



8464 Static Dissipative, Anti-Corrosive Grease

MG Chemicals UK Limited

Version No: A-1.01

Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date: 20/06/2019

Revision Date: 18/03/2020

L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	8464
Synonyms	SDS Code: 8464; 8464-3ML, 8464-24ML, 8464-1, 8464-2, 8464-85ML, 8464-1P
Other means of identification	Static Dissipative, Anti-Corrosive Grease

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

Relevant identified uses	Low volatility, static dissipative grease
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	+(44) 1663 362888	+(1) 800-201-8822
Fax	Not Available	+(1) 800-708-9888
Website	Not Available	www.mgchemicals.com
Email	sales@mgchemicals.com	Info@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	Verisk 3E (Access code: 335388)
Emergency telephone numbers	+(44) 20 35147487
Other emergency telephone numbers	+(0) 800 680 0425

SECTION 2 HAZARDS IDENTIFICATION

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	H410 - Chronic Aquatic Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

2.2. Label elements

Hazard pictogram(s)	
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SIGNAL WORD **WARNING**

Hazard statement(s)

H410	Very toxic to aquatic life with long lasting effects.
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Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P273	Avoid release to the environment.
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Precautionary statement(s) Response

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P391	Collect spillage.
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Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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2.3. Other hazards

REACH - Art.57-59: The mixture does not contain Substances of Very High Concern (SVHC) at the SDS print date.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**3.1. Substances**

See 'Composition on ingredients' in Section 3.2

3.2. Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.1314-13-2 2.215-222-5 3.030-013-00-7 4.01-2119463881-32-XXXX 01-2120089607-43-XXXX	37	<u>zinc oxide</u>	Chronic Aquatic Hazard Category 1, Acute Aquatic Hazard Category 1; H410, H400 ^[2]
1.1344-28-1 2.215-691-6 3.Not Available 4.01-2119529248-35-XXXX	30	<u>aluminium oxide</u>	Not Applicable
1.7782-42-5 2.231-955-3 266-010-4 3.Not Available 4.01-2119486977-12-XXXX	3	<u>graphite, natural</u>	Specific target organ toxicity - repeated exposure Category 2; H373 ^[1]
1.1333-86-4 2.215-609-9 422-130-0 3.Not Available 4.01-2119384822-32-XXXX 01-2120767622-50-XXXX 01-0000016864-62-XXXX	1.5	<u>carbon black</u>	Carcinogenicity Category 2; H351 ^[1]
Legend:	1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available		

SECTION 4 FIRST AID MEASURES**4.1. Description of first aid measures**

Eye Contact	<p>If this product comes in contact with eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with water. ▶ If irritation continues, seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

- ▶ Absorption of zinc compounds occurs in the small intestine.
- ▶ The metal is heavily protein bound.
- ▶ Elimination results primarily from faecal excretion.
- ▶ The usual measures for decontamination (Ipecac Syrup, lavage, charcoal or cathartics) may be administered, although patients usually have sufficient vomiting not to require them.
- ▶ CaNa2EDTA has been used successfully to normalise zinc levels and is the agent of choice.

[Ellenhorn and Barceloux: Medical Toxicology]

- ▶ Manifestation of aluminium toxicity include hypercalcaemia, anaemia, Vitamin D refractory osteodystrophy and a progressive encephalopathy (mixed dysarthria-apraxia of speech, asterixis, tremulousness, myoclonus, dementia, focal seizures). Bone pain, pathological fractures and proximal myopathy can occur.
- ▶ Symptoms usually develop insidiously over months to years (in chronic renal failure patients) unless dietary aluminium loads are excessive.
- ▶ Serum aluminium levels above 60 ug/ml indicate increased absorption. Potential toxicity occurs above 100 ug/ml and clinical symptoms are present when levels exceed 200 ug/ml.
- ▶ Deferoxamine has been used to treat dialysis encephalopathy and osteomalacia. CaNa2EDTA is less effective in chelating aluminium.

