines to intercalate into DNA is necessary for their antitumor activity. The strength and kinetics of binding acridine to DNA have a crucial impact on the activity of this type of anticancer agent. Examination of a large number of such derivatives proved that there is a good correlation between their strength together with the time of binding to DNA and their biological activity. Acridine derivatives perturb the function of cancer cells by decreasing the activity of some enzymes that are crucial for proper DNA actions, such as topoisomerases, telomerases and cyclin-dependent kinases.</p>
BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS & TITANIUM DIOXIDE	<p>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.</p> <p>For titanium dioxide:</p> <p>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.</p> <p>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.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>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.</p> <p>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</p>

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particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.

Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.

Animal carcinogenicity data

Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.

In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-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.

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PAINT PRIMARY COLOURS &
CALCIUM CARBONATE & 2-
BROMO-2-NITROPROPAN-
1,3-DIOL & TITANIUM
DIOXIDE & C.I. PIGMENT
YELLOW 43 & UMBER
BROWN OXIDE & C.I.
PIGMENT BLACK 11**

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.

**BYRON MATTE ACRYLIC
PAINT PRIMARY COLOURS &
C.I. PIGMENT RED 122**

For HIF ((hypoxia-inducible factor) inhibitors
Considering that endothelial HIF-1alpha was shown to be critical for left heart adaptation to overload, systemically targeting HIFs might have unintended consequences for ventricular adaptation in pulmonary hypertension (PH). HIF-2 inhibition appeared to improve right ventricular haemodynamics over a short period, but a detailed functional analysis at later time points would be prudent.
Under normoxic conditions, HIF-1alpha and HIF-2alpha are hydroxylated by PHD (prolyl hydroxylase domain) proteins (particularly PHD2), ubiquitinated, and rapidly degraded. PHD activity becomes rate limited during hypoxia, allowing accumulation of HIF-1alpha/2alpha and induction of HIF activity.
Additionally, the observation that mice with loss of PHD2 developed severe PH should raise a cautionary flag regarding the clinical use of PHD inhibitors, which are currently in development for chronic anemia. Early clinical trials did not report any major side effects, but assessments were made based on short-term use. Serious pulmonary side effects could be possible with chronic use of PHD inhibitors.
For MCT (monocarboxylate transporter) inhibitors
The important roles exerted by MCTs in physiology call for attention on possible toxicities associated with MCT inhibitors.
In genetically engineered mouse models, a full knockout of MCT1 was found to be embryonically lethal due to neuronal defects [205]. Comparatively, a systemic MCT1 genotype and an oligodendrocyte-selective MCT1 knockdown produced living mice, but these animals had impaired axon myelination, leading to axon damage and decreased neuron survival in the central nervous system. The regeneration of motor and sensory peripheral nerves after a lesion was also delayed in MCT1 knockdown mice. These results are consistent with the decreased expression of MCT1 observed in neurodegenerative human diseases, such as amyotrophic lateral sclerosis (ALS) and Alzheimer's disease suggesting an important role of this transporter in the maintenance of axon integrity, putatively because it facilitates lactate shuttles between oligodendrocytes and neurons.
In the brain, MCT2 is preferentially expressed in neurons where it conveys lactate uptake Adult rats injected with antisense oligonucleotides in the hippocampus showed memory defects. MCT2-deficiency did not alter short-term memory but significantly disrupted long-term memory. Neither glucose nor lactate rescued amnesia, indicating that processes dependent on MCT2 are essential for long-term memory. Accordingly, MCT2 expression was found to be decreased in animal models of Alzheimer's disease.
In eyes, MCT3 facilitates lactate export by the retina. It is therefore not surprising that MCT3 knockout mice developed visual defects. They were attributed to a decrease in photoreceptor currents in response to light and associated to a 4-fold increase in lactate levels in the retina and, possibly, acidification of the subretinal space. However, histological features of the eyes were preserved.
In humans, genetic polymorphisms of MCT1 impact the oxidative clearance of lactate by slow-twitching muscle fibers, with certain variants showing poorer lactate clearance during high intensity exercise.
Novel MCT1 mutations (either homozygous or heterozygous) have been identified in several patients. These resulted in recurrent and severe episodes of keto-acidosis, i.e., accumulation of ketone bodies in the blood due to an imbalance between their production in the liver and their use in peripheral tissues, possibly resulting from a decreased uptake capacity of ketone bodies by MCT1-deficient cells. Thus, keto-acidosis is important to consider upon therapeutic MCT1 inhibition as well.
For quinacridone pigments
It is considered unlikely that the quinacridone pigments of this category become systemically bioavailable after dermal or inhalation exposure.

