

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium

Geveko Markings

Chemwatch: 5682-00

Version No: 5.1

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

Chemwatch Hazard Alert Code: 3

Issue Date: 19/09/2024

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L.GHS.AUS.EN.E

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

Product Identifier

Product name	Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium, Premium Plus
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Waterborne pavement marking paint for use on bituminous and concrete substrates. Use according to manufacturer's directions.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Geveko Markings
Address	69-77 Williamson Road Ingleburn NSW 2565 Australia
Telephone	+61 2 9998 3000
Fax	Not Available
Website	www.geveko-markings.com
Email	enquiry@gevekomarkings.com.au

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	+61 1800 951 288
Other emergency telephone number(s)	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Poisons Schedule	Not Applicable
Classification ^[1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Hazardous to the Aquatic Environment Acute 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

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium

Hazard pictogram(s)



Signal word

Danger

Hazard statement(s)

H315	Causes skin irritation.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H401	Toxic to aquatic life.

Precautionary statement(s) Prevention

P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1317-65-3	<65	<u>calcium carbonate</u>
13463-67-7	<30	<u>C.I. Pigment White 6</u>
1309-38-2	<10	<u>magnetite</u>
64-17-5	<5	<u>ethanol</u>
1317-61-9	<5	<u>C.I. Pigment Black 11</u>
1309-37-1	<5	<u>C.I. Pigment Red 101</u>
1336-21-6	<5	<u>ammonium hydroxide</u>
14059-33-7	<5	<u>C.I. Pigment Yellow 184</u>
57455-37-5	<5	<u>C.I. Pigment Blue 29</u>
2786-76-7	<5	<u>C.I. Pigment Red 170</u>
Not Available	balance	Ingredients determined not to be hazardous

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

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SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	For thermal burns: ▶ Decontaminate area around burn. ▶ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▶ Use compresses if running water is not available. ▶ Cover with sterile non-adhesive bandage or clean cloth. ▶ Do NOT apply butter or ointments; this may cause infection. ▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▶ Cool the burn by immerse in cold running water for 10-15 minutes. ▶ Use compresses if running water is not available. ▶ Do NOT apply ice as this may lower body temperature and cause further damage. ▶ Do NOT break blisters or apply butter or ointments; this may cause infection. ▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▶ Lay the person flat. ▶ Elevate feet about 12 inches. ▶ Elevate burn area above heart level, if possible. ▶ Cover the person with coat or blanket. ▶ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▶ Separate burned toes and fingers with dry, sterile dressings. ▶ Do not soak burn in water or apply ointments or butter; this may cause infection. ▶ To prevent shock see above. ▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▶ Have a person with a facial burn sit up. ▶ Check pulse and breathing to monitor for shock until emergency help arrives. ▶ Gently brush or vacuum off adherent fibres. ▶ Wash affected areas thoroughly with water (and soap if available). ▶ Seek medical attention if irritation exists and persists.
Inhalation	▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
for simple esters:

BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ Monitor and treat, where necessary, for pulmonary oedema .
- ▶ Monitor and treat, where necessary, for shock.
- ▶ **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

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- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

Severe bismuth intoxication may be treated with dimercaptol (BAL in oil). Induction of acidosis by administration of ammonium chloride has been claimed to promote mobilisation of bismuth from tissue depots and increase the rate of urinary excretion.

[Martindale: The Extra Pharmacopoeia]

In mouse models D-penicillamine (Cuprimine M.S. & D) is a useful chelating agent. [Ellenhorn & Barceloux: Medical Toxicology]

Mineral fibres are a mechanical irritant, and are not expected to produce any chronic health effects from acute exposures.

Treatment should be directed toward removing the source of irritation with symptomatic treatment as necessary.

Lung function should be monitored, periodically, in individuals chronically exposed to fibres in an occupational setting

for irritant gas exposures:

- the presence of the agent when it is inhaled is evanescent (of short duration) and therefore, cannot be washed away or otherwise removed
- arterial blood gases are of primary importance to aid in determination of the extent of damage. Never discharge a patient significantly exposed to an irritant gas without obtaining an arterial blood sample.
- supportive measures include suctioning (intubation may be required), volume cycle ventilator support (positive and expiratory pressure (PEEP), steroids and antibiotics, after a culture is taken
- If the eyes are involved, an ophthalmologic consultation is recommended

Occupational Medicine: Third Edition; Zenz, Dickerson, Horvath 1994 Pub: Mosby

For acute or short term repeated exposures to ammonia and its solutions:

- Mild to moderate inhalation exposures produce headache, cough, bronchospasm, nausea, vomiting, pharyngeal and retrosternal pain and conjunctivitis. Severe inhalation produces laryngospasm, signs of upper airway obstruction (stridor, hoarseness, difficulty in speaking) and, in excessively, high doses, pulmonary oedema.
- Warm humidified air may soothe bronchial irritation.
- Test all patients with conjunctival irritation for corneal abrasion (fluorescein stain, slit lamp exam)
- Dyspneic patients should receive a chest X-ray and arterial blood gases to detect pulmonary oedema.

SECTION 5 Firefighting measures

Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.

Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.

In such an event consider:

- foam.
- dry chemical powder.
- carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding area. ▸ DO NOT approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ The material is not readily combustible under normal conditions. ▸ However, it will break down under fire conditions and the organic component may burn.

