

Jasco Pty Limited

Chemwatch: **5398-64**Version No: **3.1.1.1**

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: **05/07/2020**Print Date: **05/07/2020**L.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Reeves Watercolour 12ml Paint	
Synonyms	Available	
Other means of identification	Not Available	

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

Details of the 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	sales@jasco.com.au	

Emergency telephone number

Association / Organisation	Australian Poisons Centre	
Emergency telephone numbers	13 11 26 (24/7)	
Other emergency telephone numbers	Not Available	

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable	
Classification ^[1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Skin Sensitizer Category 1, Respiratory Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation)	
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI	

Label elements

Hazard pictogram(s)





SIGNAL WORD

DANGER

Hazard statement(s)

H315

Causes skin irritation.

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H319	Causes serious eye irritation.	
H317	lay cause an allergic skin reaction.	
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.	
H335	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/face protection.	
P285	In case of inadequate ventilation wear respiratory protection.	
P272	P272 Contaminated work clothing should not be allowed out of the workplace.	

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.	
P321	Specific treatment (see advice on this label).	
P342+P311	f experiencing respiratory symptoms: Call a POISON CENTER or doctor/physician.	
P362	te off contaminated clothing and wash before reuse.	
P302+P352	F 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.	
P312	Call a POISON CENTER or doctor/physician if you feel unwell.	
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.	
P337+P313	If eye irritation persists: Get medical advice/attention.	

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
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	<u>bentonite</u>
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
6486-23-3	NotSpec	C.I. Pigment Yellow 3
2512-29-0	NotSpec	C.I. Pigment Yellow 1
20344-49-4	NotSpec	ferric hydroxide

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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

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 a 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

Fire Incompatibility

• Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may

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Advice for firefighters

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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 (CO2) hydrogen chloride phosgene nitrogen oxides (NOx) phosphorus oxides (POx) metal oxides other pyrolysis products typical of burning organic material. May emit corrosive 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 (Na2S2O5) or sodium bisulfite (NaHSO3), or 12% sodium sulfite (Na2SO3) and 8% hydrochloric acid (HCI). 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

- ▶ 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.

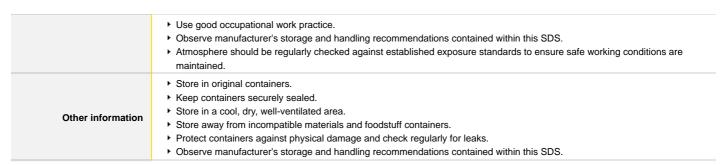
▶ DO NOT allow material to contact humans, exposed food or food utensils.

- Safe handling

 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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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	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 Black 7	Carbon black	3 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
aluminium hydroxide	Aluminum hydroxide	8.7 mg/m3	73 mg/m3	440 mg/m3
glycerol	Glycerine (mist); (Glycerol; Glycerin)	45 mg/m3	180 mg/m3	1,100 mg/m3
magnesium nitrate	Magnesium nitrate; (Magnesium(II) nitrate (1:2))	30 mg/m3	330 mg/m3	2,000 mg/m3
magnesium nitrate	Magnesium(II) nitrate (1:2), hexahydrate	16 mg/m3	180 mg/m3	1,100 mg/m3
C.I. Pigment White 6	Titanium oxide; (Titanium dioxide)	30 mg/m3	330 mg/m3	2,000 mg/m3
ferric hydroxide	Ferric hydroxide; (Iron(III) hydroxide)	30 mg/m3	330 mg/m3	2,000 mg/m3
ferric hydroxide	Iron oxide; (Ferric oxide)	15 mg/m3	360 mg/m3	2,200 mg/m3
ferric hydroxide	Iron hydroxide oxide	24 mg/m3	260 mg/m3	1,600 mg/m3
C.I. Pigment Red 101	Iron oxide; (Ferric oxide)	15 mg/m3	360 mg/m3	2,200 mg/m3
C.I. Pigment Black 7	Carbon black	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
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

