KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB CLASSIC PACK 12

Jasco Pty Limited

Chemwatch Hazard Alert Code: **3**

Chemwatch: **9659-83**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **27/10/2026**
Revision Date: **27/10/2026**
Print Date: **05/11/2026**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB CLASSIC PACK 12
Chemical Name	Not Applicable
Synonyms	ONYX; SNOW; BUTTERCUP; PACIFIC BLUE; RUBY RED; EMERALD; TANGERINE; BUBBLEGUM; LAGOON; IRIS; COFFEE; PEWTER
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Pen. Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

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

Emergency telephone number

Association / Organisation	**Australian Poisons Centre**	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone number(s)	**13 11 26 (24/7)**	**+61 1800 951 288 (ID#: 9659-83)**
Other emergency telephone number(s)	**Not Available**	**+61 3 9573 3188**

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Germ Cell Mutagenicity Category 2, Carcinogenicity Category 1B, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 4
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

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

Hazard statement(s)

Page 1 continued...

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H373	May cause damage to organs through prolonged or repeated exposure.
H402	Harmful to aquatic life.
H413	May cause long lasting harmful effects to aquatic life.

Precautionary statement(s) Prevention

P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
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.

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
7732-18-5	<=74	water
13463-67-7	30	titanium dioxide
56-81-5	<=13.46	glycerol
57-55-6	<=10.38	propylene glycol
1333-86-4	<=3.07	carbon black
55406-53-6	0.02	3-iodo-2-propynyl butyl carbamate
5567-15-7	<=1.88	C.I. Pigment Yellow 83
215247-95-3	<=1.3	C.I. Pigment Violet 23
1328-53-6	<=1.33	C.I. Pigment Green 7
2650-18-2	<=1.83	C.I. Acid Blue 9
980-26-7	<=3.53	C.I. Pigment Red 122
2786-76-7	<=1.31	C.I. Pigment Red 170
52-51-7	<=0.05	2-bromo-2-nitropropan-1,3-diol
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	

Continued...

Skin Contact	
Inhalation	
Ingestion	

Indication of any immediate medical attention and special treatment needed

SECTION 5 Firefighting measures

Extinguishing media

Special hazards arising from the substance or mixture

Fire Incompatibility	

Advice for firefighters

Fire Fighting	
Fire/Explosion Hazard	
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	
Major Spills	

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	
Other information	

Conditions for safe storage, including any incompatibilities

Suitable container	
Storage incompatibility	

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	glycerol	Glycerin mist	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	glycerol	Glycerin mist	10 mg/m3	Not Available	Not Available	Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	propylene glycol	Propane-1,2-diol 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

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	propylene glycol	Propane-1,2-diol total (vapour and particulates)	50 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Workers exposed to this chemical may require specific health monitoring (see regulations 368-378, Schedule 14 to the model WHS Regulations).

MATERIAL DATA

Exposure controls

Appropriate engineering controls	
Individual protection measures, such as personal protective equipment	
Eye and face protection	
Skin protection	See Hand protection below
Hands/feet protection	
Body protection	See Other protection below
Other protection	

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
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
VITON	C

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

Ansell Glove Selection

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

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P3	-	A-PAPR-AUS / Class 1 P3
up to 50 x ES	-	A-AUS / Class 1 P3	-
up to 100 x ES	-	A-2 P3	A-PAPR-2 P3 ^

^ - 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)

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured liquid.

Physical state	Not Available	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**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 (mm2/s)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**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
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	
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

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

Inhaled	
Ingestion	
Skin Contact	
Eye	
Chronic	

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KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB CLASSIC PACK 12	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
water	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >90000 mg/kg[2]	Not Available
titanium dioxide	**TOXICITY**	**IRRITATION**
	dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >2.28 mg/l4h[1]	Skin (Human): 300ug/3D - Mild
	Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
glycerol	**TOXICITY**	**IRRITATION**
	Dermal (Guinea Pig) LD50: 58500 mg/kg[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Inhalation (Rat) LC50: >5.85 mg/L4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; 4090 mg/kg[2]	Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating)[1]
propylene glycol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 11890 mg/kg[2]	Eye (Rodent - rabbit): 100mg - Mild
	Inhalation (Rat) LC50: >44.9 mg/l4h[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Oral (Rat) LD50: 20000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (Human - child): 30%/96H - Moderate
		Skin (Human - man): 10%/2D
		Skin (Human - woman): 30%/96H - Mild
		Skin (Human): 104mg/3D - Moderate
		Skin (Human): 20%
		Skin (Human): 500mg/7D - Mild
		Skin: no adverse effect observed (not irritating)[1]
carbon black	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
3-iodo-2-propynyl butyl carbamate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye: adverse effect observed (irreversible damage)[1]
	Inhalation (Rat) LC50: 0.63 mg/l4h[1]	Skin (Human): 0.3%/48H
	Oral (Rat) LD50: 1056 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Yellow 83	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >3000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >0.23 mg/L4h[1]	Skin (Rodent - rabbit): 500mg - Mild
	Oral (Mouse) LD50; >5000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Violet 23	**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 Green 7	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >1.084<5.212 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; 8400 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
C.I. Acid Blue 9	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >2000 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]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