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[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 FIREFIGHTING MEASURES**5.1. Extinguishing media**

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

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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5.3. Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use water delivered as a fine spray to control fire and cool adjacent 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	<p>Under certain conditions the material may become combustible because of the ease of ignition which occurs after the material reaches a high specific area ratio (thin sections, fine particles, or molten states). However, the same material in massive solid form is comparatively difficult to ignite. Nearly all metals will burn in air under certain conditions. Some are oxidised rapidly in the presence of air or moisture, generating sufficient heat to reach their ignition temperatures.</p> <p>Others oxidise so slowly that heat generated during oxidation is dissipated before the metal becomes hot enough to ignite.</p> <p>Particle size, shape, quantity, and alloy are important factors to be considered when evaluating metal combustibility. Combustibility of metallic alloys may differ and vary widely from the combustibility characteristics of the alloys' constituent elements.</p> <p>Combustible. Will burn if ignited. metal oxides</p> <p>When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES**6.1. Personal precautions, protective equipment and emergency procedures**

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid contact with skin and eyes. ▶ Wear impervious gloves and safety glasses. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▶ Do NOT use air hoses for cleaning ▶ Place spilled material in clean, dry, sealable, labelled container.
Major Spills	<p>Environmental hazard - contain spillage. Moderate hazard.</p> <ul style="list-style-type: none"> ▶ CAUTION: Advise personnel in area. ▶ Alert Emergency Services and tell them location and nature of hazard. ▶ Control personal contact by wearing protective clothing. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Recover product wherever possible. ▶ IF DRY: Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. IF WET: Vacuum/shovel up and place in labelled containers for disposal. ▶ ALWAYS: Wash area down with large amounts of water and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise Emergency Services.

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE**7.1. Precautions for safe handling**

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area.
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	<ul style="list-style-type: none"> ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Fire and explosion protection	See section 5
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry area protected from environmental extremes. ▶ 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. <p>For major quantities:</p> <ul style="list-style-type: none"> ▶ Consider storage in banded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). ▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<p>Derivative of electropositive metal. For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate.</p> <p>Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.</p> <p>Zinc oxide:</p> <ul style="list-style-type: none"> ▶ slowly absorbs carbon dioxide from the air. ▶ may react, explosively with magnesium and chlorinated rubber when heated ▶ is incompatible with linseed oil (may cause ignition) ▶ WARNING: Avoid or control reaction with peroxides. All <i>transition metal</i> peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive. ▶ Avoid reaction with borohydrides or cyanoborohydrides ▶ Metals and their oxides or salts may react violently with chlorine trifluoride and bromine trifluoride. ▶ These trifluorides are hypergolic oxidisers. They ignite on contact (without external source of heat or ignition) with recognised fuels - contact with these materials, following an ambient or slightly elevated temperature, is often violent and may produce ignition. ▶ The state of subdivision may affect the results. ▶ Avoid strong acids, bases.

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: respirable dust	4 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: inhalable dust	10 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m3	7 mg/m3	Not Available	Not Available

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EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
zinc oxide	Zinc oxide	10 mg/m ³	15 mg/m ³	2,500 mg/m ³
aluminium oxide	Aluminum oxide; (Alumina)	5.7 mg/m ³	15 mg/m ³	25 mg/m ³
graphite, natural	Graphite; (Mineral carbon)	6 mg/m ³	16 mg/m ³	95 mg/m ³
carbon black	Carbon black	9 mg/m ³	99 mg/m ³	590 mg/m ³

Ingredient	Original IDLH	Revised IDLH
zinc oxide	500 mg/m ³	Not Available
aluminium oxide	Not Available	Not Available
graphite, natural	1,250 mg/m ³	Not Available
carbon black	1,750 mg/m ³	Not Available

MATERIAL DATA

for zinc oxide:

Zinc oxide intoxication (intoxication zincale) is characterised by general depression, shivering, headache, thirst, colic and diarrhoea.

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

For graphite:

Graphite pneumoconiosis resembles coal workers' pneumoconiosis. Data indicate that the higher the crystalline silica content of graphite the more likely the disease will increase in severity. The presence of anthracite coal in the production of some synthetic grades of graphite appears to make arbitrary the use of the term, 'synthetic', 'artificial' or 'natural'.

