

Reeves Gouache 22ml Paint

Jasco Pty Limited

Chemwatch Hazard Alert Code: 3

Chemwatch: 5397-26

Issue Date: 05/07/2020

Version No: 2.1.1.1

Print Date: 05/08/2020

Safety Data Sheet according to WHS and ADG requirements

L.GHS.AUS.EN

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

Product Identifier

Product name	Reeves Gouache 22ml Paint
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains C.I. Pigment Red 4, C.I. Pigment Blue 1 and C.I. Pigment Blue 27)
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 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]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Germ cell mutagenicity Category 2, Carcinogenicity Category 1A, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - repeated exposure Category 2, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
SIGNAL WORD	DANGER

Reeves Gouache 22ml Paint

Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H335	May cause respiratory irritation.
H373	May cause damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P310	Immediately call a POISON CENTER or doctor/physician.
P321	Specific treatment (see advice on this label).
P362	Take off contaminated clothing and wash before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: 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
9004-53-9	<30	<u>dextrins</u>
7631-86-9	<10	<u>silica amorphous</u>
7727-43-7	<30	<u>barium sulfate</u>
13463-67-7	<20	<u>titanium dioxide</u>
6486-23-3	<20	<u>C.I. Pigment Yellow 3</u>
2512-29-0	<20	<u>C.I. Pigment Yellow 1</u>

Reeves Gouache 22ml Paint

20344-49-4	<20	<u>ferric hydroxide</u>
1309-37-1	<20	<u>ferric oxide</u>
1328-53-6	<20	<u>C.I. Pigment Green 7</u>
147-14-8	<20	<u>C.I. Pigment Blue 15</u>
1325-87-7	<20	<u>C.I. Pigment Blue 1</u>
1333-86-4	<20	<u>carbon black</u>
2814-77-9	<20	<u>C.I. Pigment Red 4</u>
6410-26-0	<20	<u>C.I. Pigment Red 21</u>
14038-43-8	<20	<u>C.I. Pigment Blue 27</u>
56-81-5	<5	<u>glycerol</u>
52-51-7	0.05	<u>2-bromo-2-nitropropan-1,3-diol</u>
13463-41-7	<0.2	<u>zinc pyrithione</u>
1314-13-2	<0.2	<u>zinc oxide</u>
7732-18-5	balance	<u>water</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY. ▶ For advice, contact a Poisons Information Centre or a doctor. ▶ Urgent hospital treatment is likely to be needed. ▶ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▶ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▶ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. <p>Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:</p> <ul style="list-style-type: none"> ▶ INDUCE vomiting with fingers down the back of the throat, ONLY IF CONSCIOUS. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. <p>NOTE: Wear a protective glove when inducing vomiting by mechanical means.</p>

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
for copper intoxication:

- ▶ Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
- ▶ Administer egg white and other demulcents.
- ▶ Maintain electrolyte and fluid balances.
- ▶ Morphine or meperidine (Demerol) may be necessary for control of pain.
- ▶ If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the

supplier's recommendations.

- ▶ Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
- ▶ If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.
- ▶ It is unlikely that methylene blue would be effective against the occasional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
- ▶ Institute measures for impending renal and hepatic failure.

[GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]

- ▶ A role for activated charcoals for emesis is, as yet, unproven.
- ▶ In severe poisoning CaNa₂EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]

For acute or short term repeated exposures to iron and its derivatives:

- ▶ Always treat symptoms rather than history.
- ▶ In general, however, toxic doses exceed 20 mg/kg of ingested material (as elemental iron) with lethal doses exceeding 180 mg/kg.
- ▶ Control of iron stores depend on variation in absorption rather than excretion. Absorption occurs through aspiration, ingestion and burned skin.
- ▶ Hepatic damage may progress to failure with hypoprothrombinaemia and hypoglycaemia. Hepatorenal syndrome may occur.
- ▶ Iron intoxication may also result in decreased cardiac output and increased cardiac pooling which subsequently produces hypotension.
- ▶ Serum iron should be analysed in symptomatic patients. Serum iron levels (2-4 hrs post-ingestion) greater than 100 ug/dL indicate poisoning with levels, in excess of 350 ug/dL, being potentially serious. Emesis or lavage (for obtunded patients with no gag reflex) are the usual means of decontamination.
- ▶ Activated charcoal does not effectively bind iron.
- ▶ Catharsis (using sodium sulfate or magnesium sulfate) may only be used if the patient already has diarrhoea.
- ▶ Deferoxamine is a specific chelator of ferric (3+) iron and is currently the antidote of choice. It should be administered parenterally. [Ellenhorn and Barceloux: Medical Toxicology]

Periodic medical surveillance should be carried out on persons in occupations exposed to the manufacture or bulk handling of the product and this should include hepatic function tests and urinalysis examination. [ILO Encyclopaedia]

- ▶ After ingestion of barium acid salts, severe gastro-intestinal irritation followed by muscle twitching, progressive flaccid paralysis and severe hypokalaemia and hypertension, occurs.
- ▶ Respiratory failure, renal failure and occasional cardiac dysrhythmias may result from an acute ingestion.
- ▶ Use sodium sulfate as a cathartic. Add 5-10 gm of sodium sulfate to lavage solution or as fluid supplement to Ipecac syrup (the sulfate salt is not absorbed)
- ▶ Monitor cardiac rhythm and serum potassium closely to establish the trend over the first 24 hours. Large doses of potassium may be needed to correct the hypokalaemia.
- ▶ Administer generous amounts of fluid replacement but monitor the urine and serum for evidence of renal failure. [Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- ▶ Foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.
- ▶ Water spray or fog - Large fires only.

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 result
-----------------------------	--

Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▶ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. ▶ 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	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered a significant fire risk, however containers may burn. <p>Decomposition may produce toxic fumes of:</p> <p>carbon dioxide (CO₂) hydrogen chloride phosgene nitrogen oxides (NO_x) phosphorus oxides (PO_x) sulfur oxides (SO_x) silicon dioxide (SiO₂)</p>

Reeves Gouache 22ml Paint

metal oxides
other pyrolysis products typical of burning organic material.
Decomposes at high temperatures to produce barium oxide. Barium oxide is strongly alkaline and, upon contact with water, is exothermic. When barium oxide reacts with oxygen to give a peroxide, there is a fire and explosion risk.
A fire in bulk finely divided carbon may not be obviously visible unless the material is disturbed and sparks appear. A straw broom may be useful to produce the disturbance.
Explosion and Ignition Behaviour of Carbon Black with Air

Lower Limit for Explosion:	50 g/m3 (carbon black in air)
Maximum Explosion Pressure:	10 bar
Maximum Rate of Pressure Rise:	30-100 bar/sec
Minimum Ignition Temperature:	315 deg. C.
Ignition Energy:	>1 kJ
Glow Temperature:	500 deg. C. (approx.)