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C.I. Pigment Red 170	**TOXICITY** / **IRRITATION** dermal (rat) LD50: >2000 mg/kg[1] / Eye: no adverse effect observed (not irritating)[1] Inhalation (Rat) LC50: >1.58 mg/L4h[1] / Skin: no adverse effect observed (not irritating)[1] Oral (Rat) LD50: >10000 mg/kg[2]
2-bromo-2-nitropropan-1,3-diol	**TOXICITY** / **IRRITATION** dermal (rat) LD50: ~1600 mg/kg[1] / Eye (Rodent - rabbit): 5mg Inhalation (Rat) LC50: >0.12<1.14 mg/l4h[1] / Eye: adverse effect observed (irreversible damage)[1] Oral (Rat) LD50: 180 mg/kg[2] / Skin (Human): 10mg - Moderate Skin (Rodent - rabbit): 500mg/24H - Mild Skin (Rodent - rabbit): 80mg - Moderate Skin (Rodent - rat): 0.2% Skin: adverse effect observed (irritating)[1]

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***

TITANIUM DIOXIDE	* IUCLID Laboratory (in vitro) and animal studies show, exposure to the material may result in a possible risk of irreversible effects, with the possibility of producing mutation. For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the 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. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
GLYCEROL	For glycerol: **Acute toxicity:** Glycerol is of a low order of acute oral and dermal toxicity with LD50 values in excess of 4000 mg/kg bw. At very high dose levels, the signs of toxicity include tremor and hyperaemia of the gastro-intestinal -tract. Skin and eye irritation studies indicate that glycerol has low potential to irritate the skin and the eye. The available human and animal data, together with the very widespread potential for exposure and the absence of case reports of sensitisation, indicate that glycerol is not a skin sensitiser. **Repeat dose toxicity:** Repeated oral exposure to glycerol does not induce adverse effects other than local irritation of the gastro-intestinal tract. The overall NOEL after prolonged treatment with glycerol is 10,000 mg/kg bw/day (20% in diet). At this dose level no systemic or local effects were observed. For inhalation exposure to aerosols, the NOAEC for local irritant effects to the upper respiratory tract is 165 mg/m3 and 662 mg/m3 for systemic effects. **Genotoxicity:** Glycerol is free from structural alerts, which raise concern for mutagenicity. Glycerol does not induce gene mutations in bacterial strains, chromosomal effects in mammalian cells or primary DNA damage ***in vitro***. Results of a limited gene mutation test in mammalian cells were of uncertain biological relevance. ***In vivo***, glycerol produced no statistically significant effect in a chromosome

