Reeves Watercolour 12ml Paint

Jasco Pty Limited

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5398-64**
Version No: **5.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **10/03/2023**
Print Date: **11/08/2024**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Reeves Watercolour 12ml Paint
Chemical Name	Not Applicable
Synonyms	Not Available
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	Use according to manufacturer's directions.

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, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 3, Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3
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)	

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Signal word	**Danger**

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H331	Toxic if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.

Precautionary statement(s) Prevention

P261	Avoid breathing mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P405	Store locked up.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
9000-01-5	30-55	gum arabic
21645-51-2	5-12	aluminium hydroxide
56-81-5	5-8	glycerol
1302-78-9	1-3	bentonite
10605-21-7	<1	carbendazim
10377-60-3	<1	magnesium nitrate
2682-20-4	<1	2-methyl-4-isothiazolin-3-one
55965-84-9	<1	isothiazolinones, mixed
26530-20-1	<1	2-octyl-4-isothiazolin-3-one
7732-18-5	10-30	water
Not Available	0-15	pigment
Not Available		may contain:
13463-67-7	NotSpec	C.I. Pigment White 6

Continued...

CAS No	%[weight]	Name
6486-23-3	NotSpec	C.I. Pigment Yellow 3
2512-29-0	NotSpec	C.I. Pigment Yellow 1
20344-49-4	NotSpec	ferric hydroxide
1309-37-1	NotSpec	C.I. Pigment Red 101
6410-26-0	NotSpec	C.I. Pigment Red 21
1328-53-6	NotSpec	C.I. Pigment Green 7
6358-30-1	NotSpec	C.I. Pigment Violet 23
147-14-8	NotSpec	C.I. Pigment Blue 15
980-26-7	NotSpec	C.I. Pigment Red 122
57455-37-5	NotSpec	C.I. Pigment Blue 29
1325-87-7	NotSpec	C.I. Pigment Blue 1
1333-86-4	NotSpec	C.I. Pigment Black 7
2814-77-9	NotSpec	C.I. Pigment Red 4
5280-68-2	NotSpec	C.I. Pigment Red 146
6528-34-3	NotSpec	C.I. Pigment Yellow 65
6358-31-2	NotSpec	C.I. Pigment Yellow 74
1345-16-0	NotSpec	C.I. Pigment Blue 28

Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ 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	If skin or hair contact occurs: ▸ 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.
Inhalation	▸ 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.
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

▸ There is no restriction on the type of extinguisher which may be used.
▸ Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Continued...

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ 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.
Fire/Explosion Hazard	▸ Non combustible. ▸ Not considered a significant fire risk, however containers may burn. Decomposes on heating and produces: carbon dioxide (CO_2) hydrogen chloride phosgene nitrogen oxides (NOx) phosphorus oxides (POx) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ 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.
Major Spills	▸ Absorb or contain isothiazolinone liquid spills with sand, earth, inert material or vermiculite. ▸ The absorbent (and surface soil to a depth sufficient to remove all of the biocide) should be shovelled into a drum and treated with an 11% solution of sodium metabisulfite ($Na_2S_2O_5$) or sodium bisulfite ($NaHSO_3$), or 12% sodium sulfite (Na_2SO_3) and 8% hydrochloric acid (HCl). ▸ Glutathione has also been used to inactivate the isothiazolinones. ▸ Use 20 volumes of decontaminating solution for each volume of biocide, and let containers stand for at least 30 minutes to deactivate microbicide before disposal. ▸ If contamination of drains or waterways occurs, advise emergency services. ▸ After clean up operations, decontaminate and launder all protective clothing ▸ and equipment before storing and re-using.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ 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.