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	▶ Not considered to be a significant fire risk. ▶ Heat may cause expansion or decomposition with violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO₂) nitrogen oxides (NO_x) sulfur oxides (SO_x) silicon dioxide (SiO₂) metal oxides other pyrolysis products typical of burning organic material. CARE: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▶ Clean up all spills immediately. ▶ Avoid all personal contact, including inhalation. ▶ Access to area should be restricted by the use of ropes or other similar barriers and appropriate signs be utilised. ▶ Employees not engaged in the clean up should not be allowed within 3 metres of the work unless wearing suitable personal protective equipment (PPE). ▶ Wear protective clothing, gloves, safety glasses and dust respirator. ▶ Wet with water to prevent dusting. ▶ Avoid generating dust/ fibres. ▶ Vacuum up or sweep up. NOTE: Vacuum cleaner must be fitted with an exhaust micro filter (HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). ▶ Dampen with water to prevent dusting before sweeping ▶ Wet mopping and wiping may be utilised in some instances. ▶ Place in sealed containers, to prevent dust/ fibre emissions, ready for disposal.
Major Spills	▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Control personal contact with the substance, by using protective equipment and dust respirator. ▶ Access to area should be restricted by the use of ropes or other similar barriers and appropriate signs be utilised. ▶ Personnel not engaged in the cleanup should not be allowed in the vicinity of the spillage unless wearing suitable personal protective equipment (PPE). ▶ Prevent spillage from entering drains, sewers or water courses. ▶ Recover product wherever possible. ▶ Avoid generating dust. Sweep / shovel up. ▶ If required, wet with water to prevent dusting. ▶ Put residues in labeled plastic bags or other containers for disposal. ▶ Wash area down with a large quantity of water and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ The use of ceramic fibres in the work place should be reviewed in the context of frequency of use and potential for exposure. ▶ In circumstances where the respiratory standards or excursion limits are approached, work areas should be designated by the use of ropes or other similar barriers and appropriate signs be utilised, where possible. This is especially true for all overhead work involving ceramic fibres. ▶ Employees not engaged in the ceramic fibre work should not be allowed within 3 metres of the work unless wearing suitable personal protective equipment (PPE). ▶ An example of the appropriate signage for the restricted area is: CERAMIC FIBRE WORK AREA; FOLLOW SAFETY INSTRUCTIONS. All installation and/ or removal practices should be designed to minimise the liberation of dusts or fibres. For Installation: ▶ The ceramic fibre material should be kept in its storage container until installation is ready to proceed.
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	▶ Containers/ bags should only be opened within the designated work areas. ▶ Empty storage bags should be stored in waste containers along with waste material. For Removal: ▶ Waste material should be wetted to prevent generation of dusts and placed in sealed containers to prevent dust/ fibre emissions. Upon completion of installation/ removal: ▶ All excess material should be sealed in bags/ containers prior to removal from designated work area. ▶ Area should then be cleaned using an industrial vacuum cleaner. ▶ Any remaining contaminant material should be removed with minimum liberation of dusts/fibres. ▶ Wet mopping and wiping may be utilised in some instances when an industrial vacuum is not available.
Other information	▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▶ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates. ▶ Avoid strong acids, bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	C.I. Pigment White 6	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ethanol	Ethyl alcohol	1000 ppm / 1880 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	C.I. Pigment Red 101	Rouge dust	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	C.I. Pigment Red 101	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	C.I. Pigment Yellow 184	Molybdenum, insoluble compounds (as Mo)	10 mg/m3	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
calcium carbonate	Not Available	Not Available
C.I. Pigment White 6	5,000 mg/m3	Not Available
magnetite	Not Available	Not Available
ethanol	Not Available	Not Available
C.I. Pigment Black 11	Not Available	Not Available
C.I. Pigment Red 101	2,500 mg/m3	Not Available
ammonium hydroxide	Not Available	Not Available
C.I. Pigment Yellow 184	5,000 mg/m3	Not Available
C.I. Pigment Blue 29	Not Available	Not Available
C.I. Pigment Red 170	Not Available	Not Available

Occupational Exposure Banding

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Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
C.I. Pigment Black 11	E	≤ 0.01 mg/m ³
ammonium hydroxide	E	≤ 0.1 ppm
Notes: <i>Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.</i>		

MATERIAL DATA

Exposure controls

Appropriate engineering controls	CARE: Explosive vapour air mixtures may be present on opening vessels which have contained liquid ammonia. Fatalities have occurred ▶ Provide good ventilation (either forced or natural) ▶ Where possible, enclose sources of dust and provide dust extraction at the source. ▶ Restrict access to work areas involved in handling man-made mineral fibres and ensure that adequate training, in the handling of such materials, has been provided. ▶ Use operating procedures which limit the generation of dusts. ▶ When working with unbonded fibres, local exhaust ventilation is generally a requirement. ▶ Exhaust ventilation should be designed to prevent accumulation and recirculation of dusts and to remove dusts from the workplace. ▶ Keep the work place clean. Use a vacuum cleaner fitted with a HEPA filter; avoid using brooms and compressed air. ▶ Where possible use products specially tailored to the application; some products can be delivered, ready for use, without further cutting or machining. Some can be treated or packaged to minimise or avoid dust emission during handling. ▶ When removing embrittled materials, the removal area should be contained to minimise the transfer of dust to other work areas and should include an intermediate changing and cleaning area. Local exhaust ventilation should be provided. ▶ If measured respirable fibre is less than the recommended occupational exposure level, wear approved dust respirator Class P1 (half-face). ▶ Use a Class P2 or P3 respirator (full-face), where exposure is above the recommended occupational exposure level ▶ Use an approved respirator if power tools without dust extraction or containment are used.
Individual protection measures, such as personal protective equipment	
Eye and face protection	▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ 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].
Skin protection	See Hand protection below
Hands/feet protection	▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber For esters: ▶ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min