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	aberrations and dominant lethal study. However, the limited details provided and the absence of a positive control, prevent any reliable conclusions to be drawn from the ***in vivo*** data. Overall, glycerol is not considered to possess genotoxic potential. **Carcinogenicity:** The experimental data from a limited 2 year dietary study in the rat does not provide any basis for concerns in relation to carcinogenicity. Data from non-guideline studies designed to investigate tumour promotion activity in male mice suggest that oral administration of glycerol up to 20 weeks had a weak promotion effect on the incidence of tumour formation. **Reproductive and developmental toxicity:** No effects on fertility and reproductive performance were observed in a two generation study with glycerol administered by gavage (NOAEL 2000 mg/kg bw/day). No maternal toxicity or teratogenic effects were seen in the rat, mouse or rabbit at the highest dose levels tested in a guideline comparable teratogenicity study (NOEL 1180 mg/kg bw/day).
PROPYLENE GLYCOL	The acute oral toxicity of propylene glycol is very low, and large quantities are required to cause perceptible health damage in humans. Serious toxicity generally occurs only at plasma concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time. It would be nearly impossible to reach toxic levels by consuming foods or supplements, which contain at most 1 g/kg of PG. Cases of propylene glycol poisoning are usually related to either inappropriate intravenous administration or accidental ingestion of large quantities by children.The potential for long-term oral toxicity is also low. Because of its low chronic oral toxicity, propylene glycol was classified by the U. S. Food and Drug Administration as "generally recognized as safe" (GRAS) for use as a direct food additive. Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce slight transient conjunctivitis (the eye recovers after the exposure is removed). Exposure to mists may cause eye irritation, as well as upper respiratory tract irritation. Inhalation of the propylene glycol vapours appears to present no significant hazard in ordinary applications. However, limited human experience indicates that inhalation of propylene glycol mists could be irritating to some individuals It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations. Propylene glycol is metabolised in the human body into pyruvic acid (a normal part of the glucose-metabolism process, readily converted to energy), acetic acid (handled by ethanol-metabolism), lactic acid (a normal acid generally abundant during digestion), and propionaldehyde (a potentially hazardous substance). Propylene glycol shows no evidence of being a carcinogen or of being genotoxic. Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but that they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis to propylene glycol may be greater than 2% in patients with eczema. One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic reactions, such as rhinitis or hives in children Another study suggested that the concentrations of PGEs (counted as the sum of propylene glycol and glycol ethers) in indoor air, particularly bedroom air, is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema, and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use of water-based paints and water-based system cleansers. Patients with vulvodynia and interstitial cystitis may be especially sensitive to propylene glycol. Women suffering with yeast infections may also notice that some over the counter creams can cause intense burning. Post menopausal women who require the use of an eostrogen cream may notice that brand name creams made with propylene glycol often create extreme, uncomfortable burning along the vulva and perianal area. Additionally, some electronic cigarette users who inhale propylene glycol vapor may experience dryness of the throat or shortness of breath . As an alternative, some suppliers will put Vegetable Glycerin in the "e-liquid" for those who are allergic (or have bad reactions) to propylene glycol. Adverse responses to intravenous administration of drugs which use PG as an excipient have been seen in a number of people, particularly with large dosages thereof. Responses may include "hypotension, bradycardia... QRS and T abnormalities on the ECG, arrhythmia, cardiac arrest, serum hyperosmolality, lactic acidosis, and haemolysis". A high percentage (12% to 42%) of directly-injected propylene glycol is eliminated/secreted in urine unaltered depending on dosage, with the remainder appearing in its glucuronide-form. The speed of renal filtration decreases as dosage increases, which may be due to propylene glycol's mild anesthetic / CNS-depressant -properties as an alcohol. In one case, intravenous administration of propylene glycol-suspended nitroglycerin to an elderly man may have induced coma and acidosis. Propylene glycol is an approved food additive for dog food under the category of animal feed and is generally recognized as safe for dogs with an LD50 of 9 mL/kg. The LD50 is higher for most laboratory animals (20 mL/kg) Similarly, propylene glycol is an approved food additive for human food as well. The exception is that it is prohibited for use in food for cats due to links to Heinz body anemia.
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported
3-IODO-2-PROPYNYL BUTYL CARBAMATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. for carbamates: Carbamates are effective insecticides by virtue of their ability to inhibit acetylcholinesterase (AChE) (EC 3.1.1.7) in the nervous system. They can also inhibit other esterases. The carbamylation of the enzyme is unstable, and the regeneration of AChE is relatively rapid compared with that from a phosphorylated enzyme. Thus, carbamate pesticides are less dangerous with regard to human exposure than organophosphorus pesticides. The ratio between the dose required to produce death and the dose required to produce minimum symptoms of poisoning is substantially larger for carbamate compounds than for organophosphorus compounds. A dose-effect relationship exists between the dose, the severity of symptoms, and the degree of cholinesterase (ChE) inhibition. Because most carbamates have a low volatility, inhalation studies are mainly carried out using a dust or mist. In these studies, the toxicity is highly dependent on the size of the particles or droplets and, therefore, difficult to evaluate. The acute dermal toxicity of carbamates is generally low to moderate. From controlled human studies, it is clear that poisoning symptoms can be seen a few minutes after exposure, and can last for a few hours. Thereafter, recovery starts and within hours, the symptoms disappear, and the ChE activity in erythrocytes and plasma returns to normal, because the carbamate is rather rapidly metabolised and the metabolites excreted. The appearance of these metabolites in the urine may be used for biological monitoring. Apart from the symptoms indicative of ChE poisoning, other signs and symptoms induced by certain carbamates have been described, such as skin and eye irritation, hyperpigmentation, and influence on the function of testes (slight increase of sperm abnormalities). These signs and symptoms were found in a few studies and should be confirmed before it can be stated that they were induced by carbamates. Epidemiological studies with persons primarily exposed to carbamates are not available. Carbamates produce slight to moderate skin and eye irritation, depending on the vehicle used, duration of contact, and on whether the substance is applied to the abraded or intact skin. From the available data, it cannot be excluded that some of the carbamates will have a slight to moderate sensitization potential. Short- and long-term toxicity studies have been carried out. Some carbamates are very toxic and others are less toxic in long- term studies. From these studies, it is evident that, apart from the anticholinesterase activity, the following changes can be found: an influence on the haemopoietic system, an influence on the functioning of, and, at higher dosages, degeneration of, the liver and kidneys, and degeneration of testes. These abnormalities in the different organ systems depend on the animal strain and on the chemical structure of the carbamate. A clear influence on the nervous system, functional as well as histological, was found, particularly in non-laboratory animals such as pigs. A considerable number of reproduction and teratogenicity studies have been carried out with different carbamates and various animal species. Different types of abnormalities were found, i.e., increase in mortality, disturbance of the endocrine system, and effects on the hypophysis and its gonadotrophic function. These effects were mainly seen at high dose levels. Generally, the fetal effects included an increase in mortality, decreased weight gain in the first weeks after birth, and induction of early embryonic death. All these effects can be summarized as embryotoxic effects. Certain carbamates also induce teratogenic effects, mainly at high dose levels applied by stomach tube. When the same dose level was administered with the diet, no effects were seen.