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

Worker DNELs for acute exposure - local effects are not derived, because quinacridone pigments of this category have not to be classified as irritating to skin or eyes, are considered unlikely to become bioavailable in the skin and are considered not to be classified regarding respiratory tract irritation. Finally, there is no established accepted methodology for the derivation of acute toxicity DNELs existing. Apart from that, relevant occupational exposure limits for inert dusts should be applied.

Repeat dose toxicity

The toxicity of the test item, C.I. Pigment Red 122, when given by oral administration (gavage) to rats for 13 consecutive weeks at dosages of 50, 200 or 1000 mg/kg/day, and recovery from any treatment-related effects over a recovery period of 4 weeks, has been investigated. No toxicologically relevant changes were observed during the in vivo phase or at the post mortem examinations.

On the basis of these results, it could be concluded that the No Observed Adverse Effect Level (NOAEL) in this study was 1000 mg/kg/day.

Liver and blood plasma samples of male and female rats of the 1000 mg/kg bw/day group collected at the end of the exposure period were below quantifiable limit concentrations of 1.5 ug/g dried liver and 0.4 / 0.6 ppm dried blood plasma.

Genetic toxicity:

Mutagenic activity of the test item was investigated in Salmonella typhimurium strains TA 1535, TA 1537, TA98, TA100 and Escherichia coli strain WP2uvrA with (induced rat liver S9 mix) and without metabolic activation at concentrations of 3, 10, 33, 100, 333, 1000, 2500, and 5000 µg/plate using the plate incorporation assay. Additionally, a preincubation assay with or without metabolic activation was performed using the concentrations 33, 100, 333, 1000, 2500, and 5000 µg/plate.

The test item did not reveal any mutagenic activity under the conditions tested.

The test item is not mutagenic in the micronucleus test.

Toxicokinetics

In one toxicokinetic study, the radiolabelled test item (Pigment Violet 19) was administered orally to groups of male and female Fisher 344 rats by gavage. The tissue distribution of radioactivity was determined by whole body autoradiography at selected times up to 48 hours after dosing. The autoradiogram showed that radioactivity was localized only in the gastrointestinal tract of both male and female rats. No radioactivity was detected in other organs and tissues of the animals. The highest concentrations of radioactivity were found at 2 hours post dosing. Most of the radioactivity was eliminated from the rats at 24 hours and it was virtually undetected at 18 hours post-dose.

For silica amorphous:

Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d.

In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.

When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body.

Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.

After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.

Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.

Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact.

Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure.

Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m³ to 150 mg/m³. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m³. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m³. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL.

Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.

For Synthetic Amorphous Silica (SAS)

Repeated dose toxicity

Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet.

Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) = 1.3 mg/m³ based on mild reversible effects in the lungs.

Inhalation (rat), 90 days, LOEL = 1 mg/m³ based on reversible effects in the lungs and effects in the nasal cavity.

For silane treated synthetic amorphous silica:

Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested.

There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.

**BYRON MATTE ACRYLIC
PAINT PRIMARY COLOURS &
SILICA AMORPHOUS**

**BYRON MATTE ACRYLIC
PAINT PRIMARY COLOURS &
STYRENE/ ACRYLIC ACID
COPOLYMER &
HYDROXYETHYLCELLULOSE
& ALCOHOLS C12-14
SECONDARY ETHOXYLATED**

No significant acute toxicological data identified in literature search.

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

<p>& TITANIUM DIOXIDE & C.I. PIGMENT GREEN 7 & CARBON BLACK & C.I. PIGMENT YELLOW 43 & FERRIC HYDROXIDE & C.I. PIGMENT RED 21 & UMBER BROWN OXIDE & C.I. PIGMENT BLACK 11 & WATER</p>	
<p>BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS & PROPYLENE GLYCOL</p>	<p>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.</p> <p>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.</p> <p>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).</p> <p>Propylene glycol shows no evidence of being a carcinogen or of being genotoxic.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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)</p> <p>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.</p>
<p>BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS & C.I. PIGMENT ORANGE 13</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 acetoacetarylides 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 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.</p> <p>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.</p> <p>For acute dermal toxicity a single LD50 of >3,000 mg/kg bw is available for Pigment Yellow 13. No deaths or clinical signs of toxicity were observed following oral or dermal exposure. The inhalation LC50 available is >4,448 mg/m3 for Pigment Yellow</p>