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	· Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▶ Disposable coveralls or long sleeve, loose fitting protective clothing, e.g. overalls (launder clothing separately from other clothing). ▶ When working above head height, use head covering. ▶ Minimise dust generation by using sharp hand cutting tools if possible. ▶ Powered tools (e.g. saws etc.) should only be used if fitted with dust extraction and containment equipment. ▶ Vacuum cleaners should be available for fibre/dust removal. ▶ Personnel involved in the installation of unbonded ceramic materials should wear disposable coveralls, or long-sleeve loose fitting clothing, gloves and suitable respirator. Such equipment should also be used by personnel employed in removing materials which have not become embrittled. ▶ Personnel involved in the removal of embrittled material should in addition, use a full-face cartridge respirator, or full-face powered air purifying respirator, each with suitable particulate filter, or a full-face pressure demand airline respirator.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
BUTYL	A
NEOPRENE	A
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- ▶ 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
- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
- The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
- Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be

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government mandated or vendor recommended.

- Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
- Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
- Use approved positive flow mask if significant quantities of dust becomes airborne.
- Try to avoid creating dust conditions.

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

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

Filtration rate: Filters at least 99.95% of airborne particles

Suitable for:

- Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
- Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
- Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
- Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos

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

Use appropriate respiratory protective equipment against excessive concentrations of fibrous dusts.

Airborne Fibre Concentration	Full Face P2	Full Face P3
Above Exposure Limit Value	Recommended	-
For short-term operation where excursions above the limit value are less than factor of 10		Required

- ▶ Correct respirator fit is essential to obtain adequate protection.
- ▶ Even though the recommended level for respirable fibre is not exceeded in normal conditions, respiratory protection is advisable in dusty areas.
- ▶ In very dusty conditions and confined spaces greater comfort may be afforded by a full-face powered air-purifying respirator.
- ▶ Preforms (batts) designed for high temperature applications (above 177 degrees Celsius), may release gases (CO2, formaldehyde, amines) irritating to the eyes, nose and throat during initial heat-up. In confined or poorly ventilated areas, use air supplied respirators during the first heat-up cycle.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured liquid paint with acrid odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.02-1.98
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	10-11	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Applicable	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

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Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	- 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	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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either - produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or - produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. 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. Limited evidence suggests that repeated exposure may cause skin cracking, flaking or drying following normal handling and use.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents. Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size. Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1). (1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996 Vanadium compounds are considered to have variable toxicity. Vanadium compounds act chiefly as an irritant to the conjunctivae and respiratory tract. Acute and chronic exposure can give rise to conjunctivitis, rhinitis, reversible irritation of the respiratory tract, and to bronchitis, bronchospasms, and asthma-like diseases in more severe cases. Industrial exposure are mostly acute, seldom chronic. Based on occupational exposure studies, human experimental studies, and studies in laboratory animals, the respiratory tract following inhalation exposure and the gastrointestinal tract, haematological system, and developing organism following oral exposure are the primary targets of toxicity. Adverse respiratory effects have been reported in humans and animals exposed to

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vanadium compounds at concentrations much higher than those typically found in the environment. Although the available data in humans are limited, signs of airway irritation (e.g., coughing, wheezing, sore throat) have been reported in subjects acutely exposed to 0.6 mg vanadium/m³ and in workers exposed to vanadium pentoxide dust. These effects have persisted for days to weeks after exposure termination and are often not associated with alterations in lung function. Studies in laboratory animals provide strong support that the respiratory tract is the most sensitive target following inhalation exposure to vanadium. A variety of lung lesions including alveolar/bronchiolar hyperplasia, inflammation, and fibrosis have been observed in rats and mice exposed to vanadium pentoxide; the severity of the lesions is related to concentration and duration. The lung effects have been observed following acute exposure to 0.56 mg vanadium/m³ and chronic exposures to 0.28 mg vanadium/m³ and have been observed after 2 days of exposure. Longer duration exposures also result in inflammation and hyperplasia in the larynx and hyperplasia in nasal goblet cells. These histological alterations result in restrictive impairments in lung function; respiratory distress is observed at vanadium pentoxide concentrations of =4.5 mg vanadium/m³. Other sensitive targets of vanadium toxicity include the gastrointestinal system following oral exposure and haematological system following inhalation or oral exposure. Symptoms of gastrointestinal irritation (diarrhea, cramps, nausea) have been observed in humans following bolus administration of sodium metavanadate, vanadyl sulfate, ammonium vanadyl tartrate, or diammonium vanado-tartrate as a treatment in non-insulin-dependent diabetics or patients with ischemic heart disease. The gastrointestinal effects occurred following ingestion of =14 mg vanadium and no effects were observed in subjects ingesting capsules containing 7.8 mg vanadium. In most studies the gastrointestinal effects occurred during the first week or two of the study suggesting that with repeated exposure, humans develop a tolerance to these effects. Diarrhea has also been observed in rats and mice orally exposed to lethal doses of vanadium. Microcytic erythrocytosis (evidenced by decreases in haematocrit, haemoglobin, and mean cell volume and increases in reticulocytes and nucleated erythrocytes) has been observed in rats exposed to 1.1 mg vanadium/m³ as vanadium pentoxide for at least 4 days. Haematological effects, including decreases in erythrocyte levels, decreases in haemoglobin, and increases in reticulocytes have also been observed in rats orally exposed to 1.18 mg vanadium/kg/day as ammonium metavanadate for 4 weeks.