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	Some carbamates induce mutagenic effects, others are negative. In general, the methyl carbamates are negative in mammalian tests, while compounds such as carbendazim, benomyl, and the 2 thiophanate derivatives showed a positive effect with very high dose levels in certain systems. The benzimidazole moiety may act as a base analogue for DNA and as a spindle poison. They are antimitotic agents and cause mitotic arrest, mitotic delay, and a low incidence of chromosome damage. Sometimes, the results are contradictory or cannot be reproduced, but positive results for point mutation and chromosome aberrations are well documented. These benzimidazole derivatives can be considered as weak mutagenic compounds. Carcinogenicity studies with benzimidazole derivatives showed either positive or equivocal results. Added to the fact that certain mutagenicity studies also give positive results, it cannot be excluded that these compounds may have carcinogenic or promotor properties. Carbamate pesticides may be converted to ***N*** -nitroso compounds. This was demonstrated in a great number of ***in vivo*** nitrosation studies in which high levels of the carbamates were administered to animals in combination with high levels of nitrite. These ***N*** - nitroso compounds have to be considered as mutagenic and carcinogenic. However, the amount of nitroso compounds that can be expected to result from dietary intake of carbamate pesticide residues is negligible in comparison with nitroso-precursors that occur naturally in food and drinking-water. The metabolic fate of carbamates is basically the same in plants, insects, and mammals. Carbamates are usually easily absorbed through the skin, mucous membranes, and respiratory and gastrointestinal tracts, but there are exceptions. Generally, the metabolites are less toxic than the parent compounds. However, in certain cases, the metabolites are just as toxic or even more toxic than the parent carbamate. In most mammals, the metabolites are mainly excreted rather rapidly in the urine. The dog seems to be different in this respect. Accumulation takes place in certain cases, but is of minor importance because of the rapid metabolism. The first step in the metabolism of carbamates is hydrolysis to carbamic acid, which decomposes to carbon dioxide (CO_2) and the corresponding amine. The rate of hydrolysis by esterases is faster in mammals than in plants and insects. The organs in which residues have been reported are the liver, kidneys, brain, fat, and muscle. The half-life in the rat is of the order of 3 - 8 h. From the limited data available, it seems that the excretion of carbamates via urine is also rapid in man, and that the metabolic pathways in man are the same as those in the rat for 3-iodo-2-propynyl butyl carbamate (IPBC): **Acute toxicity:** Acceptable acute toxicity studies with IPBC indicate low toxicity except eye irritation. In a primary eye irritation study in rabbi. IPBC technical was severely irritating to the eyes of white rabbits, with corneal opacity and corneal vascularization reported in unwashed eyes by day 21 post-treatment. The technical grade of IPBC was slightly irritating to the skin of white rabbits. In a dermal sensitization study in Guinea pigs IPBC technical, at a concentration of 0.32%, produced no evidence of sensitization in male and female Guinea pigs. **Subchronic toxicity:** In a subchronic oral toxicity study, male and female Sprague-Dawley rats received IPBC technical by gavage for 13 weeks at doses of 0, 20, 50, and 125 mg/kg/day. At the 125 mg/kg/day dose level, body weight gain was decreased by 19% in male rats for weeks 1-13 of the study, and by 12% in female rats over the same period. Absolute liver weight was increased by 20% in male rats at the 125 mg/kg/day dose, and by 31% in female rats at this dose level. Liver to body weight ratio was significantly increased by approximately 31% in both male and female rats at the 125 mg/kg/day dose level, while kidney to body weight ratio in female rats was increased 18% at the 125 mg/kg/day dose level. The systemic NOEL was considered to be 20 mg/kg/day, while the systemic LEL was considered to be 50 mg/kg/day, based on increased liver to body weight ratio. In a subchronic dermal toxicity study, male and female Sprague-Dawley rats (10/sex/dose) received dermal doses of 50, 200, and 500 mg/kg/day IPBC technical grade (97.5%) to the shaved skin for five days a week, six hours per day. At the 500 mg/kg/day dose, decreased body weight (4-6%) and weight gain (11%) were observed in male rats, but not in female rats. In female rats, significant increases in haemoglobin, haematocrit, and eosinophils were observed at the 500 mg/kg/day dose level. Reticulocytes as a percentage of red cells were decreased in the 50 and 200 mg/kg/day dose groups but not at the 500 mg/kg/day dose level. Females in this study showed inhibition of plasma cholinesterase at 500 mg/k/day test article, which may have been the result of either direct liver toxicity or inhibition of cholinesterase itself. Based upon the results of this study, the systemic NOEL is 200 mg/kg/day, the systemic LEL is 500 mg/kg/day for male and female rats. **Carcinogenicity:** In a 2-year chronic toxicity/carcinogenicity study, technical grade IPBC (98.68% ai) was administered to male and female Sprague Dawley rats (50/sex/group) at dose levels of 0, 20, 40, and 80 mg/kg/day. There were no statistically significant increases in tumor incidences in male rats. The incidence of mammary gland fibroadenoma and combined fibroadenoma/carcinoma in female rats was significantly increased at the 20 mg/kg/day dose level but there was no dose-related trend. **Developmental and reproductive toxicity:** The developmental toxicity of IPBC was assessed in pregnant Sprague-Dawley rats on gestation days six through 15 by oral administration of the test chemical at doses of 0, 20, 50, and 125 mg/kg/day. Maternal toxicity as reduced body weight gain during dosing was observed at the 125 mg/kg/day dose level. Developmental toxicity consisted of an increased incidence of skeletal abnormalities at the 125 mg/kg/day dose level. The maternal toxicity NOEL was determined to be 50 mg/kg/day, and the maternal toxicity LEL was determined to be 125 mg/kg/day, based on reduced body weight gain. The developmental toxicity NOEL was determined to be 50 mg/kg/day, and the developmental toxicity LEL was determined to be 125 mg/kg/day, based on incompletely ossified frontal skull bones and pelvic girdles. A 2-generation reproductive toxicity study was conducted in male and female Sprague- Dawley rats. IPBC technical was administered over two generations at doses of 0, 120, 300, and 750 ppm (0, 6, 15, and 37.5 mg/kg/day). Reduced body weight and food consumption was observed for P1 and F1 males during the premating period at the 37.5 mg/kg/day dose. A decreased mean live birth index was reported for P1 and F1 generations without an effect on viability and development of pups. No adverse effects on reproductive indices or mating performance were observed at any dose level. The parental toxicity NOEL was determined to be 15 mg/kg/day, and the parental toxicity LEL was determined to be 37.5 mg/kg/day, based on decreased body weight and food consumption during premating for P1 and F1 males, and decreased mean live birth index for the P1 and F1 generations. The reproductive toxicity Noel was determined to be 37.5 mg/kg/day, and the reproductive toxicity LEL was determined to be >37.5 mg/kg/day. **Mutagenicity:** In a mutagenicity study, IPBC technical was tested for the ability to cause mutations in ***Salmonella typhimurium*** strains TA 1535, TA 1537, TA 1538, TA 98, and TA 100. In the five strains used, IPBC was found to be non-mutagenic in the presence or absence of metabolic activation at the concentrations tested, 1-1000 jig/plate . In a micronucleus assay in mice, IPBC at doses of 200, 600, and 2000 mg/kg did not induce any significant increase of the PCE containing micronuclei from the treated mice when compared to that of the vehicle control mice. In two independent unscheduled DNA synthesis (UDS) assays in primary rat hepatocytes, eight doses of IPBC ranging from 3.0 to 13.5 ug/ml did not cause an appreciable increase in mean net nuclear grain counts. Doses >13.5 ug/ml were cytotoxic, supporting the conclusion that IPBC induced cytotoxicity but no genotoxicity in this assay. **Metabolism:** Based on the metabolite identification data, a scheme for metabolism of IPBC was proposed. According to this scheme, IPBC undergoes reductive dehalogenation followed by dealkylation to form the URM-9 and URM-10 metabolites. In addition, de-carboxylation following reductive dehalogenation yields carbon dioxide. Various other metabolites formed from dehalogenation are glucuronidated and constitute minor metabolites of IPBC..
C.I. PIGMENT YELLOW 83	For diarylide (disazo) pigments (3,3'-dichlorobenzidine-containing): 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. Diarylide pigments are synthesized by bis-diazotizing diamino-diphenyl derivatives, mainly 3,3'-dichlorobenzidine (DCB), and coupling with acetoacetarylides or arylsubstituted pyrazolones 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.