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

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents

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	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 Exposure Standards	C.I. Pigment White 6	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	ferric hydroxide	Iron oxide fume (Fe2O3) (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	C.I. Pigment Red 101	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	C.I. Pigment Red 101	Iron oxide fume (Fe2O3) (as Fe)	5 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	C.I. Pigment Black 7	Carbon black	3 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
aluminium hydroxide	8.7 mg/m3	73 mg/m3	440 mg/m3
glycerol	45 mg/m3	180 mg/m3	1,100 mg/m3
magnesium nitrate	30 mg/m3	330 mg/m3	2,000 mg/m3
magnesium nitrate	16 mg/m3	180 mg/m3	1,100 mg/m3
C.I. Pigment White 6	30 mg/m3	330 mg/m3	2,000 mg/m3
ferric hydroxide	30 mg/m3	330 mg/m3	2,000 mg/m3
ferric hydroxide	15 mg/m3	360 mg/m3	2,200 mg/m3
ferric hydroxide	24 mg/m3	260 mg/m3	1,600 mg/m3
C.I. Pigment Red 101	15 mg/m3	360 mg/m3	2,200 mg/m3
C.I. Pigment Black 7	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
gum arabic	Not Available	Not Available
aluminium hydroxide	Not Available	Not Available
glycerol	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
bentonite	Not Available	Not Available
carbendazim	Not Available	Not Available
magnesium nitrate	Not Available	Not Available
2-methyl-4-isothiazolin-3-one	Not Available	Not Available
isothiazolinones, mixed	Not Available	Not Available
2-octyl-4-isothiazolin-3-one	Not Available	Not Available
water	Not Available	Not Available
C.I. Pigment White 6	5,000 mg/m3	Not Available
C.I. Pigment Yellow 3	Not Available	Not Available
C.I. Pigment Yellow 1	Not Available	Not Available
ferric hydroxide	2,500 mg/m3	Not Available
C.I. Pigment Red 101	2,500 mg/m3	Not Available
C.I. Pigment Red 21	Not Available	Not Available
C.I. Pigment Green 7	Not Available	Not Available
C.I. Pigment Violet 23	Not Available	Not Available
C.I. Pigment Blue 15	Not Available	Not Available
C.I. Pigment Red 122	Not Available	Not Available
C.I. Pigment Blue 29	Not Available	Not Available
C.I. Pigment Blue 1	1,000 mg/m3	Not Available
C.I. Pigment Black 7	1,750 mg/m3	Not Available
C.I. Pigment Red 4	Not Available	Not Available
C.I. Pigment Red 146	Not Available	Not Available
C.I. Pigment Yellow 65	Not Available	Not Available
C.I. Pigment Yellow 74	Not Available	Not Available
C.I. Pigment Blue 28	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
gum arabic	E	≤ 0.01 mg/m³
aluminium hydroxide	E	≤ 0.01 mg/m³
bentonite	E	≤ 0.01 mg/m³
carbendazim	E	≤ 0.01 mg/m³
magnesium nitrate	E	≤ 0.01 mg/m³
2-methyl-4-isothiazolin-3-one	D	> 0.01 to ≤ 0.1 mg/m³
isothiazolinones, mixed	E	≤ 0.1 ppm
2-octyl-4-isothiazolin-3-one	E	≤ 0.1 ppm
C.I. Pigment Yellow 3	E	≤ 0.01 mg/m³
C.I. Pigment Yellow 1	E	≤ 0.01 mg/m³
C.I. Pigment Red 21	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
C.I. Pigment Blue 28	E	≤ 0.01 mg/m³
Notes:	***Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.***	

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.

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Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Individual protection measures, such as personal protective equipment

Eye and face protection

- Chemical goggles.
- 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. 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], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

- 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.
- Butyl rubber gloves

· Nitrile rubber gloves (Note: Nitric acid penetrates nitrile gloves in a few minutes.)

Body protection

See Other protection below

Other protection

- Overalls.
- P.V.C apron.
- Barrier cream.
- Skin cleansing cream.
- Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Reeves Watercolour 12ml Paint

Material	CPI

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.

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BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	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.

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)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured paste; mixes with water.

Physical state	Non Slump Paste	**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 Applicable
pH (as supplied)	9-10	**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 Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**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	▸ 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

Continued...

Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. 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.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Studies indicate that diets containing large amounts of non-absorbable polysaccharides, such as cellulose, might decrease absorption of calcium, magnesium, zinc and phosphorus. Polysaccharides are polymeric carbohydrates that consist of monosaccharide units, which are connected together with glycosidic bonds. Due to the structural variation of different monosaccharides as well as the innumerable ways that these building blocks link with each other, polysaccharides can be considered as structurally complex biomacromolecules. Polysaccharides originating from plants (e.g., starch and guar gum), microbes (e.g., xanthan), algae (e.g., alginates and carrageenans) and animals (e.g., glycogen and chitin) are frequently used in food. Starch, a high molar mass compound consisting of (1->4)-linked alpha-D-glucopyranosyl units, is an important energy nutrient that is abundant in common foods, such as cereals and root crops. Although many other food polysaccharides are not digested in the upper gastrointestinal tract of humans, they often serve functions other than being components giving nutritional value. For example, plant cell-wall polysaccharides, such as arabinoxylans and beta-glucan, exist in cereal-based foods, and “plant gums” are used as thickeners, emulsifiers, emulsion stabilizers, gelling agents and encapsulating agents. These non-digestible polysaccharides are important for health because they are considered as dietary fibre, which promote colon health, regulate post-prandial blood glucose levels and reduce serum cholesterol levels. Despite the fact that nature provides various sources of polysaccharides, and that scientific research on their exploitation as food materials is increasingly active, a relatively low number of polysaccharides are authorized for use as food ingredients. For example, in the European Union (EU) and in Switzerland, among the permitted food additives (identified by an E number) only a