Information on the potential of vanadium to induce developmental effects in humans is limited, but developmental effects have been observed in laboratory animals. Decreases in pup growth have been observed at maternal doses of 2.1 mg vanadium/kg/day. At higher doses, decreases in pup survival and gross, skeletal, and visceral malformations and anomalies have been reported; marked decreases in maternal body weight are also observed at these dose levels.

An increase in lung carcinoma incidence has been observed in mice chronically exposed to vanadium pentoxide; there is also marginal evidence for lung cancer in male rats (incidence of carcinoma was higher than historical controls but not concurrent controls). Carcinogenicity has not been adequately assessed in laboratory animals following oral exposure.

Vanadium is thought to be an essential trace element with the required level in human nutrition thought to be very low. Feeding trials in humans conducted over 45-94 days (1575-8375 mg of ammonium vanadyl tartrate) produced gastrointestinal distress but no changes in clinical chemistry. Ingestion of 50 mg/day resulted in transient green discolouration of the tongue. Amongst workers in a vanadium refinery exposed at levels of up to 12 mg/m³ cases of respiratory irritation and chronic bronchitis have been described. Emphysema and intoxication was found in boiler cleaners (vanadium is found in soot generated in oil-burning facilities) where vanadium exposures ranged from 30-104 mg/m³. Vanadium exposed workers complain of significantly more wheezing than their matched controls although no differences appear in chest radiography, forced vital capacity (FCV) or FEV1 in workers exposed at levels of 0.1 to 3.9 mg/m³.

Occupational studies showed that urinary vanadium levels significantly increased in exposed workers. Male and female workers exposed to 0.1-0.19 mg/m³ vanadium in a manufacturing company, had significantly higher urinary levels (20.6 ug/L) than the non-occupationally exposed control subjects (2.7 ug/L). The correlation between ambient vanadium levels and urinary levels of vanadium is difficult to determine from these epidemiological studies. In most instances, no other excretion routes were monitored. Analytical studies have shown very low levels in human milk. Evidence from animal studies supports the occupational findings. Vanadium administered intratracheally to rats was reported to be excreted predominantly in the urine at levels twice that found in the faeces. Epidemiological studies and animal studies suggest that elimination of vanadium following inhalation exposure is primarily in the urine.

Biological function

A number of vanadium dependent enzymes have been found in lower organisms, such as bacteria and algae. In higher animals and humans, however, no specific biochemical function has yet been identified for vanadium. Nevertheless, the possibility has been considered that vanadium might play a role in the regulation of some enzymes, such as the Na⁺/K⁺-exchanging ATPase, phosphoryl-transfer enzymes, adenylate cyclase and protein kinases. Therefore, its role in hormone, glucose, lipid, bone and tooth metabolism has also been discussed. Vanadium substances have been shown to mimic the action of insulin in isolated cell systems, animal models and diabetic patients. Therefore, their use in the therapy of diabetes mellitus has been considered. Vanadium has also been suggested as an aid in body building, but there is no evidence that it is effective. Altogether, vanadium has not been shown to be essential for humans and does not have a nutritional value. Even though some signs of vanadium deficiency have been reported in goats and rats, vanadium deficiency has not been identified in humans.

Metabolism

Vanadium is an element, and as such, is not subject to metabolism as such. However, vanadium transforms rather quickly to predominantly pentavalent vanadium species upon dissolution which can be expected to represent the predominant species under all physiological circumstances perhaps except for inhalation and subsequent uptake by the lysosomes of macrophages. Once systemically available, vanadium is subject to changes in speciation or valence, i.e. interconversion of the two oxidation states, the tetravalent form, vanadyl (VO²⁺) and the pentavalent form vanadate (VO₃⁻). The anionic pentavalent form is reported to predominate in extracellular fluids whilst the cationic tetravalent vanadyl ion appears to be the most common intracellular form. Thus, in the oxygenated blood, it circulates as vanadate but in tissues, it is retained mainly as vanadyl. Depending on the availability of reducing agents, including reduced glutathione-SH, NADPH, NADH, and oxygen, vanadium may be reduced, reoxidised, and/or undergo redox cycling.

Vanadium can reversibly bind to transferrin protein in the blood and then be taken up into erythrocytes. These two factors may affect the biphasic clearance of vanadium that occurs in the blood. There is a slower uptake of vanadyl into erythrocytes compared to the vanadate form. Five minutes after an intravenous administration of radiolabeled vanadate or vanadyl in dogs, 30% of the vanadate dose and 12% of the vanadyl dose is found in erythrocytes. It is suggested that this difference in uptake is due to the time required for the vanadyl form to be oxidized to vanadate.

In bioaccessibility tests of tri-, tetra- and pentavalent vanadium substances and metallic vanadium, tetra- and pentavalent forms dissolved completely within 2h in various media selected to simulate relevant human-chemical interactions. However, only < 2% of metallic vanadium and <20% of V₂O₃ went into solution. For metallic vanadium, it may be hypothesised that the oxide layer covering the particles goes more or less immediately into solution, whereas after this initial dissolution process vanadium remains practically inert. Similar observations were made for V₂O₃, suggesting that also in this case an oxidised layer covers the

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particle surface, resulting in a higher initial release rate that levels off during the initial 2 hours incubation period. Despite differences in solubility, the bioaccessibility data suggest the following:

- All vanadium substances upon dissolution transform predominantly into pentavalent forms in physiological media, with the exception of artificial lung fluid (ALF) in which tetravalent V was the predominant species present after 2 and 24 hours.
- Metallic vanadium is rather inert in physiological media, but any dissolved material transforms rather quickly to tetra- or pentavalent vanadium species, which are the main species in all physiological media tested.
- The low concentration of vanadium normally present in urine compared with the daily intake and the faecal levels indicate, that less than 5% of ingested vanadium is absorbed. The results of animal studies are in general in agreement with this conclusion. Other studies in rats have indicated that amounts greater than 10% can be absorbed from the gastrointestinal tract under some conditions
- In-vivo data are available on soluble tetra- and pentavalent vanadium substances (V₂O₅, NaVO₃, and VOSO₄), suggesting an oral absorption value of 16%. For the trivalent vanadium trioxide, this value is also adopted for lack of substance-specific information, albeit recognising that this constitutes a somewhat conservative value in view of the moderate bioaccessibility. For vanadium metal, the very poor bioavailability (i.e., approx. 100-fold less than for soluble tetra- and pentavalent vanadium substances) supports assuming a default oral absorption factor of 1%.