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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	Not Available	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	Е	≤ 0.01 mg/m³
aluminium hydroxide	Е	≤ 0.01 mg/m³
bentonite	Е	≤ 0.01 mg/m³
carbendazim	С	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
magnesium nitrate	Е	≤ 0.01 mg/m³
2-methyl-4-isothiazolin-3-one	D	> 0.01 to ≤ 0.1 mg/m³
isothiazolinones, mixed	Е	≤ 0.1 ppm
2-octyl-4-isothiazolin-3-one	Е	≤ 0.1 ppm
C.I. Pigment Yellow 3	Е	≤ 0.01 mg/m³
C.I. Pigment Yellow 1	Е	≤ 0.01 mg/m³
C.I. Pigment Red 21	С	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
C.I. Pigment Blue 1	Е	≤ 0.01 mg/m³
C.I. Pigment Blue 28	Е	≤ 0.01 mg/m³
Notes:	potency and the adverse health outcomes associ	issigning chemicals into specific categories or bands based on a chemical's iated with exposure. The output of this process is an occupational exposure posure concentrations that are expected to protect worker health.

MATERIAL DATA

Exposure 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.

Appropriate engineering controls

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

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"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.

Personal protection









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

Wear chemical protective gloves, e.g. PVC.

▶ Wear safety footwear or safety gumboots, e.g. Rubber

Hands/feet protection

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:

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Material CI	PI
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Respiratory protection

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

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

Required Minimum	Half-Face	Full-Face	Powered Air
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BUTYL	C
NATURAL RUBBER	С
NATURAL+NEOPRENE	С
NEOPRENE	С
NITRILE	С
PVA	С
VITON	С

^{*} CPI - Chemwatch Performance Index

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.

Protection Factor	Respirator	Respirator	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	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

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled

Ingestion

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.

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.

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.

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.

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.

Chronic

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.

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- ▶ 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.

Reeves Watercolour 12ml	TOXICITY	IRRITATION
Paint	Not Available	Not Available
	TOXICITY	IRRITATION
gum arabic	Oral (rat) LD50: >16000 mg/kg ^[2]	Eye (rabbit): 36 mg/5h SEVERE
	TOXICITY	IRRITATION
aluminium hydroxide	Oral (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
glycerol	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available
	TOXICITY	IRRITATION
bentonite	Oral (rat) LD50: >5000 mg/kg ^[2]	Not Available
	TOXICITY	IRRITATION
	dermal (rat) LD50: 2000 mg/kg ^[2]	Eye (rabbit): non-irritating *
carbendazim	Oral (rat) LD50: >5050 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): non-irritating *
		Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
magnesium nitrate	dermal (rat) LD50: >5000 mg/kg ^[1]	Eye (rabbit): 500 mg/24h - mild
	Oral (rat) LD50: >2000 mg/kg ^[1]	Skin (rabbit): 500 mg/24h - mild
	TOXICITY	IRRITATION
2-methyl- 4-isothiazolin-3-one	dermal (rat) LD50: 242 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
4-130thia20iii1-3-01ie	Oral (rat) LD50: 120 mg/kg ^[1]	Skin: adverse effect observed (corrosive) ^[1]
	TOXICITY	IRRITATION
	dermal (rat) LD50: >1008 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
isothiazolinones, mixed	Oral (rat) LD50: 53 mg/kg ^[2]	Skin: adverse effect observed (corrosive) ^[1]
		Skin: adverse effect observed (irritating) ^[1]
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 690 mg/kg ^[2]	Eye (rabbit): 0.5% non irritant
2-octyl-4-isothiazolin-3-one	Oral (rat) LD50: 550 mg/kg ^[2]	Eye (rabbit): 45% conc CORROSIVE
		Eye (rabbit): 5% conc moderate