Notes on Test Methods:

Tests 1, 2 and 3 were conducted by Bergwerkeschaftliche Versuchstrecke, Dortmunde-Derne, using a 1 m3 vessel with two chemical igniters having an intensity of 5000 W.S.
Tests 1 and 2 results are confirmed by information in the Handbook of Powder Technology, Vol. 4 (P. Field)
In Test 4, a modified Godbert-Greenwald furnace was used. See U.S. Bureau of Mines, Report 5624, 1960, p.5, "Lab Equipment and Test Procedures".
Test 5 used a 1 m3 vessel with chemical igniters of variable intensity.
Test 6 was conducted in a laboratory oven. Active glowing appeared after 3 minutes exposure.
(European Committee for Biological Effects of Carbon Black) (2/84)

HAZCHEM •3Z

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked.
----------------------	--

Reeves Gouache 22ml Paint

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

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid strong acids, bases. ▶ 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	silica amorphous	Silica, fused	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated) (respirable dust)	2 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	barium sulfate	Barium sulphate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ferric hydroxide	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric oxide	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	C.I. Pigment Blue 27	Cyanides (as CN)	5 mg/m3	Not Available	Not Available	Not Available

Reeves Gouache 22ml Paint

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	zinc oxide	Zinc oxide (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	zinc oxide	Zinc oxide (fume)	5 mg/m3	10 mg/m3	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
silica amorphous	Silica gel, amorphous synthetic	18 mg/m3	200 mg/m3	1,200 mg/m3
silica amorphous	Silica, amorphous fumed	18 mg/m3	100 mg/m3	630 mg/m3
silica amorphous	Siloxanes and silicones, dimethyl, reaction products with silica; (Hydrophobic silicon dioxide, amorphous)	120 mg/m3	1,300 mg/m3	7,900 mg/m3
silica amorphous	Silica, amorphous fume	45 mg/m3	500 mg/m3	3,000 mg/m3
silica amorphous	Silica amorphous hydrated	18 mg/m3	740 mg/m3	4,500 mg/m3
barium sulfate	Barium sulfate	15 mg/m3	170 mg/m3	990 mg/m3
titanium dioxide	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
ferric oxide	Iron oxide; (Ferric oxide)	15 mg/m3	360 mg/m3	2,200 mg/m3
carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3
glycerol	Glycerine (mist); (Glycerol; Glycerin)	45 mg/m3	180 mg/m3	1,100 mg/m3
zinc oxide	Zinc oxide	10 mg/m3	15 mg/m3	2,500 mg/m3

Ingredient	Original IDLH	Revised IDLH
dextrins	Not Available	Not Available
silica amorphous	3,000 mg/m3	Not Available
barium sulfate	Not Available	Not Available
titanium dioxide	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
ferric oxide	2,500 mg/m3	Not Available
C.I. Pigment Green 7	Not Available	Not Available
C.I. Pigment Blue 15	Not Available	Not Available
C.I. Pigment Blue 1	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
C.I. Pigment Red 4	Not Available	Not Available
C.I. Pigment Red 21	Not Available	Not Available
C.I. Pigment Blue 27	25 mg/m3	Not Available
glycerol	Not Available	Not Available
2-bromo-2-nitropropan-1,3-diol	Not Available	Not Available
zinc pyrithione	Not Available	Not Available

Reeves Gouache 22ml Paint

zinc oxide	500 mg/m ³	Not Available
water	Not Available	Not Available

OCCUPATIONAL EXPOSURE BANDING

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

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

for zinc oxide:

Zinc oxide intoxication (intoxication zincale) is characterised by general depression, shivering, headache, thirst, colic and diarrhoea. Exposure to the fume may produce metal fume fever characterised by chills, muscular pain, nausea and vomiting. Short-term studies with guinea pigs show pulmonary function changes and morphologic evidence of small airway inflammation. A no-observed-adverse-effect level (NOAEL) in guinea pigs was 2.7 mg/m³ zinc oxide. Based on present data, the current TLV-TWA may be inadequate to protect exposed workers although known physiological differences in the guinea pig make it more susceptible to functional impairment of the airways than humans.

For amorphous crystalline silica (precipitated silicic acid):

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

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

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

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

For iron oxide (ferric oxide):

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

Barium sulfate has been identified as a nontoxic dust. However high dust levels have caused benign pneumoconiosis (baritosis). The TLV-TWA is thought to be protective against the risk of eye, nose and upper respiratory tract irritation and perhaps, pneumoconiosis.

An increased incidence of non-specific symptoms including headache, weakness, fatigue, anorexia and joint and muscle weakness has been reported to occur in mining and metallurgy workers exposed to 60-600 mg (as Mo). Some investigators have attributed gout and elevated uric acid concentration found in some Armenians to result from exposures to Armenian soils rich in molybdenum, whilst exposure has been implicated as a cause of bone disease amongst Indians. "These involvements are speculative". [US National Research Council]. As far as it is known, the recommended TLV-TWA incorporates a large margin of safety against potential pulmonary or systemic effects.

Animals exposed by inhalation to 10 mg/m³ titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

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

for pyrithiones:

CEL TWA: 1 mg/m³ (compare MAK value sodium pyrithione)

(CEL = Chemwatch Exposure Limit)

Sodium pyrithione is readily absorbed from the gastrointestinal tract and through the skin. Single high doses produce reversible damage of the peripheral nervous system of rodents; such damage is not seen in monkeys or dogs even after exposure for several days or a year. Sodium pyrithione is a mild irritant of the eyes and skin but does not cause sensitisation, phototoxicity or photosensitisation. Tested at low levels, the substance is not genotoxic (higher levels are cytotoxic).

Although no signs or neurotoxicity are evident in primates (even after intubation of high doses for a year), the possibility still exist that such effects might occur in man after exposure to high levels. The NOEL of 1.1 mg/m³ was determined in rats after a 13 week inhalation study and the MAK value reflects this finding.

Because the half-life of the substance is short in the initial kinetic phase, sodium pyrithione is classified in Peak limitation category II, 1. As a warning that substance can readily be absorbed through th skin, it is designated with and "H". Effects on reproduction occur only at maternally toxic doses leading to the Pregnancy risk group C categorisation.


for barium compounds:

The recommended TLV-TWA is based on satisfactory results achieved while employing an internal limit for barium nitrate at a national laboratory. It is not known what degree of added safety this limit incorporates.

The TLV-TWA for carbon black is recommended to minimise complaints of excessive dirtiness and applies only to commercially produced carbon blacks or to soots derived from combustion sources containing absorbed polycyclic aromatic hydrocarbons (PAHs). When PAHs are present in carbon black (measured as the cyclohexane-extractable fraction) NIOSH has established a REL-TWA of 0.1 mg/m³ and considers the material to be an occupational carcinogen.

The NIOSH REL-TWA was "selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs". This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.