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small percentage are polysaccharide-based (native or structurally modified). The difference between other food ingredients and food additives is mainly the quantity used in any given product. Food ingredients can be consumed alone as food (e.g., starch), whereas food additives (e.g., carboxymethyl cellulose) are used in small quantities (usually less than 2%) relative to the total food composition but they, nonetheless, play an important role in the food products. Regarding food additive use in Europe, the European Food Safety Authority (EFSA) has an expert Panel on Food Additives and Nutrient Sources Added to Food (ANS), which evaluates the safety of food additives. Similarly, if new ingredients are released into the market, EFSA's Panel on Dietetic Products, Nutrition and Allergies (NDA) has the responsibility of evaluating the safety of Novel Food ingredients
The vast majority of polysaccharides used as food ingredients are plant-based. In addition to the cellulosic polysaccharides, other types of food-grade ingredients or additives, such as, vanillin aroma, glycerol esters of wood rosins (E445), xylitol (E967) and steryls/stanols, are derived from wood. The main components of wood are polysaccharides: cellulose (40–50 wt%) and hemicelluloses (20–35%), while lignin comprises 15–30% of wood mass.
There are reports of lung damage due to excessive inhalation of alumina dust. Ingestion of large amounts of aluminium hydroxide for prolonged periods may cause phosphate depletion, especially if phosphate intake is low. This may cause loss of appetite, muscle weakness, muscular disease and even softening of the bones. These effects have not been reported in people occupationally exposed to aluminium hydroxide.
The isothiazolinones are known contact sensitisers. Data are presented which demonstrate that, in comparison with the chlorinated and dichlorinated compounds which share immunological cross-reactivity, the non-chlorinated isothiazolinones have a lower potential for sensitization and no documented immunological cross-reaction with the chlorinated isothiazolinones. The risk of sensitization depends on how contact with the product occurs. The risk is greater when the skin barrier has been damaged and smaller when the skin is healthy. Dermatological studies have demonstrated that mixed isothiazolinone concentrations below 20 ppm may cause sensitisation and that allergic reactions can be provoked in sensitized persons even with concentrations in the range of 7-15 ppm active isothiazolinones.
The isothiazolinones are a group of heterocyclic sulfur-containing compounds. In general all are electrophilic molecules containing an activated N-S bond that enables them with nucleophilic cell entities, thus exerting biocidal activity. A vinyl activated chlorine atom makes allows to molecule to exert greater antimicrobial efficiency but at the same time produces a greater potential for sensitisation.
Several conclusions relating to the sensitising characteristics of the isothiazolinones may therefore be drawn* :

- The strongest sensitisers are the chlorinated isothiazolinones.
- There are known immunological cross-reactions between at least 2 different chlorinated isothiazolinones.
- There appears to be no immunological cross reaction between non-chlorinated isothiazolinones and chlorinated isothiazolinones.
- Although classified as sensitisers, the nonchlorinated isothiazolinones are considerably less potent sensitisers than are the chlorinated isothiazolinones.
- By avoiding the use of chlorinated isothiazolinones, the potential to induce sensitisation is greatly reduced.
- Despite a significant percentage of the population having been previously sensitised to chlorinated and non-chlorinated species, it is likely that careful and judicious use of non-chlorinated isothiazolinones will result in reduced risk of allergic reactions in those persons.
- Although presently available data promise that several non-chlorinated isothiazolinones will offer effective antimicrobial protection in industrial and personal care products, it is only with the passage of time that proof of their safety in use or otherwise will become available.

* B.R. Alexander: Contact Dermatitis 2002, 46, pp 191-196
Although there have been conflicting reports in the literature, it has been reported by several investigators that isothiazolinones are mutagenic in ***Salmonella typhimurium*** strains (Ames test). Negative results were obtained in studies of the DNA-damaging potential of mixed isothiazolinones (Kathon) in mammalian cells ***in vitro*** and of cytogenetic effects and DNA-binding ***in vivo.*** The addition of rat liver S-9 (metabolic activation) reduced toxicity but did not eliminate mutagenicity. These compounds bind to the proteins in the S-9. At higher concentrations of Kathon the increase in mutagenicity may be due to an excess of unbound active compounds.
A study of cutaneous application of Kathon CG in 30 months, three times per week at a concentration of 400 ppm (0.04%) a.i. had no local or systemic tumourigenic effect in male mice. No dermal or systemic carcinogenic potential was observed.
Reproduction and teratogenicity studies with rats, given isothiazolinone doses of 1.4-14 mg/kg/day orally from day 6 to day 15 of gestation, showed no treatment related effects in either the dams or in the foetuses
Respiratory sensitisation may result in allergic/asthma like responses; from coughing and minor breathing difficulties to bronchitis with wheezing, gasping.

	TOXICITY	IRRITATION
Reeves Watercolour 12ml Paint	Not Available	Not Available
gum arabic	**TOXICITY**	**IRRITATION**
	Oral (Rabbit) LD50; 8000 mg/kg[2]	Eye (rabbit): 36 mg/5h SEVERE
aluminium hydroxide	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >2.3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
glycerol	**TOXICITY**	**IRRITATION**
	Dermal (Guinea Pig) LD50: 58500 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >5.85 mg/L4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; 4090 mg/kg[2]	
bentonite	**TOXICITY**	**IRRITATION**