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

The NIOSH REL-TWA was 'selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs'.

This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1.2 µm) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m³ over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For aluminium oxide:

The experimental and clinical data indicate that aluminium oxide acts as an 'inert' material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.

[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition

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

Because the margin of safety of the quartz TLV is not known with certainty and given the associated link between silicosis and lung cancer it is recommended that quartz concentrations be maintained as far below the TLV as prudent practices will allow.

Exposure to respirable crystalline silicas (RCS) represents a significant hazard to workers, particularly those employed in the construction industry where respirable dusts of cement and concrete are common. Cutting, grinding and other high speed processes, involving their finished products, may further result in dusty atmospheres. Bricks are also a potential source of RCSs under such circumstances.

It is estimated that half of the occupations, involved in construction work, are exposed to levels of RCSs, higher than the current allowable limits. Beaudry et al: Journal of Occupational and Environmental Hygiene 10: 71-77; 2013

8.2. Exposure controls

8.2.1. Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

- ▶ Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction.
- ▶ Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace.
- ▶ If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of:

(a): particle dust respirators, if necessary, combined with an absorption cartridge;

(b): filter respirators with absorption cartridge or canister of the right type;

(c): fresh-air hoods or masks

- ▶ Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding.

- ▶ Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting.


Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to efficiently remove the contaminant.

Type of Contaminant:	Air Speed:
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents

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	2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity
	3: Intermittent, low production.	3: High production, heavy use
	4: Large hood or large air mass in motion	4: Small hood-local control only
	<p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 4-10 m/s (800-2000 f/min) for extraction of crusher dusts generated 2 metres distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	
8.2.2. Personal protection		
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 	
Skin protection	See Hand protection below	
Hands/feet protection	<p>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.</p> <p>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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> - frequency and duration of contact, - chemical resistance of glove material, - glove thickness and - dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> - 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. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> - Excellent when breakthrough time > 480 min - Good when breakthrough time > 20 min - Fair when breakthrough time < 20 min - Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> - 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 <p>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.</p> <p>Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.</p> <ul style="list-style-type: none"> ▶ polychloroprene. ▶ nitrile rubber. ▶ butyl rubber. ▶ fluoroacoutchouc. ▶ polyvinyl chloride. <p>Gloves should be examined for wear and/ or degradation constantly.</p>	
Body protection	See Other protection below	
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. 	

Respiratory protection

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, ANSI Z88 or national equivalent)

Required Minimum Protection Factor

Half-Face Respirator

Full-Face Respirator

Powered Air Respirator

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up to 10 x ES	P1 Air-line*	-	PAPR-P1
up to 50 x ES	Air-line**	-	-
up to 100 x ES	-	P2 P3	PAPR-P2
100+ x ES	-	Air-line* Air-line**	- PAPR-P3

* - Negative pressure demand ** - Continuous flow

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)

If inhalation risk above the TLV exists, wear approved dust respirator.

Use respirators with protection factors appropriate for the exposure level.

- ▶ Up to 5 X TLV, use valveless mask type; up to 10 X TLV, use 1/2 mask dust respirator
- ▶ Up to 50 X TLV, use full face dust respirator or demand type C air supplied respirator
- ▶ Up to 500 X TLV, use powered air-purifying dust respirator or a Type C pressure demand supplied-air respirator
- ▶ Over 500 X TLV wear full-face self-contained breathing apparatus with positive pressure mode or a combination respirator with a Type C positive pressure supplied-air full-face respirator and an auxiliary self-contained breathing apparatus operated in pressure demand or other positive pressure mode
- ▶ 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 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.
- ▶ Use approved positive flow mask if significant quantities of dust becomes airborne.
- ▶ Try to avoid creating dust conditions.

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

9.1. Information on basic physical and chemical properties

Appearance	Dark Grey, grease		
Physical state	Solid	Relative density (Water = 1)	2.10
Odour	No Odour	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	>20.5
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	285	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 TOXICOLOGICAL INFORMATION

Continued...