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		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
water	Oral (rat) LD50: >90000 mg/kg ^[2]	Not Available
	TOXICITY	IRRITATION
	dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
C.I. Pigment White 6	Oral (rat) LD50: >2000 mg/kg ^[1]	Skin (rabbit)
		Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
C.I. Pigment Yellow 3	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[1]	
	TOXICITY	IRRITATION
C.I. Pigment Yellow 1	dermal (rat) LD50: >2000 mg/kg ^[1]	Non-irritating/non-sensitising
<u> </u>	Oral (rat) LD50: >2000 mg/kg ^[1]	
	TOXICITY	IRRITATION
ferric hydroxide	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available
	TOXICITY	IRRITATION
C.I. Pigment Red 101	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available
	TOXICITY	IRRITATION
C.I. Pigment Red 21	Not Available	Not Available
01 8: 7	TOXICITY	IRRITATION
C.I. Pigment Green 7	Oral (rat) LD50: >2000 mg/kg ^[1]	Not Available
	TOXICITY	IRRITATION
N. Diamana Walat oo	Oral (rat) LD50: 5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
C.I. Pigment Violet 23		Skin (rabbit): Non-irritating *
		Skin: no adverse effect observed (not irritating) $^{[1]}$
	TOXICITY	IRRITATION
C.I. Pigment Blue 15	Oral (rat) LD50: >10,000 mg/kg ^[2]	Eye (human): non-irritant
		Skin (human): non-irritant
	TOXICITY	IRRITATION
C.I. Pigment Red 122	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[1]	
	TOXICITY	IRRITATION
C.I. Pigment Blue 29	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available
	TOXICITY	IRRITATION
C.I. Pigment Blue 1	Oral (rat) LD50: >5000 mg/kg ^[2]	Not Available
	TOXICITY	IRRITATION
C.I. Pigment Black 7	dermal (rat) LD50: >2000 mg/kg ^[1]	

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	Oral (rat) LD50: >15400 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
C.I. Pigment Red 4	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
C.I. Pigment Red 146	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[1]	
	TOXICITY	IRRITATION
C.I. Pigment Yellow 65	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: non-irritating *
	Oral (rat) LD50: >2000 mg/kg ^[1]	Skin: non-irritatng *
	TOXICITY	IRRITATION
C.I. Pigment Yellow 74	Oral (rat) LD50: >1500 mg/kg ^[2]	Eye (human): non irritant
		Skin (human): non irritant
C.I. Diamont Div. 22	TOXICITY	IRRITATION
C.I. Pigment Blue 28	Not Available	Not Available
Legend:	Value obtained from Europe ECHA Registered S	Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS.

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.

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.

GLYCEROL

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

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1180 mg/kg bw/day).

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.

BENTONITE

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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.

Hypokalaemia and microcytic iron-deficiency anaemia may occur in patients after repeat doses of clay. Chronic ingestion has been reported to cause myositis.

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 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.

CARBENDAZIM

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

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MAGNESIUM NITRATE

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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 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)

Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man.

appropriate studies with similar materials using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.

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.

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.

2-METHYL4-ISOTHIAZOLIN-3-ONE
Many countries are placing regulatory pr

For titanium dioxide:

This concern is raised, generally, on the basis of

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.

Considered to be a minor sensitiser in Kathon CG (1) (1). Bruze et al - Contact Dermatitis 20: 219-39, 1989

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

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

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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.

Substance has been investigated as a mutagen, tumorigen and primary irritant.

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

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

After cleavage of the azo linkage by bacteria, the component aromatic amines are absorbed in the intestine and excreted in the urine. Twenty-two of the component amines are recognised as potential human carcinogens, and/or several of them have shown carcinogenic potential on experimental animals. Sulfonation of the dye reduces the toxicity by enhancement of the excretion. The component amines which may be released from azo dyes are mostly aromatic amines (compounds where an amine group or amine-generating group(s) are connected to an aryl moiety). In general, aromatic amines known as carcinogenic may be grouped into five groups

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

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

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

C.I. PIGMENT YELLOW 3

No carcinogenic effects observed during a 43 day test animal feeding study on Pigment Violet 23. [Manufacturer]

C.I. PIGMENT RED 122

551acrid

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]

GUM ARABIC & 2-METHYL-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

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ISOTHIAZOLINONES, MIXED & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE & C.I. PIGMENT BLUE 28 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-METHYL4-ISOTHIAZOLIN-3-ONE &
ISOTHIAZOLINONES,
MIXED & 2-OCTYL4-ISOTHIAZOLIN-3-ONE &
C.I. PIGMENT BLUE 28

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

ALUMINIUM HYDROXIDE &
BENTONITE & 2-METHYL4-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.