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	Oral (Cat) LD50; >1.25 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
carbendazim	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 2000 mg/kg[2]	Eye (rabbit): non-irritating *
	Oral (Dog) LD50; >2500 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): non-irritating *
		Skin: no adverse effect observed (not irritating)[1]
magnesium nitrate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 5440 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
		Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 500 mg/24h - mild
		Skin: no adverse effect observed (not irritating)[1]
2-methyl-4-isothiazolin-3-one	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 242 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Inhalation (Rat) LC50: 0.1 mg/l4h[1]	Skin: adverse effect observed (corrosive)[1]
	Oral (Rat) LD50: 120 mg/kg[1]	
isothiazolinones, mixed	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >1008 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Inhalation (Rat) LC50: 0.171 mg/l4h[1]	Skin: adverse effect observed (corrosive)[1]
	Oral (Rat) LD50: 53 mg/kg[2]	Skin: adverse effect observed (irritating)[1]
2-octyl-4-isothiazolin-3-one	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 311 mg/kg[2]	Eye (rabbit): 0.5% non irritant
	Oral (Rat) LD50: 248 mg/kg[2]	Eye (rabbit): 45% conc CORROSIVE
		Eye (rabbit): 5% conc moderate
		Eye(rabbit):100 mg SEVERE
		Eye: adverse effect observed (irreversible damage)[1]
		Skin (rabbit): 45% conc SEVERE
		Skin (rabbit): 500 mg/24 hours
		Skin: adverse effect observed (corrosive)[1]
		Skin: adverse effect observed (irritating)[1]
water	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >90000 mg/kg[2]	Not Available
C.I. Pigment White 6	**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 (rabbit) Draize 0.3mg/3hrInt Mild
	Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Yellow 3	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >10000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Yellow 1	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >5000 mg/kg[2]	Non-irritating/non-sensitising [Dominion]
		Skin: no adverse effect observed (not irritating)[1]

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ferric hydroxide	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Red 101	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >5000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Red 21	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
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. Pigment Violet 23	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): Non-irritating * * [Ravenswood]
		Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Blue 15	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (human): non-irritant [Manuf. C.G.]
	Inhalation (Rat) LC50: >1.084<5.212 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin (human): non-irritant
		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]
C.I. Pigment Blue 29	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >10000 mg/kg[2]	Not Available
C.I. Pigment Blue 1	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >5000 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin: adverse effect observed (irritating)[1]
C.I. Pigment Black 7	**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]
C.I. Pigment Red 4	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; >10000 mg/kg[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 Yellow 65	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Eye: non-irritating *
		Skin: no adverse effect observed (not irritating)[1]
		Skin: non-irritatng *

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C.I. Pigment Yellow 74	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (human): non irritant [CCINFO-Bayer]
	Oral (Rat) LD50: >1500 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (human): non irritant
		Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Blue 28	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >5.06 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >10000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

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

GUM ARABIC	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. Gum arabic is a technical name for Acacia Senegal Gum. Gum arabic is comprised of various sugars and glucuronic acid residues in a long chain of galactosyl units with branched oligosaccharides. Gum arabic is generally recognized as safe as a direct food additives. Toxicity data on gum arabic indicates little or no acute, short-term, or subchronic toxicity. Gum arabic is negative in several genotoxicity assays, is not a reproductive or developmental toxin, and is not carcinogenic when given intraperitoneally or orally. Clinical testing indicated some evidence of skin sensitization with gum arabic. The material may produce severe irritation to the eye causing pronounced 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 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).
BENTONITE	for bentonite clays: Bentonite (CAS No. 1302-78-9) consists of a group of clays formed by crystallisation of vitreous volcanic ashes that were deposited in water. The expected acute oral toxicity of bentonite in humans is very low (LD50>15 g/kg). However, severe anterior segment inflammation, uveitis and retrocorneal abscess from eye exposure were reported when bentonite had been used as a prophypaste. In a 33 day dietary (2 and 6%) and a 90 day dietary (1, 3 and 5%) studies in chickens, no changes in behaviour, overall state, clinical and biochemical parameters and electrolytic composition of the blood. Repeat dietary administration of bentonite did not affect calcium or phosphorus metabolism. However, larger amounts caused decreased growth, muscle weakness, and death with marked changes in both calcium and phosphorus metabolism. Bentonite did not cause fibrosis after 1 year exposure of 60 mg dust (<5 um) in a rat study. However, in a second rat study, where 5 um particles were intratracheally instilled at 5, 15 and 45 mg/rat, dose-related fibrosis was observed. Bentonite clay dust is believed to be responsible for bronchial asthma in workers at a processing plant in USA. Ingestion of bentonite without adequate liquids may result in intestinal obstruction in humans.