Exposure controls

<p>Appropriate engineering controls</p>	<p>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.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>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.</p> <p>An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>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.</p> <table border="1" data-bbox="384 779 1489 1108"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="384 1153 1201 1339"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	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.)	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
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.)																				
Lower end of the range	Upper end of the range																				
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents																				
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity																				
3: Intermittent, low production.	3: High production, heavy use																				
4: Large hood or large air mass in motion	4: Small hood-local control only																				
<p>Personal protection</p>																					
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ 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] 																				
<p>Skin protection</p>	<p>See Hand protection below</p>																				
<p>Hands/feet protection</p>	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber 																				
<p>Body protection</p>	<p>See Other protection below</p>																				
<p>Other protection</p>	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. 																				

Reeves Gouache 22ml Paint

- ▶ 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 Gouache 22ml Paint

Material	CPI
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.

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-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	Ointment; does not mix 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)	Not Applicable	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	Immiscible	pH as a solution (1%)	9-10
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Bronchial and alveolar exudate are apparent in animals exposed to molybdenum by inhalation. Molybdenum fume may produce bronchial irritation and moderate fatty changes in liver and kidney.</p> <p>Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Ingestion may result in nausea, abdominal irritation, pain and vomiting</p>
Skin Contact	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p>
Chronic	<p>On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</p> <p>Because of similarities in structure to thalidomide, concerns have been raised about the potential teratogenicity of all</p>

phthalimides (the basic building block of phthalocyanine). Animal studies, in part, appear to support this proposition. The phthalimide fungicides are typically teratogens. Captan, for example, is teratogenic, foetotoxic, and/or embryotoxic in a variety of rodent species and has caused stillbirths in dogs. Folpet was teratogenic in rabbits, chicks, and hamsters. Captafol has been embryo- or foetotoxic in experimental animals. Captan, folpet, and captafol have all induced testicular degeneration in laboratory animals.

The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compared to crystalline silicas and are considered to be nuisance dusts.

When heated to high temperature and a long time, amorphous silica can produce crystalline silica on cooling. Inhalation of dusts containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to develop. Discrepancies between various studies showing that fibrosis associated with chronic exposure to amorphous silica and those that do not may be explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is either weakly fibrogenic or nonfibrogenic and that fibrosis is due to contamination by crystalline silica content.

Some workers may develop chronic occupational dermatitis (generally mild) through the handling of starch products. When starch is used as a lubricant in surgical gloves, small amounts, released into the patient during the course of surgery, have resulted in granulomas and peritonitis.

Many azo dyes have been found to be carcinogenic in laboratory animals, affecting the liver, urinary bladder and intestines. Specific toxicity effects in humans have not been established but some dyes are known to be mutagenic.

The simplest azo dyes, which raise concern, have an exocyclic amino-group that is the key to any carcinogenicity for it is this group which undergoes biochemical N-oxidation and further reaction to reactive electrophiles. The DNA adducts formed by covalent binding through activated nitrogen have been identified. However not all azo compounds possess this activity and delicate alterations to structure vary the potential of carcinogenicity / acid, reduces or eliminates the effect. Complex azo dyes consisting of more than one azo (N=N) linkage may be metabolised to produce complexed carcinogenic aromatic amines such as benzidine.

Benzidine and its metabolic derivatives have been detected in the urine of workers exposed to Direct azo dyes. An epidemiological study of silk dyers and painters with multiple exposures to benzidine based and other dyes indicate a strong association with bladder cancer.

Most organic azo dyes are potential skin sensitizers, the most important of which are para-phenylenediamine and its analogs. Water soluble azo dyes are more likely to cause clinical sensitisation than insoluble dyes. In addition to allergic eczematous contact dermatitis, color developing solutions have caused lichen planus like eruptions.

Repeated exposure to synthetic amorphous silicas may produce skin dryness and cracking.

Available data confirm the absence of significant toxicity by oral and dermal routes of exposure.

Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted in a number of species, at airborne concentrations ranging from 0.5 mg/m³ to 150 mg/m³. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m³. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m³. Differences in values may be due to particle size, and therefore the number of particles administered per unit dose. Generally, as particle size diminishes so does the NOAEL/ LOAEL. Exposure produced transient increases in lung inflammation, markers of cell injury and lung collagen content. There was no evidence of interstitial pulmonary fibrosis.

Chronic copper poisoning is rarely recognised in man although in one instance, at least, symptoms more commonly associated with exposures to mercury, namely infantile acro-dynia (pink disease), have been described. Tissue damage of mucous membranes may follow chronic dust exposure. A hazardous situation is exposure of a worker with the rare hereditary condition (Wilson's disease or hereditary hepatolenticular degeneration) to copper exposure which may cause liver, kidney, CNS, bone and sight damage and is potentially lethal. Haemolytic anaemia (a result of red-blood cell damage) is common in cows and sheep poisoned by copper derivatives. Overdosing of copper feed supplements has resulted in pigmentary cirrhosis of the liver. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products]

Persons, exposed for long periods to molybdenum oxides, suffer from anaemia. Animals exposed to certain insoluble molybdenum compounds show anorexia, diarrhoea, weight loss, listlessness, and liver and kidney damage. Molybdenum disturbs bone metabolism, giving rise to lameness, bone joint abnormalities, osteoporosis and high serum phosphatase levels. Cattle, rabbits, and chicks on high dietary levels of molybdenum exhibited deformities of joints of the extremities. Low molybdenum intake has been attributed to the high incidence of oesophageal cancer in South Africa among the Bantu of Transkei, in China and in Russia.

Chronic exposure of workers in Russian molybdenum-copper plant resulted in a fall in the albumin/globulin ratio owing to a rise in globulins (particularly alpha-globulins) which is interpreted as evidence of liver dysfunction with hyperbilirubinaemia. Hepatotoxic effects are also found in animals given molybdenum salts with a rise in alpha-globulin levels, hypoalbuminaemia and increased serum bilirubin reported. Other reported biochemical effects include an early depletive effect on tissue nicotinamide nucleotides, hyperaminoaciduria, reduction in red blood cell life-span and hyper-thyroidism. Industrial exposure to some insoluble molybdenum compounds is thought to have resulted in an increased incidence of weakness, fatigue, anorexia, headache and joint and muscular pain. Under the conditions of a 2-year inhalation study* there was equivocal evidence of carcinogenic activity of molybdenum trioxide in male rats, male mice and female mice based on a marginally significant positive trend of alveolar/bronchiolar adenoma or carcinoma. There was no evidence of carcinogenic activity in female rats exposed to 10, 30 and 100 mg/m³. Exposure of male and female rats to molybdenum trioxide by inhalation resulted in increased incidences of chronic alveolar inflammation, hyaline degeneration of the olfactory epithelium (females), hyaline degeneration of the respiratory epithelium and squamous metaplasia of the epiglottis. Exposure of female and male mice to molybdenum trioxide resulted in an increased incidence of metaplasia of the alveolar epithelium, histiocyte cellular inflammation (males), hyaline degeneration of the respiratory epithelium, hyaline degeneration of the olfactory epithelium (females), squamous metaplasia of the epiglottis, and hyperplasia of the larynx.