No significant acute toxicological data identified in literature search.

MAGNESIUM NITRATE & 2-METHYL4-ISOTHIAZOLIN-3-ONE & ISOTHIAZOLINONES, MIXED

PIGMENT BLUE 28

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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 & 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: X − Data either not available or does not fill the criteria for classification

✓ – Data available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Reeves Watercolour 12ml Paint	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
aluminium hydroxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.134mg/	_ 2
	EC50	48	Crustacea	0.7364mg/L	2
	EC50	72	Algae or other aquatic plants	0.001-0.05mg/L	2

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	NOEC	168	Crustacea	0.001-mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
glycerol	LC50	96	Fish	>0.011-mg/L	2
	EC50	96	Algae or other aquatic plants	77712.039mg/L	3
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
bentonite	LC50	96	Fish	19000mg/L	4
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.007mg/L	4
carbendazim	EC50	48	Crustacea	0.02mg/L	4
	EC50	96	Algae or other aquatic plants	3.945mg/L	3
	NOEC	480	Crustacea	<0.0031mg/L	4
	ENDPOINT	TEST DUD ATION (UD)	SPECIES	VALUE	SOURC
	LC50	TEST DURATION (HR)	Fish	1-378mg/L	2
magnesium nitrate	EC50	48	Crustacea	490mg/L	2
		i I	i i	1	
	NOEC	720	Fish	58mg/L	2
2-methyl-	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.07mg/L	4
	EC50	48	Crustacea	0.18mg/L	4
4-isothiazolin-3-one	EC50	72	Algae or other aquatic plants	0.05mg/L	4
	EC10	72	Algae or other aquatic plants	0.0346mg/L	2
	NOEC	96	Algae or other aquatic plants	0.01mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	LC50	96	Fish	0.129mg/L	2
isothiazolinones, mixed	EC50	48	Crustacea	0.007mg/L	2
	EC50	72	Algae or other aquatic plants	0.0063mg/L	2
	NOEC	48	Algae or other aquatic plants	0.00049mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	LC50	96	Fish	0.047mg/L	4
	EC50	48	Crustacea	0.18mg/L	4
?-octyl-4-isothiazolin-3-one	EC50	96	Algae or other aquatic plants	0.146mg/L	3
	BCF	1608	Fish	0.05mg/L	4
	NOEC	504	Crustacea	0.035mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
water	LC50	96	Fish	897.520mg/L	3
	EC50	96	Algae or other aquatic plants	8768.874mg/L	3
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	LC50	96	Fish	>1-mg/L	2
C.I. Pigment White 6	EC50	48	Crustacea	>1-mg/L	2
c ig.iione iiinio 0	EC50	72	Algae or other aquatic plants	5.83mg/L	4
	NOEC	336	Fish	0.089mg/L	4
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	LC50	96	Fish	>1mg/L	2
C.I. Pigment Yellow 3	EC50	48	Crustacea	>100mg/L	2
	EC50	96	Algae or other aquatic plants	2.610mg/L	3
	NOEC	72	Algae or other aquatic plants	1mg/L	2