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

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 sensitizers

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/m³ (local LOAEL). Systemically no effects were observed at the highest concentration tested, 410 mg/m³ (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/m³) 2 hours per day for 7 days. In another study, 10 rats were exposed to an unspecified concentration of 3,3 -dichlorobenzidine dihydrochloride dust particles for 1 hour and then observed for 14 days. Slight-to-moderate pulmonary congestion and one pulmonary abscess were observed upon necropsy. The effects observed in the study using the ionized (hydrochloride) form of 3,3 -dichlorobenzidine may have been due to the irritative properties of hydrochloric acid released from the salt in combination with particulate toxicity.

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 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 21 controls). Hepatic tumors were observed in 4/18 strain D mice exposed to 11.2-1.9 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

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

(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.

**BYRON MATTE ACRYLIC
PAINT PRIMARY COLOURS &
HYDROCARBON OILS**

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

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 LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (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 NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m³ and for systemic effects NOAEL > 980 mg/m³.

Sub-chronic toxicity

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

Repeat dose toxicity:

Oral

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

Inhalation

The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m³. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m³.

Dermal

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

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 NOAEL 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 NOAEL for this study is =1000 mg/kg/day and no LOAEL 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.

<p>BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS & TITANIUM DIOXIDE & CARBON BLACK</p>	<p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
<p>BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS & CALCIUM CARBONATE</p>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p>BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS & CALCIUM CARBONATE & PROPYLENE GLYCOL & 2-BROMO-2-NITROPROPAN-1,3-DIOL</p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

<p>BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS & C.I. PIGMENT YELLOW 3 & C.I. PIGMENT RED 8</p>	<p>▶ 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.</p> <p>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.</p> <p>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.</p> <p>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</p> <ul style="list-style-type: none"> ▶ 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. <p>The aromatic amines containing moieties of anilines, extended anilines and fused ring amines are components of the majority of the industrially important azo dyes.</p> <p>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.</p> <p>There are now epidemiological indications that occupational exposure to benzidine-based azo colourants can increase the incidence of bladder carcinoma.</p> <p>The acute toxicity of azo dyes is low.. However, potential health effects are recognised.</p> <p>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.</p>
<p>SILICA AMORPHOUS & UMBER BROWN OXIDE</p>	<p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
<p>2,2,4-TRIMETHYL-1,3-PENTANEDIOL MONOISOBUTYRATE & HYDROCARBON OILS</p>	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p>2,2,4-TRIMETHYL-1,3-PENTANEDIOL MONOISOBUTYRATE & HYDROCARBON OILS & TITANIUM DIOXIDE</p>	<p>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.</p>
<p>UMBER BROWN OXIDE & C.I. PIGMENT BLACK 11</p>	<p>No data of toxicological significance identified in literature search.</p>

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

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

SECTION 12 Ecological information

Toxicity

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
calcium carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	1h	Fish	4-320mg/l	4
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	LC50	96h	Fish	>165200mg/L	4

Continued...

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

styrene/ acrylic acid copolymer	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
propylene glycol	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	336h	Algae or other aquatic plants	<5300mg/l	1
	EC50	96h	Algae or other aquatic plants	19000mg/l	2
	EC50	72h	Algae or other aquatic plants	19300mg/l	2
	EC50	48h	Crustacea	>114.4mg/L	4
silica amorphous	Endpoint	Test Duration (hr)	Species	Value	Source
	EC0(ECx)	24h	Crustacea	>=10000mg/l	1
	EC50	72h	Algae or other aquatic plants	14.1mg/l	2
	EC50	96h	Algae or other aquatic plants	217.576mg/l	2
	EC50	48h	Crustacea	>86mg/l	2
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	15mg/l	Not Available
	NOEC(ECx)	72h	Algae or other aquatic plants	3.28mg/l	1
	LC50	96h	Fish	16mg/l	Not Available
	EC50	48h	Crustacea	>19mg/l	2
monoisobutanolamine	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	100mg/l	1
	EC50	72h	Algae or other aquatic plants	>103mg/l	2
	EC50	48h	Crustacea	193mg/l	1
	EC0(ECx)	48h	Crustacea	100mg/l	1
hydrocarbon oils	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
alcohols C12-14 secondary ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	72h	Algae or other aquatic plants	0.013mg/l	2
	EC50	72h	Algae or other aquatic plants	0.026mg/l	2
2-bromo-2-nitropropan-1,3-diol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	0.02-0.025mg/L	4
	EC50	48h	Crustacea	1.1-3.52mg/L	4
	LC50	96h	Fish	10.274-14.454mg/L	4
	Endpoint	Test Duration (hr)	Species	Value	Source
titanium dioxide	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	1.85-3.06mg/l	4
	BCF	1008h	Fish	<1.1-9.6	7
EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4	

Continued...