Guinea pigs exposed to molybdenum trioxide dust for 1 hour daily at 250 mg/m³ showed extreme irritation. Symptoms include loss of appetite, weight loss, diarrhoea, muscular incoordination and loss of hair. Of the 51 animals exposed, 26 died after the tenth exposure. Exposure to freshly generated MoO₂ fume under about the same exposure conditions proved unexpectedly less toxic, with only 8.3% mortality compared with 51% mortality with the dust, and no mortality when the exposure level was reduced to about one-third (57 mg Mo/m³). Explanation for this unexpected finding was felt to reside in the more rapid solution and elimination of the large surface area fume particle. [Patty's]

Exposure of male and female rats to molybdenum trioxide resulted in the development of respiratory system lesions. In the lung, the incidence and severity of chronic alveolar inflammation increased with increasing exposure concentration in male and female rats. In some male rats, exposure to the material resulted in alveolar/ bronchiolar adenomas or carcinomas. Lesions in the nose (hyaline degeneration) and larynx (squamous metaplasia) were considered to be a non-specific defensive or adaptive response to chronic inhalation exposure. Inhalation exposure of mice to molybdenum trioxide was associated with the development of lung neoplasms and an increased incidence of alveolar/ bronchiolar carcinoma or adenoma in both sexes. Chronic inflammatory lesions were not present in the lungs. Lesions of the nose and larynx were similar to those observed in rats.

Molybdenum trioxide was not mutagenic in any of five strains of *Salmonella typhimurium* and did not induce sister chromatid exchanges or chromosomal aberrations in cultured Chinese hamster ovary cells in vitro. All tests were conducted with or without S9 metabolic activation enzymes.

Pneumoconiosis has been described in experimental animals exposed sub-chronically to molybdenum trioxide.

The mechanism of molybdenum trioxide action in lung carcinogenicity is not known; the material is not mutagenic.

Non-neoplastic lesions of the nose and larynx of rats and in the nose, larynx and lungs of mice were apparently due to the development of a more durable epithelium in response to chronic exposure.

The US Department of Health and Human Services (1) concluded that there was equivocal evidence of carcinogenic activity in male F344/N rats based on a marginally significant positive trend of alveolar/ bronchiolar adenoma or carcinoma; that there was no evidence of carcinogenic activity in female F344/N rats; that there was some evidence of carcinogenic activity in male B6C3F1 mice and that there was evidence of carcinogenic activity in female B6C3F1 mice

National Toxicology Program: Technical Report Series 462, April 1997

Chronic intoxication with ionic bromides, historically, has resulted from medical use of bromides but not from environmental or occupational exposure; depression, hallucinosis, and schizophreniform psychosis can be seen in the absence of other signs of intoxication. Bromides may also induce sedation, irritability, agitation, delirium, memory loss, confusion, disorientation, forgetfulness (aphasias), dysarthria, weakness, fatigue, vertigo, stupor, coma, decreased appetite, nausea and vomiting, diarrhoea, hallucinations, an acne like rash on the face, legs and trunk, known as bronchoderma (seen in 25-30% of case involving bromide ion), and a profuse discharge from the nostrils (coryza). Ataxia and generalised hyperreflexia have also been observed. Correlation of neurologic symptoms with blood levels of bromide is inexact. The use of substances such as brompheniramine, as antihistamines, largely reflect current day usage of bromides; ionic bromides have been largely withdrawn from therapeutic use due to their toxicity.

In test animals, brominated vegetable oils (BVOs), historically used as emulsifiers in certain soda-based soft drinks, produced damage to the heart and kidneys in addition to increasing fat deposits in these organs. In extreme cases BVO caused testicular damage, stunted growth and produced lethargy and fatigue.

Brominism produces slurred speech, apathy, headache, decreased memory, anorexia and drowsiness, psychosis resembling paranoid schizophrenia, and personality changes

Several cases of foetal abnormalities have been described in mothers who took large doses of bromides during pregnancy.

Reproductive effects caused by bromide (which crosses the placenta) include central nervous system depression, brominism, and bronchoderma in the newborn.

Chronic excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas. Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.

Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.

High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.

Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up.

[K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]

Azo dyes as a class are a concern for their potential induction of mutagenicity and carcinogenicity

Reductive cleavage or degradation into component aromatic amines is one of the mechanisms leading to the genotoxicity of azo dyes. The aromatic amines that arise from the azo reduction and cleavage of azo dyes are thought to be activated as mutagens through their *N*-oxidation by cytochrome P450 isozymes. The *N*-hydroxylarylamines that are formed may be further glucuronated (activated) or acetylated (inactivated), which may influence their mutagenicity. Under acidic pH, they form reactive nitrenium ions that can alkylate bases in DNA, particularly the nucleophilic centres in guanine. This mechanism is thought to contribute to the carcinogenicity of many azo dyes, and as a result, azo dyes should be assessed for toxicity and classified similarly to their component amines.

Many azo dyes (aromatic amines) have been found to be carcinogenic in laboratory animals, affecting the liver, urinary bladder and intestines. Specific toxicity effects in humans have not been established but some dyes are known to be mutagenic.

Benzidine and its metabolic derivatives have been detected in the urine of workers exposed to Direct azo dyes. An epidemiological study of silk dyers and painters with multiple exposures to benzidine based and other dyes indicate a strong association with bladder cancer.

Not all azo dyes are genotoxic, only those dyes that contain either phenylenediamine or benzidine in the molecule would become mutagenic. Therefore, phenylenediamine and benzidine are the major mutagenic moieties of carcinogenic azo dyes.

Many functional groups (i.e. NO₂, CH₃ and NH₂) within the molecules of these amines affected their genotoxicities. Many aromatic amines are carcinogenic and/or mutagenic. This appears to involve bioactivation by various organs and/ or bacterial

intervention

The simplest azo dyes, which raise concern, have an exocyclic amino-group that is the key to any carcinogenicity for it is this group which undergoes biochemical N-oxidation and further reaction to reactive electrophiles. The DNA adducts formed by covalent binding through activated nitrogen have been identified. However not all azo compounds possess this activity and delicate alterations to structure vary the potential of carcinogenicity / acid, reduces or eliminates the effect. Complex azo dyes consisting of more than one azo (N=N) linkage may be metabolised to produce complexed carcinogenic aromatic amines such as benzidine.

The carcinogenic aromatic amines are generally recognized to be bioactivated in two steps: N-hydroxylation catalyzed by cytochrome P450 enzymes to give N-hydroxyarylamines and subsequent acetyl-CoA-dependent o-acetylation. The N-acetoxy esters formed by acetylation of hydroxylamines are reactive electrophiles which give rise to covalent DNA-adduct probably via the loss of an active anion, which yields a nitrenium ion.

In the past, azo colorants based on benzidine, 3,3'-dichlorobenzidine, 3,3'-dimethylbenzidine (o-tolidine), and 3,3'-dimethoxybenzidine (o-dianisidine) have been synthesized in large amounts and numbers. Studies in exposed workers have demonstrated that the azoreduction of benzidine-based dyes occurs in man. The metabolic conversion of benzidine-, 3,3'-dimethylbenzidine- and 3,3'-dimethoxybenzidine-based dyes to their (carcinogenic) amine precursors in vivo is a general phenomenon that must be considered for each member of this class of chemicals.