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ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	i i i i i i i i i i i i i i i i i i i	I I	1	2
	i I	i I		2
EC50	96	Algae or other aquatic plants	-	3
NOEC	72	Algae or other aquatic plants	1mg/L	2
ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
LC50	96	Fish	0.05mg/L	2
EC50	48	Crustacea	5.11mg/L	2
EC50	72	Algae or other aquatic plants	18mg/L	2
NOEC	504	Fish	0.52mg/L	2
LC50	96	Fish	0.05mg/L	2
EC50	48	Crustacea	5.11mg/L	2
EC50	72	Algae or other aquatic plants	18mg/L	2
NOEC	504	Fish	0.52mg/L	2
ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
LC50	96	Fish	0.05mg/L	2
EC50	48	Crustacea	5.11mg/L	2
EC50	72	Algae or other aquatic plants	18mg/L	2
NOEC	504	Fish	0.52mg/L	2
ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
Not Available	Not Available	Not Available	Not Available	Not Availab
ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	153.6mg/L	2
EC50	72	Algae or other aquatic plants	>100mg/L	2
NOEC	504	Crustacea	>=1mg/L	2
ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	>100mg/L	2
EC50	72	Algae or other aquatic plants	>100mg/L	2
EC0	48	Crustacea	>=100mg/L	2
NOEC	72	Algae or other aquatic plants	>=100mg/L	2
ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
LC50	96	Fish	>3-200mg/L	2
EC50	48	Crustacea	>100mg/L	2
EC50	72	Algae or other aquatic plants	>100mg/L	2
NOEC	504	Crustacea	>1mg/L	2
ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	>100mg/L	2
NOEC	72 504	Algae or other aquatic plants Crustacea	>10mg/L >0.02mg/L	2
NOLO		O usidota	>0.02IIIg/L	1
ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
ENDPOINT LC50 EC50	TEST DURATION (HR) 96 48	SPECIES Fish Crustacea	VALUE >=90mg/L >21mg/L	2 2
	NOEC ENDPOINT LC50 EC50 NOEC LC50 EC50 NOEC ENDPOINT LC50 EC50 NOEC ENDPOINT LC50 EC50 NOEC ENDPOINT LC50 EC50 NOEC ENDPOINT LC50 EC50 NOEC ENDPOINT LC50 EC50 NOEC	EC50	EC50 48 Crustacea EC50 96 Algae or other aquatic plants NOEC 72 Algae or other aquatic plants ENDPOINT TEST DURATION (HR) SPECIES ENDPOINT TEST DURATION (HR) SPECIES EC50 48 Crustacea EC50 72 Algae or other aquatic plants NOEC 504 Fish EC50 48 Crustacea EC50 72 Algae or other aquatic plants NOEC 504 Fish ENDPOINT TEST DURATION (HR) SPECIES EC50 72 Algae or other aquatic plants EC50 48 Crustacea EC50 72 Algae or other aquatic plants NOEC 504 Fish ENDPOINT TEST DURATION (HR) SPECIES NOEC 504 Fish ENDPOINT TEST DURATION (HR) SPECIES EC50 48 Crustacea EC50 72 Algae or other aquatic plan	EC50 48 Crustacea >+100mg/L EC50 96 Algae or other aquatic plants 3,244mg/L NOEC 72 Algae or other aquatic plants 1mg/L ENDPOINT TEST DURATION (HR) SPECIES VALUE LC50 96 Fish 0.05mg/L EC50 48 Crustacea 5.11mg/L EC50 72 Algae or other aquatic plants 18mg/L LC50 96 Fish 0.05mg/L EC50 48 Crustacea 5.11mg/L EC50 48 Crustacea 5.11mg/L EC50 72 Algae or other aquatic plants 18mg/L LC50 96 Fish 0.05mg/L EC50 72 Algae or other aquatic plants 18mg/L LC50 96 Fish 0.05mg/L EC50 48 Crustacea 5.11mg/L EC50 48 Crustacea 5.11mg/L EC50 72 Algae or other aquatic plants 18mg/L

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		I	Crustacea	>=26mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
C.I. Pigment Blue 1	Not Available	Not Available	Not Available	Not Available	Not Availab
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
C.I. Pigment Black 7	EC50	72	Algae or other aquatic plants	>10-mg/L	2
-	EC10	72	Algae or other aquatic plants	>10-mg/L	2
-	NOEC	96	Fish	>=1-mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
C.I. Pigment Red 4	EC50	48	Crustacea	>100mg/L	2
-	NOEC	72	Algae or other aquatic plants	>0.006mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
-	LC50	96	Fish	>100mg/L	2
C.I. Pigment Red 146	EC50	48	Crustacea	>100mg/L	2
-	EC50	72	Algae or other aquatic plants	>1mg/L	2
-	NOEC	72	Algae or other aquatic plants	1mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
	LC50	96	Fish	>1mg/L	2
C.I. Pigment Yellow 65	EC50	48	Crustacea	>100mg/L	2
-	EC50	72	Algae or other aquatic plants	>100mg/L	2
-	NOEC	72	Algae or other aquatic plants	1mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
0 B'mm and Wallana 74	LC50	96	Fish	12.405mg/L	3
C.I. Pigment Yellow 74	EC50	96	Algae or other aquatic plants	25.568mg/L	3
	NOEC	504	Crustacea	>1mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOUR
C.I. Pigment Blue 28	Not Available	Not Available	Not Available	Not Available	Not Availat