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	Hypokalaemia and microcytic iron-deficiency anaemia may occur in patients after repeat doses of clay. Chronic ingestion has been reported to cause myositis.
CARBENDAZIM	Intraperitoneal (Rat, adult male) LD50: 7320 mg/kg * Intraperitoneal (Rat, adult female) LD50: 15000 mg/kg * Inhalation LC50 (4 h) for rats, rabbits, guinea pigs or cats no effect with suspension (10 g/l water). * NOEL (2 y) for dogs 300 mg/kg diet, corresponding to 6-7 mg/kg b.w. ADI 0.01 mg/kg b.w. * Toxicity Class WHO III;EPA IV Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. for carbendazim: Benomyl (a precursor to carbendazim) causes dermal sensitization in humans. Benomyl and carbendazim represent a very low risk for acute poisoning in humans. In animal systems, carbendazim is metabolized to (5-hydroxy- 1H-benzimidazol-2-yl)-carbamate (5-HBC) and other polar metabolites, which are rapidly excreted. Carbendazim has not been observed to accumulate in any biological system. Carbendazim has low acute toxicity. The LD50 values range from > 2000 to 15 000 mg/kg in a wide variety of test animals and routes of administration. However, significant adverse reproductive effects have been noted following a single exposure Carbendazim is well absorbed (80-85%) after oral exposure but much less so by dermal exposure. Absorbed carbendazim is metabolised into many compounds within the organism. The main metabolites are 5-HBC and 5,6-HOBC-N-oxides. The tissue distribution of carbendazim showed no bioconcentration. In the rat, the highest concentration after oral carbendazim administration (< 1% of the dose) occurred in the liver. It was distributed as carbendazim in the mitochondria, 5-HBC in the cytostol, and 2-aminobenzimidazole (2-AB) in the microsomes. Carbendazim is excreted in the urine and faeces within 72 h after oral dosing in rats. In rats and mice, high doses of carbendazim, both in the diet and by gavage, affect certain liver microsomal enzymes. **Short-term exposure** Dietary administration of carbendazim for up to 90 days produced slight effects on liver weight in female rats exposed to 360 mg/kg body weight per day. In a 90-day gavage study in the rat, the NOEL was 16 mg/kg per day based on hepatotoxicity. Short-term feeding studies on dogs were not adequate for establishing a NOEL. A 10-day dermal study in the rabbit revealed no systemic toxicity at the only dose tested (200 mg/kg). **Long-term exposure** Male and female rats fed 2500 mg/kg diet showed reduced erythrocyte count and haemoglobin and haematocrit values. No liver-related toxicity was noted. Male rats fed 2500 mg/kg diet or more presented a marginal increase in diffuse testicular atrophy and prostatitis. The NOEL in the rat is 500 mg/kg diet. Male and female mice fed 5000 mg/kg diet showed increased absolute liver weight. There was also significant centrilobular hypertrophy, necrosis and swelling of the liver in male mice fed 1500 mg/kg diet. **Reproduction, embryotoxicity and teratogenicity** Carbendazim was without adverse effects on reproduction when it was fed to rats in a three-generation reproduction study at levels up to and including 500 mg/kg diet. Male fertility was depressed in rats when carbendazim (200 mg/kg per day) was administered by gavage for 85 days. A dose of 50 mg/kg body weight per day in this study caused a significant decrease in epididymal sperm count. Following a single oral dose to rats, histological examination revealed early (0-2 days) disruption of spermatogenesis with occlusion of efferent ducts and increased testicular weights at 100 mg/kg body weight. No effect was observed at 50 mg/kg in this single dose study. These effects persisted until day 70 in rats treated with 400 mg/kg. Carbendazim caused an increase in malformations and anomalies in rats when administered at daily dose levels greater than 10 mg/kg on days 7-16 of gestation. There was a slightly decreased rate of implantation in rabbits administered 20 and 125 mg/kg per day on days 7-19 of gestation and an increased incidence of resorption at 125 mg/kg per day. Maternal toxicity was observed at 20 mg/kg per day and 125 mg/kg per day in the rat and rabbit, respectively. In rats there was a significant increase in foetal malformations at 90 mg/kg per day. These consisted primarily of hydrocephaly, microphthalmia, anophthalmia, malformed scapulea and axial skeletal malformations (vertebral, rib and sternebral fusions, exencephaly, hemivertebrae and rib hyperplasia). However, in the rabbit there were no significant malformations. **Mutagenicity and related end-points** Assays in mammalian and non-mammalian systems ***in vitro*** and ***in vivo*** and in somatic cells as well as in germ cells show that carbendazim does not interact with DNA, induce point mutation or cause germ cell mutation. Carbendazim does, however, cause numerical chromosome aberrations (aneuploidy and/or polyploidy) in experimental systems, both ***in vitro*** and ***in vivo.*** **Carcinogenicity:** Benomyl and its decomposition product carbendazim feeding resulted in an increase in the incidence of hepatocellular tumours in CD-1 and SPF Swiss mice. A carcinogenicity study of carbendazim using CD-1 mice showed a statistically significant dose-related increase in the incidence of hepatocellular neoplasia in females. There was also a statistically significant increase in the mid-dose (1500 mg/kg diet) males, but not in the high-dose males because of a high mortality rate. A carcinogenicity study of carbendazim in a genetically related mouse strain, SPF mice (Swiss random strain) at doses of 0, 150, 300 and 1000 mg/kg diet (increased to 5000 mg/kg during the study) showed an increase in the incidence of combined hepatocellular adenomas and carcinomas. Carcinogenicity studies of both benomyl and carbendazim in rats were negative. **Mechanism of toxicity - mode of action** The biological effects of benomyl and carbendazim result from their interaction with cell microtubules. These structures are involved in vital functions such as cell division, which is inhibited by benomyl and carbendazim. Benomyl and carbendazim toxicities in mammals are linked to microtubular dysfunction. Benomyl and carbendazim, as well as other benzimidazole compounds, display species-selective toxicity. This selectivity is, at least in part, explained by the different binding of benomyl and carbendazim to tubulins of target and non-target species ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]***
MAGNESIUM NITRATE	Magnesium nitrate heaxahydrate is a methaemoglobin-forming agent which if inhaled or ingested in high enough concentrations may cause fatigue, headache, dizziness. (Source: I.L.O. Encyclopaedia)
2-METHYL-4-ISOTHIAZOLIN-3-ONE	Considered to be a minor sensitiser in Kathon CG (1) (1). Bruze etal - Contact Dermatitis 20: 219-39, 1989 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 with similar materials using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.
2-OCTYL-4-ISOTHIAZOLIN-3-ONE	ROHM & HAAS Data ADI: 0.03 mg/kg/day NOEL: 60 mg/kg/day
C.I. PIGMENT WHITE 6	Substance has been investigated as a mutagen, tumorigen and primary irritant.