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11.1. Information on toxicological effects

Inhaled	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p>
Ingestion	<p>Acute toxic responses to aluminium are confined to the more soluble forms.</p> <p>The material has NOT been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>Soluble zinc salts produces irritation and corrosion of the alimentary tract (in a manner similar to copper salts) with pain, vomiting, etc. Delayed deaths have been ascribed to inanition (weakness and extreme weight loss resulting from prolonged and severe food insufficiency) following severe strictures of the oesophagus, and pylorus. Vomiting, abdominal cramps, and diarrhea, in several cases with blood, have been observed after ingestion of zinc sulfate. Several cases of gastrointestinal disturbances have been reported after ingestion of zinc sulfate. A significant reduction in erythrocyte superoxide dismutase activity (47% decrease), hematocrit, and serum ferritin, compared to pretreatment levels, occurred in female subjects who received supplements (as capsules) of 50 mg zinc/day as zinc gluconate for 10 weeks. A 15% decrease in erythrocyte superoxide dismutase activity was reported in male volunteers receiving 50 mg zinc/day as zinc gluconate for 6 weeks. Another study reported increases in bone specific alkaline phosphatase levels (~25%) and extracellular superoxide dismutase (~15%), while significant decreases were seen in mononuclear white cell 5'-nucleotidase (~30%) and plasma 5'-nucleotidase activity (~36%) following exposure of postmenopausal women to a combined (dietary+supplemental) 53 mg zinc/day as zinc glycine chelate. Healthy men given 200 mg zinc/day as elemental zinc for 6 weeks showed a reduction in lymphocyte stimulation response to phytohemagglutinin as well as chemotaxis and phagocytosis of bacteria by polymorphonuclear leukocytes.; however, no changes in lymphocyte cell number or in the proportion of lymphocyte populations were noted. Exposure of male volunteers to 0.48 mg zinc/kg/day, as zinc glycine chelate, had no effect on markers of coagulation relative to unexposed subjects. While the changes in hematological end points following long-term zinc exposure in humans are noteworthy, they were subclinical in nature, and therefore, are generally considered to be non-adverse. In animals, following oral administration of zinc compounds, decreased hemoglobin, hematocrit, erythrocyte, and/or leukocyte levels were observed in rats, mice, rabbits, dogs, ferrets, and pruruminant calves. A number of intermediate-duration studies have demonstrated renal effects in animals exposed to zinc oxide, zinc sulfate, and zinc acetate. Zinc sulfate caused an increase in the absolute and relative kidney weights and regressive kidney lesions (not specified) in female mice that consumed 1,110 mg zinc/kg/day in the diet for 13 weeks, but no effects occurred in rats that consumed 565 mg zinc/kg/day or in mice that consumed 104 mg zinc/kg/day under similar conditions. Severe diffuse nephrosis was observed in ferrets exposed to 195 mg zinc/kg/day as zinc oxide in the diet. In rats exposed to 191 mg zinc/kg/day as zinc acetate for 3 months, epithelial cell damage in the glomerulus and proximal convoluted tubules and increased plasma creatinine and urea levels were observed. Zinc plays a role in the normal development and maintenance of the immune system, such as in the lymphocyte response to mitogens and as a cofactor for the thymic hormone thymulin. Oral exposure to zinc at levels much higher than the recommended daily dose has impaired immune and inflammatory responses. This was observed in in vivo investigations of the immune competence of blood components taken from 11 healthy adult men after ingestion of 4.3 mg zinc/kg/day as zinc sulfate for 6 weeks. The mitogenic response elicited from peripheral blood lymphocytes and the chemotactic and phagocytic responses of polymorphonuclear leukocytes were impaired after zinc ingestion. No effects were seen on total numbers of lymphocytes or relative numbers of T cells, T cell subsets, or B cells. The relationship between these observations and decreased levels of immune competence that might lead to increased susceptibility to disease is unknown. A later study reported no effects of supplementation of male volunteers with 30 mg zinc/day (0.43 mg zinc/kg/day assuming a reference male body weight of 70 kg) as zinc glycine chelate for 14 weeks on levels of peripheral blood leucocytes or on the frequency of lymphocyte subsets.</p> <p>Zinc appears to be necessary for normal brain function, but excess zinc is toxic. A 16-year-old boy who ingested .86 mg zinc/kg/day of metallic zinc over a 2-day period in an attempt to promote wound healing, developed signs and symptoms of lethargy, light-headedness, staggering, and difficulty in writing clearly. Lethargy was also observed in a 2-year-old child who ingested a zinc chloride solution (.1,000 mg zinc/kg). It is not known whether these observations represent direct effects on the nervous system. Very limited data were located regarding neurological effects in animals. Minor neuron degeneration and proliferation of oligodendroglia occurred in rats dosed with 487 mg zinc/kg/day as zinc oxide for 10 days. Rats receiving 472 mg zinc/kg/day for 10 days had increased levels of secretory material in the neurosecretory nuclei of the hypothalamus. Mice exposed postnatally to 0.5 mg zinc/kg/day as zinc acetate for 28 days showed no changes in memory formation, but showed a gradual decrease in learning extinction throughout the study.</p>
Skin Contact	<p>The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>Although the material is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may cause transient discomfort characterised by tearing or conjunctival redness (as with windburn). Slight abrasive damage may also result. The material may produce foreign body irritation in certain individuals.</p>
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminium oxide during abrasives production. Very fine Al₂O₃ powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.</p> <p>When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.</p> <p>Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.</p> <p>There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.</p> <p>Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.</p> <p>Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies,</p>