Azo dyes containing phenylenediamine are mutagenic in certain assays most likely due to the formation of oxidized p-phenylenediamine. p-Phenylenediamine are oxidised by the liver microsomal enzymes (S9). Pure p-phenylenediamine is non-mutagenic but becomes mutagenic after it is oxidized. Modification of the moieties that can be metabolized to p-phenylenediamine by sulfonation, carboxylation or copper complexation eliminated the mutagenic responses.

Bioavailability of azo dyes also determines whether they are to be metabolically converted to carcinogens. As a majority of azo pigments are based on 3,3'-dichlorobenzidine, much of the available experimental data are focused on this group. Long-term animal carcinogenicity studies performed with pigments based on 3,3'-dichlorobenzidine did not show a carcinogenic effect. Hence, it is very unlikely that occupational exposure to insoluble azo pigments would be associated with a substantial risk of (bladder) cancer in man. According to current EU regulations, azo dyes based on benzidine, 3,3'-dimethoxybenzidine and 3,3'-dimethylbenzidine have been classified as carcinogens of category 2 as "substances which should be regarded as if they are carcinogenic to man" This is not the case for 3,3'-dichlorobenzidine-based azo pigments.

It is also postulated that some of the aromatic amines metabolically produced from azo dyes may be responsible for the induction of autoimmune diseases such as lupus. This is probably due to the fact that lupus inducing drugs are amines in nature. They also have the similar metabolic activation pathways as the human bladder procarcinogens. The only difference between lupus inducing drugs and procarcinogens is that carcinogens interact with DNA to form covalent adducts which produce mutations, while lupus inducing drugs interact with DNA to provoke the immunoresponses.

Azo dyes are widely used in industry. A large amount of these dyes are discharged into streams and rivers, and they are considered as an environmental pollutant. Some of these compounds may accumulate into food chains and eventually reach the human body through ingestion. Intestinal microbiota and to a lesser extent, the liver enzymes, are responsible for the cleavage of azo dyes into aromatic amines. Some of human endogenous bacteria that contaminate bladder can metabolically activate aromatic amines that are produced from azo dyes (procarcinogens). The addition of the nitro-group to these aromatic amines would convert them into direct mutagens.

These findings may also explain, partly, the close relationships between chronic infection and cancer development.

Skin bacteria are thought to be responsible for cleavage of certain azo dyes to produce carcinogens; of importance are dye-stuffs found in cosmetics, hair dyes, textiles and tattoo inks.

Several *in vitro* and *in vivo* studies suggest that certain azo dyes may be reductively cleaved when applied to the skin also under aerobic conditions. Results obtained with the various azo dyes suggest that reductive cleavage to aromatic amines has to be considered a significant degradation pathway. It is generally thought that about 30% of the dye may be cleaved in this manner.

From the available literature, on this chemical class of azo dyes, it can be deduced that all azo dyes which are split into carcinogenic arylamines are possible carcinogens.

Both water-soluble and lipophilic azo dyes of this class have been shown experimentally to undergo cleavage to potential carcinogens.

Workers exposed to barium compounds have been reported to show an increased incidence of hypertension, irritation of the respiratory system, and damage to the spleen, liver and bone marrow. Long term exposure to some barium compounds (especially inorganic species) may produce a condition known as baritosis, a form of benign pneumoconiosis. X-ray may show this when no other abnormal signs are present.

Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. Barium sulfate produces noncollagenous pneumoconiosis identified by minimal stromal reaction, consisting mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible. Miners of ores containing barium sulfate do not show symptoms, abnormal physical signs, an incapacity to work, diminished lung function, an increased likelihood of developing pulmonary or other bronchial infections or other thoracic disease despite the fact that particulate matter may have been retained in the lungs for many years.

No changes in mortality were observed in rats chronically exposed to doses as high as 60 mg barium/kg/day as barium chloride in the drinking water. An increase in mortality, attributable to nephropathy, was observed in mice chronically exposed to 160 mg barium/kg/day as barium chloride in drinking water; the number of deaths was similar to controls in mice exposed to 75 mg barium/kg/day. In male mice exposed to 0.95 mg barium/kg/day as barium acetate in drinking water, a significant decrease in longevity (defined as average lifespan of the last five surviving animals) was observed; however, no significant differences in mean lifespan were observed. Similarly, lifespan was not significantly altered in female mice exposed to 0.95 mg barium/kg/day or male or female rats exposed to 0.7 mg barium/kg/day as barium acetate in drinking water.

The potential for barium to induce reproductive and developmental effects has not been well investigated. Decreases in the number of sperm and sperm quality and a shortened estrous cycle and morphological alterations in the ovaries were observed in rats exposed to 2.2 mg barium/m³ and higher in air for an intermediate duration. Interpretation of these data is limited by the poor

Reeves Gouache 22ml Paint

	<p>reporting of the study design and results, in particular, whether the incidence was significantly different from controls. In general, oral exposure studies have not found morphological alterations in reproductive tissues of rats or mice exposed to 180 or 450 mg barium/kg/day, respectively, as barium chloride in drinking water for an intermediate duration. Additionally, no significant alterations in reproductive performance was observed in rats or mice exposed to 200 mg barium/kg/day as barium chloride in drinking water. Decreased pup birth weight and a nonsignificant decrease in litter size have been observed in the offspring of rats exposed to 180/200 mg barium/kg/day as barium chloride in drinking water prior to mating. Several studies have examined the carcinogenic potential of barium following oral exposure and did not find significant increases in the tumour incidence. On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p>	
Reeves Gouache 22ml Paint	TOXICITY	IRRITATION
	Not Available	Not Available
dextrins	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^[2]	Not Available
silica amorphous	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye (rabbit): non-irritating *
	Inhalation (rat) LC50: >0.139 mg/l/14h**[Grace] ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: 3160 mg/kg ^[2]	Skin (rabbit): non-irritating * Skin: no adverse effect observed (not irritating) ^[1]
barium sulfate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (mouse) LD50: >3000 mg/kg ^[2]	Not Available
titanium dioxide	TOXICITY	IRRITATION
	dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: >2000 mg/kg ^[1]	Skin (human): 0.3 mg /3D (int)-mild * Skin: no adverse effect observed (not irritating) ^[1]
C.I. Pigment Yellow 3	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (rat) LD50: >2000 mg/kg ^[1]	Not Available
C.I. Pigment Yellow 1	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (rat) LD50: >2000 mg/kg ^[1]	Non-irritating/non-sensitising
ferric hydroxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available
ferric oxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available
C.I. Pigment Green 7	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^[1]	Not Available
C.I. Pigment Blue 15	TOXICITY	IRRITATION
	Oral (rat) LD50: >10,000 mg/kg ^[2]	Eye (human): non-irritant Skin (human): non-irritant
C.I. Pigment Blue 1	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg ^[2]	Not Available
carbon black	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]