Genetic toxicity:

Representative tri-, tetra- and pentavalent vanadium substances have been verified unequivocally in vitro to be void of gene mutation activity both in bacterial as well as mammalian in-vitro cell systems. Testing in bacteria reverse mutation assays, although of limited relevance for metals, also yield negative results. Negative as well as positive results were obtained in clastogenicity assays in vitro. However, these in vitro studies of effects upon eukaryotic cells in vitro employed high concentrations of soluble vanadium producing significant levels of cytotoxicity and only weak genotoxic responses. Vanadium substances may express some clastogenicity in vitro, this occurs only at unphysiologically high concentrations and via mechanisms that appear to lack physiological relevance, and is not mirrored in corresponding in vivo assays via the oral and the inhalation route.

Based on the available weight-of-evidence, and considering guideline-conform studies conducted under GLP both in vitro as well as in vivo, vanadium should be considered void of genotoxicity.

Toxicity to reproduction:

Studies in animals exposed during pregnancy have shown that vanadium can cause decreases in growth and increases in the occurrence of birth defects. These effects are usually observed at levels which cause effects in the mother. Effects have also been observed at vanadium doses which did not cause effects in the mother

In studies with sodium ortho vanadate, embryoletality and teratogenicity were not observed at maternally toxic doses and below, but foetal toxicity was evidenced by a significant delay in the ossification process of some skeletal districts at 30 mg/kg body weight/day. The NOAEL for maternal toxicity was 7.5 mg/kg body weight/day and 15 mg/kg body weight/day represented a NOAEL for developmental toxicity in mice under the conditions of this study.

Neurotoxicity:

The effect of vanadate on selected neurotransmitters was limited to the hypothalamic region.

Vanadate caused a dose-related decrease of norepinephrine and its metabolite homovanillic acid (HVA) as well as of dopamine in the hypothalamus which are sensitive to oxidation.

5-HT as well as other metabolites of biogenic amines were not significantly reduced

The study indicated effects on neurotransmitter levels and their metabolism in rat brain following exposure to vanadate.

Pure calcium carbonate does not produce pneumoconiosis probably being eliminated from the lungs slowly by solution.

As mined, unsterilised particulates can carry bacteria into the air passages and lungs, producing infection and bronchitis.

High blood concentrations of calcium ion may give rise to vasodilation and depress cardiac function leading to hypotension and syncope. Calcium ions enhance the effects of digitalis on the heart and may precipitate digitalis intoxication. Calcium salts also reduce the absorption of tetracyclines

In neonates calcification of soft-tissue has been observed following therapeutic administration.

Some studies show that large quantities of calcium intake can cause hypercalcemia, which can in turn lead to renal failure. Renal failure can occur within hours or days or, alternatively, settles gradually, evolving over several years until it reaches terminal stages. Similarly, acute renal failure can also develop into chronic forms of the disease.

Hypercalcaemia conditions can be associated with normal or reduced calcium serum levels, as the body tends to maintain a balanced metabolism of the mineral, known as the compensation phase. When there is a slight increase in the concentration of ions in the blood, calcium excretion markedly increases, while intestinal absorption decreases. After kidney damage has set in, a loss of calcium may occur, thereby decreasing the serum concentration.

Serum protein levels may decrease as a result of proteinuria in cases of renal complications. Proteinuria is an indicator of kidney disease and represents an independent risk factor for the progression of such a condition. Increased serum creatinine levels may represent an important parameter, given that kidney diseases are associated with increased serum creatinine levels. When renal pathology occurs, a progressive loss of glomerular filtration begins, resulting in increased plasma creatinine concentrations. During the course of kidney failure, discrete, but constant, increments in plasma creatinine levels occur.

Renal disease with albuminuria may also be the cause of hypoalbuminemia in patients with liver disease. In cases of established liver damage, increased calcium urinary excretion may occur. Therefore, a similar increase may cause the decline in serum calcium levels in the current study.

Long term exposure to the dusts of titanium and several of its compounds produces chronic lung disease (fibrosis) in animals.

Radiological evidence exists amongst titanium dioxide workers suggesting chronic lung changes which resemble a slight form of silicosis. Workers chronically exposed to titanium or titanium dioxide dusts show a high incidence of chronic bronchitis (endobronchitis and peribronchitis). Early stages of this disease are characterised by impaired pulmonary respiration and ventilatory capacity and by reduced blood alkalinity. Cardiac changes characteristic of pulmonary disease (with hypertrophy of the right auricle) have also been observed amongst workers.

Titanium employed in implants has provoked immune responses which occur locally as metallosis and systemically as raised serum levels of activated T-lymphocytes. Some concern has been expressed about the potential for generating bone-resorbing mediators associated with titanium wear-debris.