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aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of 'tau' a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade.

Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia*, August 1995]

Chronic symptoms produced by crystalline silicas included decreased vital lung capacity and chest infections. Lengthy exposure may cause silicosis a disabling form of pneumoconiosis which may lead to fibrosis, a scarring of the lining of the air sacs in the lung.

The form and severity in which silicosis manifests itself depends in part on the type and extent of exposure to silica dusts: chronic, accelerated and acute forms are all recognized. In later stages the critical condition may become disabling and potentially fatal. Restrictive and/or obstructive lung function changes may result from chronic exposure. A risk associated with silicosis is development of pulmonary tuberculosis (silico-tuberculosis). Respiratory insufficiencies due to massive fibrosis and reduced pulmonary function, possibly with accompanying heart failure, are other potential causes of death due to silicosis.

Not all individuals with silicosis will exhibit symptoms (signs) of the disease. However, silicosis can be progressive, and symptoms may potentially appear years after exposures have ceased. Symptoms of silicosis may include (but are not limited to): Shortness of breath; difficulty breathing with or without exertion; coughing; diminished work capacity; diminished chest expansion; reduction of lung volume; heart enlargement and/or failure.

Respirable dust containing newly broken particles has been shown to be more hazardous to animals in laboratory tests than respirable dust containing older silica particles of similar size. Respirable silica particles which had aged for sixty days or more showed less lung injury in animals than equal exposures of respirable dust containing newly broken pieces of silica. There are reports in the literature indicating that crystalline silica exposure may be associated with adverse health effects involving the kidney, scleroderma (thickening of the skin caused by swelling and thickening of fibrous tissue) and other autoimmune and immunity-related disorders. Several studies of persons with silicosis or silica exposure also indicate or suggest increased risk of developing lung cancer, a risk that may increase with the duration of exposure. Many of these studies of silicosis do not account for lung cancer confounders, especially smoking.