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Many diarylide pigments are derived from DCB. Therefore, the diarylide pigments on DCB basis have been tested toxicologically very extensively. Diarylide pigments with their LD50 values above 2 000 mg/kg show no acute toxicity according to the EU classification criteria. They are not irritating to the skin or mucous membranes.
For acute dermal toxicity a single LD50 of >3,000 mg/kg bw is available for Pigment Yellow 13. No deaths or clinical signs of toxicity were observed following oral or dermal exposure. The inhalation LC50 available is >4,448 mg/m3 for Pigment Yellow 13. Tachypnoea, dyspnoea, exophthalmos, ruffled fur and curved or ventral body position were observed, although all animals recovered and no gross abnormalities were observed at necropsy.
Based on the available data the pigments have a minimal to slight potential for eye irritation. There is no indication that they are sensitisers
No adverse effects were seen after 4-7 weeks oral administration of Pigment Yellow 12 at 1000 mg/kg/day (NOAEL), the highest dose tested in a well conducted and reported test of repeated dose toxicity study. Furthermore, in the cases of Pigment Yellow 12 and 83, no toxicologically significant effects were observed in a range of chronic toxicity studies of lesser quality (in terms of reporting) in rats and mice at doses up to 6500 mg/kg/day. Based on the kinetics of the three pigments and the chemical similarities, it can be concluded that these findings can be extrapolated to most if not all diarylide pigments.
For the inhalation route the effects seen are related to the deposition of dust particles in the lungs, leading to Pigment Yellow 13 related effects even at the lowest exposure concentration of 54 mg/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 2 1 controls). Hepatic tumors were observed in 4/l 8 strain D mice exposed to 11.2-l 1.9 mg 3,3 -dichlorobenzidine/kg/day in the diet for 10 months
No bladder carcinomas were observed in rats exposed to 0.03% 3,3 -dichlorobenzidine in the diet
(27 mg/kg/day) for 4 or 40 weeks , nor were any mammary tumors observed in rats administered approximately 49 mg 3,3 -dichlorobenzidine dihydrochloride/kg/day by gavage once every 3 days over a 30-day period and sacrificed 8 months later.
In a study in which rats were exposed to 10-20 mg 3,3 -dichlorobenzidine per day (120 mg/kg/day) in feed 6 days per week for 12 months, tumors were observed at a variety of sites, including the Zymbal gland (7 of 29 animals), mammary gland (7/29), bladder (3/29), hematopoietic system (3/29), skin (3/29), ileum (2/29), connective tissue (2/29), salivary gland (2/29), liver (l/29), and thyroid (l/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.