The largest of the cohort studies was among white male production workers in the titanium dioxide industry in six European countries. The study indicated a slightly increased risk for lung cancer compared with the general population. However, there was no evidence of an exposure-response relationship within the cohort. No increase in the mortality rates for kidney cancer was found when the cohort was compared with the general population, but there was a suggestion of an exposure-response relationship in internal analyses. The other cohort studies, both of which were conducted in the USA, did not report an increased risk for lung cancer or cancer at any other site; no results for kidney cancer were reported, presumably because there were few cases.

Continued...

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One population-based case-control study conducted in Montreal did not indicate an increased risk for lung or kidney cancer. In summary, the studies do not suggest an association between occupational exposure to titanium dioxide as it occurred in recent decades in western Europe and North America and risk for cancer.

All the studies had methodological limitations; misclassification of exposure could not be ruled out. None of the studies was designed to assess the impact of particle size (fine or ultrafine) or the potential effect of the coating compounds on the risk for lung cancer.

An increased incidence of lung adenomas in rats of both sexes and of cystic keratinising lesions, diagnosed as squamous cell carcinomas in female rats, was seen in animals subject to high doses of inhaled titanium dioxide. Intratracheal delivery of titanium dioxide in combination with benz[a]pyrene produced an increase in benign and malignant tumours of the larynx, trachea and lungs in hamsters.

Squamous cell carcinomas developed after exposure to 250 mg/m³ for 6 hours/day, 5 days/week for 2 years in rats; the type of carcinoma that developed was considered to be a unique experimentally induced tumour and to be of questionable relevance for extrapolation of the results to humans. Given the extremely high level of dust in the lungs, the carcinomas were postulated to be the result of saturation of the normal pulmonary clearance mechanisms. At 50 mg/m³, massive accumulations of dust-laden macrophages, foamy dust cells and free particles were considered indicative of such overload.

Symptoms of chronic bismuth toxicity in humans consist of decreased appetite, weakness, rheumatic pain, diarrhoea, fever, foul breath, gingivitis and dermatitis. A blue line of the gums, the "bismuth line", may persist for years after exposure has ceased. Jaundice and conjunctival haemorrhage are rare, but have been reported. Bismuth neuropathy (kidney damage), with proteinuria may occur. The kidney is the site of highest concentration with liver concentrations being significantly lower. Renal failure may be reversible if treated early but anuria and death have occurred.

Loose and granular forms produce more dust than preforms (batts) but handling of batts results in fibre dislodgement and dusting. Repeated exposure results in immune response (toughening of skin) so that irritation (rash) often subsides in 2-3 weeks. The irritation and response recurs if exposure is intermittent. If irritation persists, worker exposure must be terminated and medical opinion sought.

There is little evidence for acute toxicity after inhalation of man-made mineral fibres (MMMF). Chronic inhalation of respirable fibres lead to pulmonary fibrosis

Rockwool contains a small proportion of respirable fibres. [CCINFO, ILO ENCYCLOPEDIA] Glasswool administered by inhalation produces little pulmonary fibrosis in experimental animals. No increase in the occurrence of mesothelioma has been observed in man-made mineral fibre / glass fibre production workers. [IARC Monograph 43]

Inhaled synthetic mineral fibres (SMFs) generally exhibit some level of biopersistence, resisting changes in number, dimension, surface chemistry, chemical composition, surface area and other characteristics, depending on their composition. Alteration to any of these parameters, in turn, alters a fibre's residence in the lung, and as a result, the lung's long-term response to the fibre. Fibres, of sufficiently small length, may undergo macrophage-mediated clearance in the lung. For fibres that are too long to be dealt with by alveolar macrophages, principal alternate clearance mechanisms include translocation to other thoracic compartments, dissolution and/or transverse breakage into shorter segments. In vitro fibre dissolution experiments show a broad range of dissolution rate constants ($K_{dis} = \text{ng/cm}^2/\text{hr}$) for the various synthetic vitreous (glass-like) fibres. For refractory ceramic fibres (RCF) the K_{dis} is 3 whilst for slag wool the K_{dis} is greater than 400. In vitro fibre-degradation studies demonstrate a direct relationship between the fibre's rate of leaching (some components dissolve more rapidly than others leaving a depleted silica matrix) and its tendency to undergo transverse fragmentation. Synthetic mineral fibres tend to break transversely in contrast to asbestos which tends to split longitudinally. This is significant for pathogenesis because over a long period of time in the lung, the actual numbers of long asbestos fibre in the lung can increase due to splitting along the long axis whilst the number of long SMFs decrease as a result of splitting along the short axis. Lung clearance by macrophages and the mucociliary escalator has been found, experimentally, to be more efficient for shorter segments.