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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 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.

C.I. PIGMENT YELLOW 3

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

The azo linkage is considered the most labile portion of an azo dye. The linkage easily undergoes enzymatic breakdown, but thermal or photochemical breakdown may also take place. The breakdown results in cleavage of the molecule and in release of the component amines. Water solubility determines the ultimate degradation pathways of the dyes. For example the azo linkage of many azo pigments is, due to very low solubility in water, not available for intracellular enzymatic breakdown but may be susceptible to endogenous micro-organisms found in the bladder or in the gut.
After cleavage of the azo linkage by bacteria, the component aromatic amines are absorbed in the intestine and excreted in the urine. Twenty-two of the component amines are recognised as potential human carcinogens, and/or several of them have shown carcinogenic potential on experimental animals. Sulfonation of the dye reduces the toxicity by enhancement of the excretion.
The component amines which may be released from azo dyes are mostly aromatic amines (compounds where an amine group or amine-generating group(s) are connected to an aryl moiety). In general, aromatic amines known as carcinogenic may be grouped into five groups

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

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

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	are now epidemiological indications that occupational exposure to benzidene-based azo colourants can increase the incidence of bladder carcinoma. The acute toxicity of azo dyes is low.. However, potential health effects are recognised. Despite a very broad field of application and exposure, sensitising properties of azo dyes have been identified in relatively few reports. Red azoic dyes have been linked to allergic contact dermatitis in heavily exposed workers. Furthermore, textiles coloured with disperse azo dyes have caused allergic dermatitis in a few cases.
C.I. PIGMENT VIOLET 23	No carcinogenic effects observed during a 43 day test animal feeding study on Pigment Violet 23. [Manufacturer]
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 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.

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	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.
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. [ICI]
C.I. PIGMENT BLUE 28	For aluminium compounds: Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate). Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability. For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe. Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium. The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account Systemic toxicity after repeated exposure No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium. When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose. The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent Reproductive and developmental toxicity: Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI. Genotoxicity Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene

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	mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels. Carcinogenicity. The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons. Neurodegenerative diseases. Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases.Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment. Contact sensitivity: It has been suggested that the body burden of aluminium may be linked to different iseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome. Aluminium acts not only as an adjuvant,stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitisers causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response.Once inside the skin, the metal ions must bind to proteins to become immunologically reactive.The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminium Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste
GUM ARABIC & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & ISOTHIAZOLINONES, MIXED & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE	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.
GUM ARABIC & GLYCEROL & BENTONITE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & ISOTHIAZOLINONES, MIXED & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE & C.I. PIGMENT BLUE 28	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.
GUM ARABIC & ALUMINIUM HYDROXIDE & BENTONITE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & ISOTHIAZOLINONES, MIXED & WATER & FERRIC HYDROXIDE & C.I. PIGMENT RED 101 & C.I. PIGMENT RED 21 & C.I. PIGMENT GREEN 7 & C.I. PIGMENT BLACK 7 & C.I. PIGMENT RED 4 & C.I. PIGMENT BLUE 28	No significant acute toxicological data identified in literature search.
MAGNESIUM NITRATE & 2-METHYL-4-ISOTHIAZOLIN-	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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3-ONE & ISOTHIAZOLINONES, MIXED	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.
2-METHYL-4-ISOTHIAZOLIN-3-ONE & ISOTHIAZOLINONES, MIXED	In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance. Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration. 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 once inside the microbe cell. Some release detectable levels of formaldehyde into the air space, above working solutions, especially when pH has dropped. 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. 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 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, ***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%.*** 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.
2-METHYL-4-ISOTHIAZOLIN-3-ONE & C.I. PIGMENT RED 4	**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.

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

Continued...

Toxicity

Reeves Watercolour 12ml Paint

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

gum arabic

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

aluminium hydroxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	0.57mg/l	2
EC50	72h	Algae or other aquatic plants	0.017mg/L	2
EC50	48h	Crustacea	>0.065mg/l	4
NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
EC50	96h	Algae or other aquatic plants	0.005mg/L	2

glycerol

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

bentonite

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	19000mg/L	4

carbendazim

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	0.6-1.1	7
EC50	72h	Algae or other aquatic plants	1.3mg/l	2
NOEC(ECx)	96h	Fish	0.001mg/L	4
EC50	48h	Crustacea	0.02mg/L	4
LC50	96h	Fish	0.006-0.009mg/L	4
EC50	96h	Algae or other aquatic plants	19.056mg/l	4

magnesium nitrate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	24h	Crustacea	6075mg/L	5

2-methyl-4-isothiazolin-3-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.057mg/L	2
EC50	48h	Crustacea	0.189-0.257mg/L	4
LC50	96h	Fish	0.081-0.122mg/L	4
NOEC(ECx)	96h	Algae or other aquatic plants	0.01mg/l	2
EC50	96h	Algae or other aquatic plants	0.061mg/L	2

isothiazolinones, mixed

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.006mg/L	2
EC50	48h	Crustacea	0.007mg/l	2
LC50	96h	Fish	0.129mg/l	2
EC50	96h	Algae or other aquatic plants	0.036mg/L	2
NOEC(ECx)	48h	Algae or other aquatic plants	<0.001mg/L	2

2-octyl-4-isothiazolin-3-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.057-0.178mg/L	4
LC50	96h	Fish	0.041-0.104mg/l	4
NOEC(ECx)	840h	Fish	0.009mg/L	4
EC50	96h	Algae or other aquatic plants	0.15mg/l	2

water

Endpoint	Test Duration (hr)	Species	Value	Source

Continued...