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	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
C.I. PIGMENT VIOLET 23	No carcinogenic effects observed during a 43 day test animal feeding study on Pigment Violet 23. [Manufacturer]
C.I. ACID BLUE 9	Tumorigenic - neoplastic by RTECS criteria. Acute toxicity: In male rats the LD50 for Brilliant Blue FCF is given as more than 2000 mg/kg body weight. The LD50 for male and female rats is > 5000 mg Basacid Blue 755/kg body weight. In cats a single administration of 1 g Patent Blue AE (set at a purity of 37 % with sodium chloride) caused neither methaemoglobin formation nor an increased incidence of Heinz bodies in erythrocytes Experiments on rabbits with Amido Blue AE showed no skin irritation; with Neptune Blue a very slight reddening was observed. In experiments with Basacid Blue 755 the irritation value could not be read, due to the skin staining. The rabbit eye was not irritated by Amido Blue AE and no effects of Neptune Blue were observed. For Basacid Blue an irritation value of . 4 is given. After 40 administrations of FD&C Blue No. 1, no irritation was determined on day 5 (score of 2), and some animals exhibited non-permanent staining of the eyes. Data are available on sensitisation in humans. In a difficult to assess sublingual test. In patients with allergic skin reactions positive reactions were seen in 14 % of 51 cases; no reactions were observed in 207 volunteers. General experience has shown particularly the eye irritation and skin sensitisation effects of a dye to depend greatly on the proportion of inorganic salts, such as sodium chloride, remaining in the finished product, since the saturated salt solution formed in contact with aqueous media can considerably increase the skin and eye damage caused by the compound. The positive results of the investigation by Mikkelsen et al. (1978) might also be due to hypersensitivity. Repeat dose toxicity: In rats the oral administration of Brilliant Blue for 75 weeks caused haemorrhaging in the adrenal gland, pituitary adenomas and uterine polyps, which the authors characterised as spontaneous effects (NOEL calculated to be 1554 and 1839 mg/kg body weight and day for male and female animals, respectively). In mice a dose of about 37.5 mg Brilliant Blue/kg body weight and day for 7 months caused liver damage, increased relative organ weights and increased activity of alkaline phosphatase (AP), aspartate aminotransferase (AST) and alanine aminotransferase (ALAT) in the plasma. Decreased AP activity was observed in the liver and heart; male animals also exhibited reduced AST activity. Reduced T3 values were determined in the serum of female animals and reduced T4 values in the serum of males. The number of white blood cells was increased in the plasma of both sexes, the number of red blood cells being additionally increased in female animals. Reproductive toxicity: Investigations on the reproductive toxicity are not available in the original. They do not indicate corresponding effects. One study showed anomalies in the kidneys of foetuses in the medium dose group only. In another investigation, the anomalies observed were equated with those seen in untreated animals Genotoxicity: C.I. Acid Blue 9 was not mutagenic to several strains of Salmonella typhimurium tested with or without S9 However, in a later recent study, C.I. Acid Blue 9 was mutagenic in the Salmonella assay and the mouse lymphoma TK+/- assay. An in vivo - in vitro unscheduled DNA synthesis assay indicated that C.I. Acid Blue 9 was mutagenic. C.I. Acid Blue 9 was not mutagenic by the DNA repair with ?rec-Assay? or the in vivo mouse micronucleus assay. However, C.I. Acid Blue 9 did produce chromosomal aberrations in vitro in CHL cells. C.I. Acid Blue 9 was negative in the Fischer rat embryo cell transformation and BHK21 cell transformation assays. Carcinogenicity: Tumours of the kidney were determined in male mice of the medium dose group after the administration of about 200 mg Brilliant Blue FCF/kg body weight in the feed for 80 weeks (IARC 1978). Dermal applications to mice for 18 months did not cause an increased incidence of neoplasias. Subcutaneous administration to rats caused sarcomas at the injection site, which were associated with interference to repair mechanisms, due to the physico-chemical properties of the compounds injected. Long-term studies carried out on mice and rats with FD&C Blue No. 1 (Brilliant Blue FCF) gave no indication of tumours, nor did another study performed with this compound on rats for 2 years (up to about 5000 mg/kg body weight and day) (* BUA Report 242 September 242 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. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.
C.I. PIGMENT RED 122	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. 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