Fibres which exhibit a rapid rate of leaching and fragmentation in the lung are less biopersistent, even though they may not dissolve completely. Fiber toxicology tends to be dominated by physical characteristics such as shape and length whilst nonfibrous dusts exhibit a chemical origin of toxicity. Early rodent studies found no tumourigenesis as a result of inhalation exposure to several types of fibreglass other than transient lung inflammation that resolved after a brief recovery period. However, hamsters exposed to a special application glass of high durability (475 glass) developed minimal lung fibrosis; one animal out of 125 developed mesothelioma. This is the first published report of permanent lung damage in laboratory animals following inhalation of glass fibre compositions (albeit of a special type). Preliminary results from another study with another high durability glass (E glass) showed fibrosis and pulmonary tumours in rats exposed to high concentrations of inhaled microfibres. (E glass is now produced as a continuous filament that is too thick to be respirable and is no longer available as microfibres). Several studies have demonstrated that glass fibres, insulation glass in particular, clear the lung more rapidly than amosite asbestos. Early inhalation studies of the chronic toxicity of refractory ceramic fibre (RCF1) reported conflicting results. In more recent studies rodents were exposed to four types of RCF (RCF1-4) for 6 hours/day, 5 days/week, at a maximum dose of 30 mg/m³ (test fibres were selected to have dimensions close to 1 μm x 20 μm); in rats RCF1 induced lung fibrosis, lung tumours (13%) and pleural mesothelioma (1.6%). In hamsters RCF1 induced lung fibrosis, mesothelioma (38%) and no lung tumours. Species related differences also raise the issue of significance of these findings in humans. Early rodent inhalation studies reported no fibrosis or tumours with chronic exposure to mineral wool. More recent studies with two compositions of mineral wool, - rock wool (MMVF21) and slag wool (MMVF22), size selected to have average dimensions of 1 μm x 20 μm , showed that neither mineral wool was tumourigenic, in rats but that MMVF21 produced minimal lung fibrosis late in the inhalation period. Biopersistence, as represented by 90% clearance rate (T-90), has been shown, experimentally to agree well with toxicity; composition with long-fibre T-90s greater than 200 days were all fibrogenic and all but MMVF21 were associated with tumourigenesis. This relationship can be explained as follows; fibres too thick to be inhaled into the lower lung or short enough to be transported by alveolar macrophages, are quickly cleared from the respiratory tract and will probably produce no other response than transient pulmonary inflammation. Long fibres with diameters less than 3 μm are able to penetrate into the lower lung but will become innocuous if they dissolve rapidly, can break transversely into smaller segments that can be cleared by alveolar macrophages and ciliated epithelium. Long thin fibres that reach the lower lung in sufficient quantities will be pathogenic if they do not dissolve or fragment. Surface chemistry may also play a role; In recent rat inhalation studies, E glass fibres but not 475 glass fibres were pathogenic in rats although both appeared to clear from the rat lung at about the same time. However during a year of post-exposure recovery 475 glass fibres underwent significant changes in chemistry due to leaching. Overexposure to the breathable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity and chest infections. Repeated exposures in the workplace to high levels of fine-divided dusts may produce a condition known as pneumoconiosis, which is the lodgement of any inhaled dusts in the lung, irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50000 inch) are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of

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breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses, the cough produces stringy phlegm, vital capacity decreases further, and shortness of breath becomes more severe. Other signs or symptoms include changed breath sounds, reduced oxygen uptake during exercise, emphysema and rarely, pneumothorax (air in the lung cavity).

Removing workers from the possibility of further exposure to dust generally stops the progress of lung abnormalities. When there is high potential for worker exposure, examinations at regular period with emphasis on lung function should be performed. Inhaling dust over an extended number of years may cause pneumoconiosis, which is the accumulation of dusts in the lungs and the subsequent tissue reaction. This may or may not be reversible.

Prolonged or repeated minor exposure to ammonia gas/vapour may cause long-term irritation to the eyes, nose and upper respiratory tract. Repeated exposure or prolonged contact may produce dermatitis, and conjunctivitis.

Other effects may include ulcerative changes to the mouth and bronchial and gastrointestinal disturbances. Adaptation to usually irritating concentrations may result in tolerance. In animals, repeated exposures to sub-lethal levels produces adverse effects on the respiratory tract, liver, kidneys and spleen. Exposure at 675 ppm for several weeks produced eye irritation in dogs and rabbits; corneal opacity, covering between a quarter to one half of the total surface area, was evident in rabbits.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium	TOXICITY	IRRITATION
	Not Available	Not Available
calcium carbonate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 750ug/24H - Severe
	Inhalation (Rat) LC50: >3 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Rodent - rabbit): 500mg/24H - Moderate
C.I. Pigment White 6	TOXICITY	IRRITATION
	dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >2.28 mg/l4h ^[1]	Skin (Human): 300ug/3D (intermittent) - Mild
	Oral (Rat) LD50: >=2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
magnetite	TOXICITY	IRRITATION
	Oral (Rat) LD50: >10000 mg/kg ^[2]	Not Available
ethanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 17100 mg/kg ^[1]	Eye (Rodent - rabbit): 0.1mL
	Inhalation (Rat) LC50: 64000 ppm4h ^[2]	Eye (Rodent - rabbit): 100mg/4S - Moderate
	Oral (Rat) LD50: 7060 mg/kg ^[2]	Eye (Rodent - rabbit): 100uL - Moderate
		Eye (Rodent - rabbit): 500mg - Severe
		Eye (Rodent - rabbit): 500mg/24H - Mild
		Eye: adverse effect observed (irritating) ^[1]
		Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Human): 70%/2D
		Skin (Rodent - rabbit): 20mg/24H - Moderate
		Skin (Rodent - rabbit): 400mg - Mild
C.I. Pigment Black 11	TOXICITY	IRRITATION
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
C.I. Pigment Red 101	TOXICITY	IRRITATION
	Oral (Rat) LD50: >5000 mg/kg ^[1]	Not Available
ammonium hydroxide	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: 2000 ppm4h ^[2]	Eye (Rodent - rabbit): 1mg/30S - Severe
	Oral (Rat) LD50: 350 mg/kg ^[2]	Eye (Rodent - rabbit): 250ug - Severe

Continued...