Reeves Gouache 22ml Paint

	Oral (rat) LD50: >15400 mg/kg ^[2]	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]
		Skin: no adverse effect observed (not irritating) ^[1]
C.I. Pigment Red 21	TOXICITY	IRRITATION
	Not Available	Not Available
C.I. Pigment Blue 27	TOXICITY	IRRITATION
	Oral (rat) LD50: 15000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Eye: non-irritating *
		Skin: no adverse effect observed (not irritating) ^[1]
		Skin: non-irritating *
glycerol	TOXICITY	IRRITATION
	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available
2-bromo-2-nitropropan-1,3-diol	TOXICITY	IRRITATION
	dermal (rat) LD50: 64 mg/kg ^[2]	Eye (rabbit): 5 mg
	Inhalation (rat) LC50: 0.8 mg/l/4h ^[2]	Skin (human): 10 mg moderate
	Oral (rat) LD50: 180 mg/kg ^[2]	Skin (rabbit): 500 mg/24h mild
		Skin (rabbit): 80 mg moderate
zinc pyrithione	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 1 mg/48h Irritant
	Inhalation (rat) LC50: 0.14 mg/l/4h ^[2]	
	Oral (rat) LD50: 177 mg/kg ^[2]	
zinc oxide	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit) : 500 mg/24 h - mild
	Inhalation (rat) LC50: >1.79 mg/l4 h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: >5000 mg/kg ^[2]	Skin (rabbit) : 500 mg/24 h- mild
		Skin: no adverse effect observed (not irritating) ^[1]
water	TOXICITY	IRRITATION
	Oral (rat) LD50: >90000 mg/kg ^[2]	Not Available
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

SILICA AMORPHOUS	<p>Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]</p> <p>For silica amorphous: Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d.</p> <p>In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.</p> <p>When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.</p> <p>After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.</p> <p>Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the</p>
-------------------------	--

required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.

Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure.

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

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

For Synthetic Amorphous Silica (SAS)

Repeated dose toxicity

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

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

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

For silane treated synthetic amorphous silica:

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

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

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.

* IUCLID

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

This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.

For titanium dioxide:

Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.

Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.

No data were available on genotoxic effects in titanium dioxide-exposed humans.

Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain.

Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols.

Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.

Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.

Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the 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

TITANIUM DIOXIDE

	<p>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.</p> <p>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.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
C.I. PIGMENT YELLOW 3	<p>▶ NOTE: Detailed analysis of the molecular structure, by various Authorities/ Agencies and in other cases by Chemwatch, indicates that the azo colourant can split off carcinogenic arylamines.</p> <p>The azo linkage is considered the most labile portion of an azo dye. The linkage easily undergoes enzymatic breakdown, but thermal or photochemical breakdown may also take place. The breakdown results in cleavage of the molecule and in release of the component amines. Water solubility determines the ultimate degradation pathways of the dyes. For example the azo linkage of many azo pigments is, due to very low solubility in water, not available for intracellular enzymatic breakdown but may be susceptible to endogenous micro-organisms found in the bladder or in the gut.</p> <p>After cleavage of the azo linkage by bacteria, the component aromatic amines are absorbed in the intestine and excreted in the urine. Twenty-two of the component amines are recognised as potential human carcinogens, and/or several of them have shown carcinogenic potential on experimental animals. Sulfonation of the dye reduces the toxicity by enhancement of the excretion. The component amines which may be released from azo dyes are mostly aromatic amines (compounds where an amine group or amine-generating group(s) are connected to an aryl moiety). In general, aromatic amines known as carcinogenic may be grouped into five groups</p> <ul style="list-style-type: none"> ▶ Anilines, e.g. o-toluidine. ▶ Extended anilines, e.g. benzidine. ▶ Fused ring amines, e.g. 2-naphthylamine. ▶ Aminoazo and other azo compounds, e.g. 4-(phenylazo)aniline. ▶ Heterocyclic amines. <p>The aromatic amines containing moieties of anilines, extended anilines and fused ring amines are components of the majority of the industrially important azo dyes.</p> <p>Reductive fission of the azo group, either by intestinal bacteria or by azo reductases of the liver and extra-hepatic tissues can cause benzidine-based aromatic amines to be released. Such breakdown products have been detected in animal experiments as well as in man (urine). Mutagenicity, which has been observed with numerous azo colourants in in vitro test systems, and the carcinogenicity in animal experiments are attributed to the release of amines and their subsequent metabolic activation. There are now epidemiological indications that occupational exposure to benzidine-based azo colourants can increase the incidence of bladder carcinoma.</p> <p>The acute toxicity of azo dyes is low.. However, potential health effects are recognised.</p> <p>Despite a very broad field of application and exposure, sensitising properties of azo dyes have been identified in relatively few reports. Red azoic dyes have been linked to allergic contact dermatitis in heavily exposed workers. Furthermore, textiles coloured with disperse azo dyes have caused allergic dermatitis in a few cases.</p>
CARBON BLACK	Inhalation (rat) TCl _o : 50 mg/m ³ /6h/90D-I Nil reported
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.
C.I. PIGMENT BLUE 27	No evidence of absorption or adverse health effects based on 90-day feeding studies in rats and mice * * Hang Zhou Pigments MSDS
GLYCEROL	<p>For glycerol:</p> <p>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.</p> <p>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.</p> <p>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 <i>in vitro</i>. Results of a limited gene mutation test in mammalian cells were of uncertain biological relevance. <i>In vivo</i>, 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 <i>in vivo</i> data. Overall, glycerol is not considered to possess genotoxic potential.</p> <p>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.</p> <p>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).</p>

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

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.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic).

This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Chemical with the aliphatic nitro group (-C-NO₂) have been added to a list of DNA-reactive subgroups recognised by the National Toxicological Program (NTP, U.S. Dept Health and Human Services) for possible carcinogenic activity.

ZINC PYRITHIONE

for pyrithiones:

Short-term studies: Zinc pyrithione was orally administered to cynomolgus monkeys daily for 14 or 28 days. In the 14-day study, treatment at 10, 20, 40 or 80 mg/kg bw/day resulted in haemorrhaging of the stomach mucosa and bodyweight loss at the highest tested dose. In the 28-day study, treatment at 0, 5.5, 11 or 22 mg/kg bw, caused a death at the highest dose. Food consumption and bodyweight gain was decreased at the highest dose together with reduced haematocrit, haemoglobin concentration and erythrocyte count. An increased concentration of ketone bodies and decreased pH of the urine was also observed. These changes were either absent or had improved after a 14-day recovery period.

In a 90-day study, rats were fed zinc pyrithione in the diet at concentrations of 0, 5, 25 or 125 ppm. Clinical signs first observed during the second week at 125 ppm were a depressed respiratory rate and the onset of progressively restricted movement of the hind limbs which finally resulted in almost complete paralysis. Other changes at 125 ppm were related to severe weight loss and dehydration, resulting from the paralysis. Based on the deaths of nearly all the rats at 125 ppm (from dehydration and/or starvation) and the reduced bodyweight observed at 25 ppm in females, the NOEL for this study was 5 ppm (0.35 mg/kg bw/day for males and 0.39 mg/kg bw/day for females).