	Not Available	Not Available	Not Available	Not Available	Not Available

C.I. Pigment White 6

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<1.1-9.6	7
EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
EC50	48h	Crustacea	1.9mg/l	2
LC50	96h	Fish	1.85-3.06mg/l	4
NOEC(ECx)	672h	Fish	>=0.004mg/L	2
EC50	96h	Algae or other aquatic plants	179.05mg/l	2

C.I. Pigment Yellow 3

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Crustacea	1mg/l	2
LC50	96h	Fish	>1mg/l	2

C.I. Pigment Yellow 1

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>1mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Crustacea	1mg/l	2

ferric hydroxide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	18mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Fish	0.52mg/l	2
LC50	96h	Fish	0.05mg/l	2
LC50	96h	Fish	0.05mg/l	2
NOEC(ECx)	504h	Fish	0.52mg/l	2
EC50	72h	Algae or other aquatic plants	18mg/l	2
EC50	48h	Crustacea	>100mg/l	2

C.I. Pigment Red 101

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	18mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Fish	0.52mg/l	2
LC50	96h	Fish	0.05mg/l	2

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 Green 7

Endpoint	Test Duration (hr)	Species	Value	Source
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
LC50	96h	Fish	>100mg/l	2
NOEC(ECx)	504h	Crustacea	>=1mg/l	2

C.I. Pigment Violet 23

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

C.I. Pigment Blue 15

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<0.33-11	7
EC50	72h	Algae or other aquatic plants	>100mg/l	2

Continued...

	EC50	48h	Crustacea	>500mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50(ECx)	504h	Crustacea	>1mg/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
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	504h	Crustacea	>0.02mg/l	2

C.I. Pigment Blue 29					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	000mg/l	Not Available

C.I. Pigment Blue 1					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	1mg/l	Not Available
	LC50	96h	Fish	0.1-1mg/l	Not Available
	EC50(ECx)	48h	Crustacea	1mg/l	Not Available

C.I. Pigment Black 7					
	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
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	24h	Crustacea	3200mg/l	1

C.I. Pigment Red 4					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	>0.006mg/L	2

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 Yellow 65					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	504h	Crustacea	1mg/l	2
	LC50	96h	Fish	>1mg/l	2

C.I. Pigment Yellow 74					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	504h	Crustacea	>1mg/l	2

C.I. Pigment Blue 28					
	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***

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
glycerol	LOW	LOW
carbendazim	HIGH	HIGH
2-methyl-4-isothiazolin-3-one	HIGH	HIGH

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
2-octyl-4-isothiazolin-3-one	HIGH	HIGH
water	LOW	LOW
C.I. Pigment White 6	HIGH	HIGH
C.I. Pigment Yellow 3	HIGH	HIGH
C.I. Pigment Yellow 1	HIGH	HIGH
C.I. Pigment Blue 15	HIGH	HIGH
C.I. Pigment Yellow 74	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
glycerol	LOW (LogKOW = -1.76)
carbendazim	LOW (BCF = 3.5)
2-methyl-4-isothiazolin-3-one	LOW (LogKOW = -0.8767)
2-octyl-4-isothiazolin-3-one	LOW (LogKOW = 2.561)
C.I. Pigment White 6	LOW (BCF = 10)
C.I. Pigment Yellow 3	MEDIUM (LogKOW = 4.1171)
C.I. Pigment Yellow 1	MEDIUM (LogKOW = 3.9388)
C.I. Pigment Green 7	LOW (BCF = 74)
C.I. Pigment Blue 15	LOW (BCF = 11)
C.I. Pigment Yellow 74	LOW (LogKOW = 2.9756)

Mobility in soil

Ingredient	Mobility
glycerol	HIGH (Log KOC = 1)
carbendazim	LOW (Log KOC = 175.8)
2-methyl-4-isothiazolin-3-one	LOW (Log KOC = 27.88)
2-octyl-4-isothiazolin-3-one	LOW (Log KOC = 2120)
C.I. Pigment White 6	LOW (Log KOC = 23.74)
C.I. Pigment Yellow 3	LOW (Log KOC = 460.5)
C.I. Pigment Yellow 1	LOW (Log KOC = 278.5)
C.I. Pigment Blue 15	LOW (Log KOC = 10000000000)
C.I. Pigment Yellow 74	LOW (Log KOC = 88.95)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

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

Continued...