Continued...

	It is considered unlikely that the quinacridone pigments of this category become systemically bioavailable after dermal or inhalation exposure. 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.
2-BROMO-2-NITROPROPAN-1,3-DIOL	Formaldehyde generators (releasers) are often used as preservatives. The maximum authorised concentration of free formaldehyde is 0.2% and must be labelled with the warning sign "contains formaldehyde" where the concentration exceeds 0.05%.The use of formaldehyde-releasing preservatives ensures that the level of free formaldehyde in the products is always low but sufficient to inhibit microbial growth - it disrupts metabolism to cause death of the organism. However there is a concern that formaldehyde generators can produce amines capable of causing cancers (nitrosamines) when used in formulations containing amines. 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.
WATER & TITANIUM DIOXIDE & CARBON BLACK & C.I. PIGMENT GREEN 7	No significant acute toxicological data identified in literature search.
TITANIUM DIOXIDE & GLYCEROL & 2-BROMO-2-NITROPROPAN-1,3-DIOL	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
TITANIUM DIOXIDE & CARBON BLACK	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
TITANIUM DIOXIDE & PROPYLENE GLYCOL & 2-BROMO-2-NITROPROPAN-1,3-DIOL	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

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

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

SECTION 12 Ecological information

Toxicity

KENT URBAN ACRYLIC PAINT MARKERS BRUSH NIB CLASSIC PACK 12

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

titanium dioxide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
BCF	1008h	Fish	<1.1-9.6	7
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

Continued...

	LC50	96h	Fish	1.85-3.06mg/l	4

glycerol

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

propylene glycol

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

carbon black

Endpoint	Test Duration (hr)	Species	Value	Source
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
LC50	96h	Fish	>100mg/l	2

3-iodo-2-propynyl butyl carbamate

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

C.I. Pigment Yellow 83

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>100mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	24h	Fish	>=0.1mg/l	2
LC50	96h	Fish	>0.1mg/l	2

C.I. Pigment Violet 23

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

C.I. Pigment Green 7

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>100mg/l	2
BCF	1008h	Fish	0.51-4.8	7
EC50	48h	Crustacea	153.6mg/l	2
NOEC(ECx)	504h	Crustacea	>=1mg/l	2
LC50	96h	Fish	>100mg/l	2

C.I. Acid Blue 9

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Crustacea	>=10mg/l	2
LC50	96h	Fish	>132<276mg/l	2

C.I. Pigment Red 122

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>10mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Crustacea	>0.02mg/l	2
LC50	96h	Fish	>100mg/l	2

C.I. Pigment Red 170

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

2-bromo-2-nitropropan-1,3-diol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.026mg/l	2

Continued...