Daily dermal application of zinc pyrithione to rats at 0, 20, 100 or 1000 mg/kg bw/day for 90 days revealed slight skin irritation, bodyweight loss and reduced food intake at 1000 mg/kg bw/day. For females at 1000 mg/kg bw/day there was an increase in relative kidney weight and some had mineralisation of the kidneys. Increased leucocyte counts and reduced erythrocyte and haematocrit was also observed at the highest dose. Dermal absorption studies in pigs showed that zinc pyrithione is very poorly absorbed through skin (<10% of dose). A maximum of 5% of the applied dose was recovered in the urine and by 48 h the levels in blood, faeces, and urine were essentially at background levels.

Whole-body exposure to an aerosol at 0, 0.5, 2.5 or 10 mg/m³ for 6 h/day, 5 days/week over 13 weeks resulted in deaths at 2.5 and 10 mg/m³, reduced bodyweight gain at 10 mg/m³ and reduced creatinine at 10 mg/m³. A dose-related increase in mean absolute lung/mainstream bronchi weight, lung/mainstream bronchi weight relative to body weight and lung/mainstream bronchi weight relative to brain weight was also observed at 2.5 and 10 mg/m³. These weight increases were accompanied by inflammation of interstitial tissue and pulmonary artery hypertrophy.

Zinc pyrithione given to monkeys at 0, 0.5, 2 or 8 mg/kg bw/day by stomach tube for 90 days induced some vomiting at 2 and 8 mg/kg bw/day within 1-3 h on the first few treatment days. Appropriate monitoring for adverse changes failed to reveal any other effects. Hence, the NOEL for the study was 8 mg/kg bw/day.

Long-Term Study: Sodium pyrithione at 0, 0.5, 1.5 or 5 mg/kg bw/day was administered to rats by gavage in a two-year chronic and oncogenicity study. After 12 weeks at 5 mg/kg bw/day, an appreciable reduction in bodyweight gain necessitated the high dose level be reduced to 3.5 mg/kg bw/day. There was reduced bodyweight gain at 3.5 mg/kg bw/day and hind limb muscle wastage at 1.5 and 3.5 mg/kg bw/day. Nerve fibre degeneration of the spinal cord and sciatic nerve was slightly increased at 3.5 mg/kg bw/day. Fibre degeneration in the hind limb skeletal muscle was increased in all rats at 3.5 mg/kg bw/day and to a lesser extent in females at 1.5 mg/kg bw/day. There was an increase in peripheral retinal atrophy in males and females at 3.5 mg/kg bw/day and at 1.5 mg/kg bw/day in females. There was no treatment-related increase in the incidence of tumours. Therefore, under the conditions of this study, the NOEL was 0.5 mg/kg bw/day.

Reproduction and Developmental Studies: In a 2-generation reproduction study, rats were given sodium pyrithione at 0, 0.5, 1.5 or 4.5 mg/kg bw/day by gavage. Owing to an appreciable reduction in bodyweight gain the highest dose was reduced after 3 weeks to 3.5 mg/kg bw/day for the rest of the study. Rats were maintained for 2 generations, with the first litter used for breeding. In the F₀ rats, salivation after dosing was seen in all treated groups, with a dose-related time of onset and severity. At 3.5 mg/kg bw/day a number of females showed hind-limb paralysis in the F₀ generation; this was not seen in F₁ animals. Body weight gain was statistically significantly decreased in both males and females at 3.5 mg/kg bw/day in the F₀ generation, and in females at this dose in the F₁ generation. Fertility was decreased at 3.5 mg/kg bw/day in the F₀ generation, with the number of rats

	<p>successfully mating and the number of rats pregnant decreased in comparison to controls. There was no effect on gestational length, the number of pups born or pup bodyweight seen. No effects on fertility were seen in the F1 generation. There was no increase in the incidence of foetal malformations in either generation. On postmortem examination, there was an increase in the incidence of hind-limb muscle atrophy at 3.5 mg/kg bw/day in females in both generations. On histopathological examination, there was an increase in atrophy of skeletal muscles at 3.5 mg/kg bw/day in the F0 generation, and from 1.5 mg/kg bw/day in the F1 generation. Salivation occurred in some F0 rats at 0.5 mg/kg bw/day but none in the F1 generation suggesting that this dose level is a probable NOEL.</p> <p>When pregnant rats had zinc pyrithione topically applied at 0, 2.5, 7.5 or 15 mg/kg bw/day (with or without prevention from ingestion) from gestation days 6 to 15 there was a reduction in bodyweight gain at 7.5 or 15 mg/kg bw/day when ingestion was not prevented. Hind-limb paralysis among dams and reductions in fetal weight were also observed at 15 mg/kg bw/day. These effects were not seen when ingestion was prevented. With oral treatment at the same doses, bodyweight gain was reduced, paralysis occurred and fetal weight was reduced at 7.5 and 15 mg/kg bw/day. There was also an increase in skeletal variations at 15 mg/kg bw/day.</p> <p>Genotoxicity: Zinc pyrithione was found to be negative in mutation tests in bacteria and Chinese hamster ovary cells. Similarly, no chromosomal aberration was observed in human lymphocytes incubated <i>in vitro</i> in the presence of zinc pyrithione or in lymphocytes harvested from monkeys following oral administration in a 28-day toxicity study. A mouse micronucleus assay also yielded negative results.</p> <p>Human metabolite study</p> <p>A study of plasma metabolites in human volunteers from a chemical factory producing pyrithiones identified 2-(methylsulfonyl)pyridine as the only metabolite in human serum and proposed that this metabolite could be used as a marker for pyrithione exposure.</p> <p>Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis). NOAEL: 11.0 mg/kg/day cynomolgus monkey * [* = Arch Chemical] Acute pulmonary oedema, dyspnea, weight loss or decreased weight gain, recordings from specific areas of the CNS, mydriasis, somnolence, changes in motor activity, recording from peripheral motor nerve, muscle weakness, spastic paralysis, reproductive system tumours, retinal changes, diarrhoea, foetotoxicity, specific developmental abnormalities (musculoskeletal system, central nervous system, effects on newborn, foetolethality recorded.</p>
<p>DEXTRINS & BARIUM SULFATE & TITANIUM DIOXIDE & FERRIC HYDROXIDE & C.I. PIGMENT GREEN 7 & CARBON BLACK & C.I. PIGMENT RED 4 & C.I. PIGMENT RED 21 & WATER</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>TITANIUM DIOXIDE & FERRIC OXIDE & GLYCEROL & 2-BROMO-2-NITROPROPAN-1,3-DIOL</p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic 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.</p>
<p>TITANIUM DIOXIDE & ZINC OXIDE</p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<p>TITANIUM DIOXIDE & CARBON BLACK</p>	<p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>

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

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

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Continued...