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

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

Not Applicable

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

Product name	Group
gum arabic	Not Available
aluminium hydroxide	Not Available
glycerol	Not Available
bentonite	Not Available
carbendazim	Not Available
magnesium nitrate	Not Available
2-methyl-4-isothiazolin-3-one	Not Available
isothiazolinones, mixed	Not Available
2-octyl-4-isothiazolin-3-one	Not Available
water	Not Available
C.I. Pigment White 6	Not Available
C.I. Pigment Yellow 3	Not Available
C.I. Pigment Yellow 1	Not Available
ferric hydroxide	Not Available
C.I. Pigment Red 101	Not Available
C.I. Pigment Red 21	Not Available
C.I. Pigment Green 7	Not Available
C.I. Pigment Violet 23	Not Available
C.I. Pigment Blue 15	Not Available
C.I. Pigment Red 122	Not Available
C.I. Pigment Blue 29	Not Available
C.I. Pigment Blue 1	Not Available
C.I. Pigment Black 7	Not Available
C.I. Pigment Red 4	Not Available
C.I. Pigment Red 146	Not Available
C.I. Pigment Yellow 65	Not Available
C.I. Pigment Yellow 74	Not Available
C.I. Pigment Blue 28	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
gum arabic	Not Available
aluminium hydroxide	Not Available
glycerol	Not Available
bentonite	Not Available
carbendazim	Not Available
magnesium nitrate	Not Available
2-methyl-4-isothiazolin-3-one	Not Available
isothiazolinones, mixed	Not Available
2-octyl-4-isothiazolin-3-one	Not Available
water	Not Available
C.I. Pigment White 6	Not Available
C.I. Pigment Yellow 3	Not Available
C.I. Pigment Yellow 1	Not Available
ferric hydroxide	Not Available
C.I. Pigment Red 101	Not Available
C.I. Pigment Red 21	Not Available
C.I. Pigment Green 7	Not Available

Continued...

Product name	Ship Type
C.I. Pigment Violet 23	Not Available
C.I. Pigment Blue 15	Not Available
C.I. Pigment Red 122	Not Available
C.I. Pigment Blue 29	Not Available
C.I. Pigment Blue 1	Not Available
C.I. Pigment Black 7	Not Available
C.I. Pigment Red 4	Not Available
C.I. Pigment Red 146	Not Available
C.I. Pigment Yellow 65	Not Available
C.I. Pigment Yellow 74	Not Available
C.I. Pigment Blue 28	Not Available

SECTION 15 Regulatory information

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

gum arabic is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

aluminium hydroxide 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)

glycerol 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)

bentonite 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)

carbendazim 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 7
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

magnesium nitrate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
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 2A: Probably carcinogenic to humans

2-methyl-4-isothiazolin-3-one 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 6
Australian Inventory of Industrial Chemicals (AIIC)

isothiazolinones, mixed is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

2-octyl-4-isothiazolin-3-one 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 6
Australian Inventory of Industrial Chemicals (AIIC)

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment White 6 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

Continued...

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 Yellow 3 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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

Australian Inventory of Industrial Chemicals (AIIC)

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)

C.I. Pigment Red 101 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 21 is found on the following regulatory lists

Not Applicable

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 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 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 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 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 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 Black 7 is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

C.I. Pigment Red 4 is found on the following regulatory lists

Continued...

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

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 Yellow 65 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment Yellow 74 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment Blue 28 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 WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (isothiazolinones, mixed; C.I. Pigment Red 21)
Canada - DSL	No (C.I. Pigment Red 21)
Canada - NDSL	No (gum arabic; aluminium hydroxide; glycerol; bentonite; carbendazim; magnesium nitrate; 2-methyl-4-isothiazolin-3-one; isothiazolinones, mixed; 2-octyl-4-isothiazolin-3-one; water; C.I. Pigment White 6; C.I. Pigment Yellow 3; C.I. Pigment Yellow 1; C.I. Pigment Red 101; C.I. Pigment Green 7; C.I. Pigment Violet 23; C.I. Pigment Blue 15; C.I. Pigment Red 122; C.I. Pigment Blue 29; C.I. Pigment Blue 1; C.I. Pigment Black 7; C.I. Pigment Red 4; C.I. Pigment Red 146; C.I. Pigment Yellow 65; C.I. Pigment Yellow 74; C.I. Pigment Blue 28)
China - IECSC	No (C.I. Pigment Red 21)
Europe - EINEC / ELINCS / NLP	No (isothiazolinones, mixed)
Japan - ENCS	No (gum arabic; bentonite; C.I. Pigment Blue 28)
Korea - KECI	No (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 (isothiazolinones, mixed)
Taiwan - TCSI	No (C.I. Pigment Red 21)
Mexico - INSQ	No (isothiazolinones, mixed; C.I. Pigment Yellow 3; C.I. Pigment Red 21; C.I. Pigment Green 7; C.I. Pigment Red 122; C.I. Pigment Blue 1; C.I. Pigment Red 146; C.I. Pigment Yellow 65)
Vietnam - NCI	Yes
Russia - FBEPH	No (gum arabic; C.I. Pigment Red 21; C.I. Pigment Blue 1; C.I. Pigment Red 146; C.I. Pigment Yellow 65)
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	10/03/2023
Initial Date	06/05/2020

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	20/08/2021	Classification change due to full database hazard calculation/update.
5.1	10/03/2023	Classification change due to full database hazard calculation/update.

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

- 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