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

	EC50	48h	Crustacea	1.9mg/l	2
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
C.I. Pigment Green 7	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>100mg/l	2
	BCF	1008h	Fish	0.51-4.8	7
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	153.6mg/l	2
	NOEC(ECx)	504h	Crustacea	>=1mg/l	2
C.I. Pigment Yellow 1	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>1mg/l	2
	EC0(ECx)	96h	Fish	1mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
C.I. Pigment Yellow 3	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>1mg/l	2
	EC0(ECx)	96h	Fish	1mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
carbon black	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	EC50	48h	Crustacea	33.076-41.968mg/l	4
	NOEC(ECx)	24h	Crustacea	3200mg/l	1
C.I. Pigment Red 146	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Pigment Blue 29	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	000mg/l	Not Available
C.I. Pigment Blue 15	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>100mg/l	2
	BCF	1008h	Fish	<0.33-11	7
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>500mg/l	2
	EC50(ECx)	504h	Crustacea	>1mg/l	2
C.I. Pigment Blue 1	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.1-1mg/l	Not Available
	EC50(ECx)	48h	Crustacea	1mg/l	Not Available
	EC50	48h	Crustacea	1mg/l	Not Available
C.I. Pigment Yellow 43	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
ferric hydroxide	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.05mg/l	2
	EC50	72h	Algae or other aquatic plants	18mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	504h	Fish	0.52mg/l	2
	NOEC(ECx)	504h	Fish	0.52mg/l	2

Continued...

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

	EC50	72h	Algae or other aquatic plants	18mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	0.05mg/l	2
red iron oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.05mg/l	2
	EC50	72h	Algae or other aquatic plants	18mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	504h	Fish	0.52mg/l	2
C.I. Pigment Red 112	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Pigment Red 21	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Pigment Orange 13	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	0.75-5.6	7
	LC50	96h	Fish	>500mg/l	2
	NOEC(ECx)	504h	Crustacea	1mg/l	2
umber brown oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Pigment Red 8	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Pigment Red 122	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	>0.02mg/l	2
	EC50	72h	Algae or other aquatic plants	>10mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
C.I. Pigment Black 11	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.05mg/l	2
	EC50	72h	Algae or other aquatic plants	18mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
umber brown oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
water	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene glycol	LOW	LOW
silica amorphous	LOW	LOW

Continued...

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

Ingredient	Persistence: Water/Soil	Persistence: Air
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	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)
silica amorphous	LOW (LogKOW = 0.5294)
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	LOW (LogKOW = 2.9966)
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)
silica amorphous	LOW (Log KOC = 23.74)
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	LOW (Log KOC = 22.28)
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 = 10000000000)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▶ 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.
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Continued...

BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

- ▶ 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

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

14.1. UN number or ID number	3082	
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains C.I. Pigment Blue 1)	
14.3. Transport hazard class(es)	Class	9
	Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	274 331 335 375 AU01
	Limited quantity	5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in;

- (a) packagings;
 - (b) IBCs; or
 - (c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

14.1. UN number	3082	
14.2. UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. (contains C.I. Pigment Blue 1)	
14.3. Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	9L
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	A97 A158 A197 A215
	Cargo Only Packing Instructions	964
	Cargo Only Maximum Qty / Pack	450 L
	Passenger and Cargo Packing Instructions	964
	Passenger and Cargo Maximum Qty / Pack	450 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y964
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3082
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BYRON MATTE ACRYLIC PAINT PRIMARY COLOURS

14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains C.I. Pigment Blue 1)	
14.3. Transport hazard class(es)	IMDG Class	9
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5 Environmental hazard	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-A , S-F
	Special provisions	274 335 969
	Limited Quantities	5 L

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
styrene/ acrylic acid copolymer	Not Available
propylene glycol	Not Available
silica amorphous	Not Available
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Not Available
monoisobutanolamine	Not Available
hydrocarbon oils	Not Available
hydroxyethylcellulose	Not Available
alcohols C12-14 secondary ethoxylated	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
carbon black	Not Available
C.I. Pigment Red 146	Not Available
C.I. Pigment Blue 29	Not Available
C.I. Pigment Blue 15	Not Available
C.I. Pigment Blue 1	Not Available
C.I. Pigment Yellow 43	Not Available
ferric hydroxide	Not Available
red iron oxide	Not Available
C.I. Pigment Red 112	Not Available
C.I. Pigment Red 21	Not Available
C.I. Pigment Orange 13	Not Available
umber brown oxide	Not Available
C.I. Pigment Red 8	Not Available
C.I. Pigment Red 122	Not Available
C.I. Pigment Black 11	Not Available
umber brown oxide	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
styrene/ acrylic acid	Not Available

Continued...