EC50	48h	Crustacea	1.1-3.52mg/L	4
EC50	96h	Algae or other aquatic plants	0.02-0.025mg/L	4
EC10(ECx)	72h	Algae or other aquatic plants	0.013mg/l	2
LC50	96h	Fish	10.274-14.454mg/L	4

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

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
water	LOW	LOW
titanium dioxide	HIGH	HIGH
glycerol	LOW	LOW
propylene glycol	LOW	LOW
3-iodo-2-propynyl butyl carbamate	HIGH	HIGH
C.I. Pigment Yellow 83	HIGH	HIGH
C.I. Acid Blue 9	HIGH	HIGH
2-bromo-2-nitropropan-1,3-diol	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
water	LOW (LogKOW = -1.38)
titanium dioxide	LOW (BCF = 10)
glycerol	LOW (LogKOW = -1.76)
propylene glycol	LOW (BCF = 1)
3-iodo-2-propynyl butyl carbamate	LOW (LogKOW = 2.4542)
C.I. Pigment Yellow 83	LOW (LogKOW = 8.6648)
C.I. Pigment Green 7	LOW (BCF = 74)
C.I. Acid Blue 9	LOW (LogKOW = 2.0459)
C.I. Pigment Red 122	LOW (LogKOW = 2.47)
2-bromo-2-nitropropan-1,3-diol	LOW (LogKOW = -0.6408)

Mobility in soil

Ingredient	Mobility
titanium dioxide	LOW (Log KOC = 23.74)
glycerol	HIGH (Log KOC = 1)
propylene glycol	HIGH (Log KOC = 1)
3-iodo-2-propynyl butyl carbamate	LOW (Log KOC = 365.3)
C.I. Pigment Yellow 83	LOW (Log KOC = 1126000)
C.I. Acid Blue 9	LOW (Log KOC = 10000000000)
2-bromo-2-nitropropan-1,3-diol	HIGH (Log KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

14.7. Maritime transport in bulk according to IMO instruments

Continued...

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

Not Applicable

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

Product name	Group
water	Not Applicable
titanium dioxide	Not Applicable
glycerol	Not Applicable
propylene glycol	Not Applicable
carbon black	Not Applicable
3-iodo-2-propynyl butyl carbamate	Not Applicable
C.I. Pigment Yellow 83	Not Applicable
C.I. Pigment Violet 23	Not Applicable
C.I. Pigment Green 7	Not Applicable
C.I. Acid Blue 9	Not Applicable
C.I. Pigment Red 122	Not Applicable
C.I. Pigment Red 170	Not Applicable
2-bromo-2-nitropropan-1,3-diol	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
water	Not Applicable
titanium dioxide	Not Applicable
glycerol	Not Applicable
propylene glycol	Not Applicable
carbon black	Not Applicable
3-iodo-2-propynyl butyl carbamate	Not Applicable
C.I. Pigment Yellow 83	Not Applicable
C.I. Pigment Violet 23	Not Applicable
C.I. Pigment Green 7	Not Applicable
C.I. Acid Blue 9	Not Applicable
C.I. Pigment Red 122	Not Applicable
C.I. Pigment Red 170	Not Applicable
2-bromo-2-nitropropan-1,3-diol	Not Applicable

SECTION 15 Regulatory information

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

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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)

glycerol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

propylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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)

3-iodo-2-propynyl butyl carbamate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Continued...

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

C.I. Pigment Yellow 83 is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans

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

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

C.I. Pigment 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. Acid Blue 9 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 Red 170 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (water; glycerol; propylene glycol; carbon black; 3-iodo-2-propynyl butyl carbamate; C.I. Pigment Yellow 83; C.I. Pigment Violet 23; C.I. Pigment Green 7; C.I. Acid Blue 9; C.I. Pigment Red 122; C.I. Pigment Red 170; 2-bromo-2-nitropropan-1,3-diol)
China - IECSC	Yes
Colombia - Colombia National Industrial Chemicals Inventory	No (water; titanium dioxide; glycerol; propylene glycol; carbon black; 3-iodo-2-propynyl butyl carbamate; C.I. Pigment Yellow 83; C.I. Pigment Violet 23; C.I. Pigment Green 7; C.I. Acid Blue 9; C.I. Pigment Red 122; C.I. Pigment Red 170; 2-bromo-2-nitropropan-1,3-diol)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (C.I. Pigment Yellow 83; C.I. Pigment Green 7; C.I. Acid Blue 9; C.I. Pigment Red 122)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (water; titanium dioxide; glycerol; propylene glycol; carbon black; 3-iodo-2-propynyl butyl carbamate; C.I. Pigment Yellow 83; C.I. Pigment Violet 23; C.I. Pigment Green 7; C.I. Acid Blue 9; C.I. Pigment Red 122; C.I. Pigment Red 170; 2-bromo-2-nitropropan-1,3-diol)
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	27/10/2026
Initial Date	27/10/2026

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.

Continued...

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

Definitions and abbreviations

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

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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