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium

		Eye (Rodent - rabbit): 44ug - Severe
C.I. Pigment Yellow 184	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: >5.1 mg/l4h ^[2]	Not Available
	Oral (Rat) LD50: >5000 mg/kg ^[2]	
C.I. Pigment Blue 29	TOXICITY	IRRITATION
	Oral (Rat) LD50: >10000 mg/kg ^[2]	Not Available
C.I. Pigment Red 170	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >1.58 mg/L4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >10000 mg/kg ^[2]	
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

CALCIUM CARBONATE	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
C.I. PIGMENT WHITE 6	Substance has been investigated as a mutagen, tumorigen and primary irritant. For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. Animal carcinogenicity data Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative. The substance is classified by IARC as Group 3:

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium

	NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
C.I. PIGMENT BLACK 11	No data of toxicological significance identified in literature search.
C.I. PIGMENT BLUE 29	NOTE: 90 day (chronic), teratological and mutagenicity tests here all provided negative results. Animal tests have also demonstrated no skin irritation or sensitization. [ICI]
CALCIUM CARBONATE & C.I. PIGMENT BLACK 11 & AMMONIUM HYDROXIDE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
CALCIUM CARBONATE & AMMONIUM HYDROXIDE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
CALCIUM CARBONATE & ETHANOL	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.
C.I. PIGMENT BLACK 11 & C.I. PIGMENT RED 101	No significant acute toxicological data identified in literature search.

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

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
calcium carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	LC50	96h	Fish	>165200mg/L	4
	NOEC(ECx)	1h	Fish	4-320mg/l	4
C.I. Pigment White 6	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
	LC50	96h	Fish	1.85-3.06mg/l	4
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
magnetite	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	168h	Fish	43.9mg/l	4
ethanol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	275mg/l	2
	EC50	48h	Crustacea	2mg/L	4

Continued...

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium

	EC50(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	4
	LC50	96h	Fish	42mg/L	4
	EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
C.I. Pigment Black 11	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	18mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	0.05mg/l	2
	NOEC(ECx)	504h	Fish	0.52mg/l	2
C.I. Pigment Red 101	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	18mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	504h	Fish	0.52mg/l	2
	LC50	96h	Fish	0.05mg/l	2
ammonium hydroxide	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	33.3mg/L	4
	EC50(ECx)	96h	Crustacea	0.83mg/l	5
C.I. Pigment Yellow 184	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>10000mg/l	2
	EC0(ECx)	48h	Crustacea	>=100mg/l	2
C.I. Pigment Blue 29	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	000mg/l	Not Available
C.I. Pigment Red 170	Endpoint	Test Duration (hr)	Species	Value	Source
	ErC50	72h	Algae or other aquatic plants	17mg/l	2
	EC50	72h	Algae or other aquatic plants	>1mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

Toxic to aquatic organisms.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
C.I. Pigment White 6	HIGH	HIGH
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
C.I. Pigment White 6	LOW (BCF = 10)
ethanol	LOW (LogKOW = -0.31)

Mobility in soil

Ingredient	Mobility
C.I. Pigment White 6	LOW (Log KOC = 23.74)
ethanol	HIGH (Log KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▶ DO NOT allow wash water from cleaning or process equipment to enter drains.
	▶ It may be necessary to collect all wash water for treatment before disposal.
	▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
	▶ Where in doubt contact the responsible authority.
	▶ Recycle wherever possible.
	▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
	▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material).
	▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
calcium carbonate	Not Available
C.I. Pigment White 6	Not Available
magnetite	Not Available
ethanol	Not Available
C.I. Pigment Black 11	Not Available
C.I. Pigment Red 101	Not Available
ammonium hydroxide	Not Available
C.I. Pigment Yellow 184	Not Available
C.I. Pigment Blue 29	Not Available
C.I. Pigment Red 170	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
calcium carbonate	Not Available
C.I. Pigment White 6	Not Available
magnetite	Not Available
ethanol	Not Available
C.I. Pigment Black 11	Not Available
C.I. Pigment Red 101	Not Available
ammonium hydroxide	Not Available
C.I. Pigment Yellow 184	Not Available
C.I. Pigment Blue 29	Not Available
C.I. Pigment Red 170	Not Available

SECTION 15 Regulatory information

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

Continued...

calcium carbonate is found on the following regulatory lists

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

C.I. Pigment White 6 is found on the following regulatory lists

- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
- 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)

magnetite is found on the following regulatory lists

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

ethanol is found on the following regulatory lists

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

C.I. Pigment Black 11 is found on the following regulatory lists

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

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

- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
- Australian Inventory of Industrial Chemicals (AIIC)
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ammonium hydroxide is found on the following regulatory lists

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

C.I. Pigment Yellow 184 is found on the following regulatory lists

- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List
- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

C.I. Pigment Blue 29 is found on the following regulatory lists

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

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

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

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (C.I. Pigment White 6; magnetite; ethanol; C.I. Pigment Black 11; C.I. Pigment Red 101; ammonium hydroxide; C.I. Pigment Yellow 184; C.I. Pigment Blue 29; C.I. Pigment Red 170)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium

National Inventory	Status
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (magnetite; C.I. Pigment Yellow 184)
Vietnam - NCI	Yes
Russia - FBEPH	No (magnetite)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	19/09/2024
Initial Date	04/06/2024

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	02/07/2024	Toxicological information - Chronic Health, Hazards identification - Classification, Composition / information on ingredients - Ingredients, Identification of the substance / mixture and of the company / undertaking - Supplier Information
5.1	19/09/2024	Hazards identification - Classification, Identification of the substance / mixture and of the company / undertaking - Supplier Information, Name

Other information

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

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

Definitions and abbreviations

- PC - TWA: Permissible Concentration-Time Weighted Average
- PC - STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit,
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas

Airfield, AquaRoute, Concrete Primer, Ecoline, Hi-Brite, High Build, Roadmarking, Roadmarking Premium

- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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