Reeves Gouache 22ml Paint

Reeves Gouache 22ml Paint	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
dextrins	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
silica amorphous	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-289.09mg/L	2
	EC50	48	Crustacea	ca.7600mg/L	1
	EC50	72	Algae or other aquatic plants	440mg/L	1
	NOEC	720	Crustacea	34.223mg/L	2
barium sulfate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>3.5mg/L	2
	EC50	48	Crustacea	0.032-mg/L	2
	EC50	72	Algae or other aquatic plants	>1.15mg/L	2
	NOEC	2016	Algae or other aquatic plants	0.004-mg/L	2
titanium dioxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>1-mg/L	2
	EC50	48	Crustacea	>1-mg/L	2
	EC50	72	Algae or other aquatic plants	5.83mg/L	4
	NOEC	336	Fish	0.089mg/L	4
C.I. Pigment Yellow 3	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>1mg/L	2
	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
C.I. Pigment Yellow 1	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>1mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	96	Algae or other aquatic plants	3.244mg/L	3
	NOEC	72	Algae or other aquatic plants	1mg/L	2
ferric hydroxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	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	
ferric oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	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

Reeves Gouache 22ml Paint

	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
C.I. Pigment Green 7	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
C.I. Pigment Blue 15	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
C.I. Pigment Blue 1	Not Available	Not Available	Not Available	Not Available	Not Available
carbon black	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	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
C.I. Pigment Red 4	EC50	48	Crustacea	>100mg/L	2
	NOEC	72	Algae or other aquatic plants	>0.006mg/L	2
C.I. Pigment Red 21	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Pigment Blue 27	LC50	96	Fish	41200000mg/L	3
	EC50	96	Algae or other aquatic plants	2330000000mg/L	3
glycerol	LC50	96	Fish	>0.011-mg/L	2
	EC50	96	Algae or other aquatic plants	77712.039mg/L	3
2-bromo-2-nitropropan-1,3-diol	LC50	96	Fish	20mg/L	4
	EC50	48	Crustacea	0.78mg/L	4
	EC50	72	Algae or other aquatic plants	0.25mg/L	2
	NOEC	72	Algae or other aquatic plants	0.08mg/L	2
zinc pyrithione	LC50	96	Fish	0.0026mg/L	2
	EC50	48	Crustacea	0.0082mg/L	2
	EC50	72	Algae or other aquatic plants	0.0005124951mg/L	4
	NOEC	120	Algae or other aquatic plants	0.00046mg/L	2
zinc oxide	LC50	96	Fish	0.001-0.58mg/L	2
	EC50	48	Crustacea	0.001-0.014mg/L	2
	EC50	72	Algae or other aquatic plants	0.037mg/L	2
	BCF	336	Fish	4376.673mg/L	4

Continued...

Reeves Gouache 22ml Paint

	NOEC	72	Algae or other aquatic plants	0.00008138mg/L	2
water	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	897.520mg/L	3
	EC50	96	Algae or other aquatic plants	8768.874mg/L	3
Legend:	<i>Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 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</i>				

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

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
silica amorphous	LOW	LOW
titanium dioxide	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 Blue 27	HIGH	HIGH
glycerol	LOW	LOW
2-bromo-2-nitropropan-1,3-diol	LOW	LOW
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
silica amorphous	LOW (LogKOW = 0.5294)
titanium dioxide	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 Blue 27	LOW (LogKOW = -6.3018)
glycerol	LOW (LogKOW = -1.76)
2-bromo-2-nitropropan-1,3-diol	LOW (LogKOW = -0.6408)
zinc pyrithione	LOW (BCF = 240)
zinc oxide	LOW (BCF = 217)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
silica amorphous	LOW (KOC = 23.74)
titanium dioxide	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 Blue 27	LOW (KOC = 1197)
glycerol	HIGH (KOC = 1)

Reeves Gouache 22ml Paint

2-bromo-2-nitropropan-1,3-diol	HIGH (KOC = 1)
water	LOW (KOC = 14.3)



SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
------------------------------	--

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains C.I. Pigment Red 4, C.I. Pigment Blue 1 and C.I. Pigment Blue 27)
Transport hazard class(es)	Class : 9 Subrisk : Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions : 274 331 335 375 AU01 Limited quantity : 5 L

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

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

Air transport (ICAO-IATA / DGR)

UN number	3082
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains C.I. Pigment Red 4, C.I. Pigment Blue 1 and C.I. Pigment Blue 27)

Reeves Gouache 22ml Paint

Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	9L
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	A97 A158 A197
	Cargo Only Packing Instructions	964
	Cargo Only Maximum Qty / Pack	450 L
	Passenger and Cargo Packing Instructions	964
	Passenger and Cargo Maximum Qty / Pack	450 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y964
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3082	
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains C.I. Pigment Red 4, C.I. Pigment Blue 1 and C.I. Pigment Blue 27)	
Transport hazard class(es)	IMDG Class	9
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number	F-A , S-F
	Special provisions	274 335 969
	Limited Quantities	5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

DEXTRINS IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

SILICA AMORPHOUS 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 10 / Appendix C

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

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

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

BARIUM SULFATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

TITANIUM DIOXIDE 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

Continued...

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

FERRIC OXIDE 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

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 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 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

CARBON BLACK 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 21 IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

C.I. PIGMENT BLUE 27 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

GLYCEROL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

2-BROMO-2-NITROPROPAN-1,3-DIOL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

ZINC PYRITHIONE 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 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

ZINC OXIDE 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 4

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

WATER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory Status

National Inventory	Status
Australia - AICS	No (C.I. Pigment Red 21)
Canada - DSL	No (C.I. Pigment Red 21)
Canada - NDSL	No (dextrins; barium sulfate; C.I. Pigment Yellow 3; C.I. Pigment Yellow 1; ferric oxide; C.I. Pigment Green 7; C.I. Pigment Blue 15; C.I. Pigment Blue 1; carbon black; C.I. Pigment Red 4; C.I. Pigment Blue 27; glycerol; 2-bromo-2-nitropropan-1,3-diol; zinc pyrrithione; water)
China - IECSC	No (C.I. Pigment Red 21)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (C.I. Pigment Blue 27)
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	Yes
Taiwan - TCSI	No (C.I. Pigment Red 21)
Mexico - INSQ	No (C.I. Pigment Yellow 3; C.I. Pigment Green 7; C.I. Pigment Blue 1; C.I. Pigment Red 21)
Vietnam - NCI	Yes
Russia - ARIPS	No (C.I. Pigment Blue 1; C.I. Pigment Red 21; zinc pyrrithione)
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	05/07/2020

SDS Version Summary

Version	Issue Date	Sections Updated
2.1.1.1	05/07/2020	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Appearance, Chronic Health, Classification, Disposal, Engineering Control, Environmental, Exposure Standard, 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), Physical Properties, Spills (major), Spills (minor), Storage (storage incompatibility), Storage (storage requirement), Storage (suitable container), Transport, Transport Information

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

This document is copyright.

Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH.

TEL (+61 3) 9572 4700.