Product name	Ship Type
copolymer	
propylene glycol	Not Available
silica amorphous	Not Available
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Not Available
monoisobutanolamine	Not Available
hydrocarbon oils	Not Available
hydroxyethylcellulose	Not Available
alcohols C12-14 secondary ethoxylated	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
carbon black	Not Available
C.I. Pigment Red 146	Not Available
C.I. Pigment Blue 29	Not Available
C.I. Pigment Blue 15	Not Available
C.I. Pigment Blue 1	Not Available
C.I. Pigment Yellow 43	Not Available
ferric hydroxide	Not Available
red iron oxide	Not Available
C.I. Pigment Red 112	Not Available
C.I. Pigment Red 21	Not Available
C.I. Pigment Orange 13	Not Available
umber brown oxide	Not Available
C.I. Pigment Red 8	Not Available
C.I. Pigment Red 122	Not Available
C.I. Pigment Black 11	Not Available
umber brown oxide	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)

styrene/ acrylic acid 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)

silica amorphous is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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

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

2,2,4-trimethyl-1,3-pentanediol monoisobutyrate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Continued...

monoisobutanolamine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
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

hydroxyethylcellulose is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

alcohols C12-14 secondary ethoxylated is found on the following regulatory lists

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

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)

carbon black 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 146 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 Blue 29 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 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 Blue 1 is found on the following regulatory lists

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

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment Yellow 43 is found on the following regulatory lists

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

ferric hydroxide is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

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

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

red iron oxide 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 6

Australian Inventory of Industrial Chemicals (AIIC)

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

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

C.I. Pigment Red 112 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)

umber brown oxide 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 8 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment Red 122 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 Black 11 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)

umber brown oxide 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)

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 Yellow 43; C.I. Pigment Red 21)
Canada - DSL	No (C.I. Pigment Yellow 43; C.I. Pigment Red 21; umber brown oxide; umber brown oxide)

Continued...

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National Inventory	Status
Canada - NDSL	No (styrene/ acrylic acid copolymer; propylene glycol; 2,2,4-trimethyl-1,3-pentanediol monoisobutyrate; monoisobutanolamine; hydrocarbon oils; hydroxyethylcellulose; alcohols C12-14 secondary ethoxylated; 2-bromo-2-nitropropan-1,3-diol; C.I. Pigment Green 7; C.I. Pigment Yellow 1; C.I. Pigment Yellow 3; carbon black; C.I. Pigment Red 146; C.I. Pigment Blue 29; C.I. Pigment Blue 15; C.I. Pigment Blue 1; C.I. Pigment Yellow 43; red iron oxide; C.I. Pigment Red 112; C.I. Pigment Orange 13; C.I. Pigment Red 8; C.I. Pigment Red 122; C.I. Pigment Black 11; water)
China - IECSC	No (C.I. Pigment Red 21)
Europe - EINEC / ELINCS / NLP	No (styrene/ acrylic acid copolymer; hydrocarbon oils; hydroxyethylcellulose; alcohols C12-14 secondary ethoxylated; C.I. Pigment Yellow 43)
Japan - ENCS	No (umber brown oxide; umber brown oxide)
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; C.I. Pigment Yellow 43)
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 146; C.I. Pigment Blue 1; C.I. Pigment Yellow 43; C.I. Pigment Red 21; umber brown oxide; C.I. Pigment Red 8; C.I. Pigment Red 122; umber brown oxide)
Vietnam - NCI	Yes
Russia - FBEPH	No (hydrocarbon oils; alcohols C12-14 secondary ethoxylated; C.I. Pigment Red 146; C.I. Pigment Blue 1; C.I. Pigment Yellow 43; C.I. Pigment Red 21; umber brown oxide; C.I. Pigment Red 8; umber brown oxide)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	15/04/2024
Initial Date	15/04/2024

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
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals

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- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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