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

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Reeves Watercolour 12ml Paint

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)		
water	LOW (LogKOW = -1.38)		
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 (KOC = 1)		
carbendazim	LOW (KOC = 175.8)		
2-methyl-4-isothiazolin-3-one	LOW (KOC = 27.88)		
2-octyl-4-isothiazolin-3-one	LOW (KOC = 2120)		
water	LOW (KOC = 14.3)		
C.I. Pigment White 6	LOW (KOC = 23.74)		
C.I. Pigment Yellow 3	LOW (KOC = 460.5)		
C.I. Pigment Yellow 1	LOW (KOC = 278.5)		
C.I. Pigment Blue 15	LOW (KOC = 10000000000)		
C.I. Pigment Yellow 74	LOW (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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

GUM ARABIC IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

ALUMINIUM HYDROXIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

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

GLYCEROL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

BENTONITE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

CARBENDAZIM IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

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

Chemical Footprint Project - Chemicals of High Concern List

MAGNESIUM NITRATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

2-METHYL-4-ISOTHIAZOLIN-3-ONE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

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

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 Inventory of Chemical Substances (AICS)

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

WATER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT WHITE 6 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

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 YELLOW 3 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT YELLOW 1 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

FERRIC HYDROXIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

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

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

C.I. PIGMENT RED 101 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

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

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

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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 Inventory of Chemical Substances (AICS)

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

C.I. PIGMENT VIOLET 23 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT BLUE 15 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

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

C.I. PIGMENT RED 122 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT BLUE 29 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT BLUE 1 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

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

C.I. PIGMENT BLACK 7 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

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

Australia Inventory of Chemical Substances (AICS)

Chemical Footprint Project - Chemicals of High Concern List

C.I. PIGMENT RED 146 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT YELLOW 65 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT YELLOW 74 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

Chemical Footprint Project - Chemicals of High Concern List

C.I. PIGMENT BLUE 28 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australia Inventory of Chemical Substances (AICS)

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

National Inventory Status

National Inventory	Status	
Australia - AICS	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)	

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Reeves Watercolour 12ml Paint

Europe - EINEC / ELINCS / NLP	No (isothiazolinones, mixed)		
Japan - ENCS	No (gum arabic; bentonite; isothiazolinones, mixed; 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 - ARIPS	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 and are not exempt from listing(see specific ingredients in brackets)		

SECTION 16 OTHER INFORMATION

Revision Date	05/07/2020
Initial Date	05/06/2020

SDS Version Summary

Version	Issue Date	Sections Updated
2.1.1.1	05/06/2020	Environmental
3.1.1.1	05/07/2020	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Chronic Health, Classification, Disposal, Engineering Control, Environmental, Fire Fighter (extinguishing media), Fire Fighter (fire/explosion hazard), Fire Fighter (fire fighting), Fire Fighter (fire incompatibility), First Aid (eye), First Aid (inhaled), First Aid (skin), First Aid (swallowed), Handling Procedure, Ingredients, Instability Condition, Personal Protection (other), Personal Protection (Respirator), Personal Protection (eye), Personal Protection (hands/feet), Spills (major), Spills (minor), Storage (storage incompatibility), Storage (storage requirement), Storage (suitable container), Transport, Name

Other information

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

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

Definitions and abbreviations

PC-TWA: Permissible Concentration-Time Weighted Average

PC-STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

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

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