Symptoms may appear 8 to 18 months after initial exposure. Smoking increases this risk. Classic silicosis is a chronic disease characterised by the formation of scattered, rounded or stellate silica-containing nodules of scar tissue in the lungs ranging from microscopic to 1.0 cm or more. The nodules isolate the inhaled silica particles and protect the surrounding normal and functioning tissue from continuing injury. Simple silicosis (in which the nodules are less than 1.0 cm in diameter) is generally asymptomatic but may be slowly progressive even in the absence of continuing exposure. Simple silicosis can develop in complicated silicoses (in which nodules are greater than 1.0 cm in diameter) and can produce disabilities including an associated tuberculous infection (which 50 years ago accounted for 75% of the deaths among silicotic workers). Crystalline silica deposited in the lungs causes epithelial and macrophage injury and activation. Crystalline silica translocates to the interstitium and the regional lymph nodes and cause the recruitment of inflammatory cells in a dose dependent manner. In humans, a large fraction of crystalline silica persists in the lungs. The question of potential carcinogenicity associated with chronic inhalation of crystalline silica remains equivocal with some studies supporting the proposition and others finding no significant association. The results of recent epidemiological studies suggest that lung cancer risk is elevated only in those patients with overt silicosis. A relatively large number of epidemiological studies have been undertaken and in some, increased risk gradients have been observed in relation to dose surrogates - cumulative exposure, duration of exposure, the presence of radiographically defined silicosis, and peak intensity exposure. Chronic inhalation in rats by single or repeated intratracheal instillation produced a significant increase in the incidences of adenocarcinomas and squamous cell carcinomas of the lung. Lifetime inhalation of crystalline silica (87% alpha-quartz) at 1 mg/m³ (74% respirable) by rats, produced an increase in animals with keratinising cystic squamous cell tumours, adenomas, adenocarcinomas, adenosquamous cell carcinomas, squamous cell carcinoma and nodular bronchiolar alveolar hyperplasia accompanied by extensive subpleural and peribronchiolar fibrosis, increased pulmonary collagen content, focal lipoproteinosis and macrophage infiltration. Thoracic and abdominal malignant lymphomas developed in rats after single intrapleural and intraperitoneal injection of suspensions of several types of quartz.

Some studies show excess numbers of cases of scleroderma, connective tissue disorders, lupus, rheumatoid arthritis chronic kidney diseases, and end-stage kidney disease in workers

NOTE: Some jurisdictions require health surveillance be conducted on workers occupationally exposed to silica, crystalline. Such surveillance should emphasise

- demography, occupational and medical history and health advice
- standardised respiratory function tests such as FEV1, FVC and FEV1/FVC
- standardised respiratory function tests such as FV1, FVC and FEV1/FVC
- chest X-ray, full size PA view
- records of personal exposure

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Following an oral intake of extremely high doses of zinc (where 300 mg Zn/d – 20 times the US Recommended Dietary Allowance (RDA) – is a 'low intake' overdose), nausea, vomiting, pain, cramps and diarrhea may occur. There is evidence of induced copper deficiency, alterations of blood lipoprotein levels, increased levels of LDL, and decreased levels of HDL at long-term intakes of 100 mg Zn/d. The USDA RDA is 15 mg Zn/d.

There is also a condition called the 'zinc shakes' or 'zinc chills' or metal fume fever that can be induced by the inhalation of freshly formed zinc oxide formed during the welding of galvanized materials.

Supplemental zinc can prevent iron absorption, leading to iron deficiency and possible peripheral neuropathy, with loss of sensation in extremities.

Zinc is necessary for normal fetal growth and development. Fetal damage may result from zinc deficiency. Only one report in the literature suggested adverse developmental effects in humans due to exposure to excessive levels of zinc. Four women were given zinc supplements of 0.6 mg zinc/kg/day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant. However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming 0.3 mg zinc/kg/day as zinc sulfate or zinc citrate or 0.06 mg zinc/kg/day as zinc aspartate during the last two trimesters. There has been a suggestion that increased serum zinc levels in pregnant women may be associated with an increase in neural tube defects, but others have failed to confirm this association. The developmental toxicity of zinc in experimental animals has been evaluated in a number of investigations. Exposure to high levels of zinc in the diet prior to and/or during gestation has been associated with increased fetal resorptions, reduced fetal weights, altered tissue concentrations of fetal iron and copper, and reduced growth in the offspring.

Animal studies suggest that exposure to very high levels of dietary zinc is associated with reduced fetal weight, alopecia, decreased hematocrit, and copper deficiency in offspring. For example, second generation mice exposed to zinc carbonate during gestation and lactation (260 mg/kg/day in the maternal diet), and then continued on that diet for 8 weeks, had reduced body weight, alopecia, and signs of copper deficiency (e.g., lowered hematocrit and occasional achromotrichia [loss of hair colour]). Similarly, mink kits from dams that ingested a time-weighted-average dose of 20.8 mg zinc/kg/day as zinc sulfate also had alopecia and achromotrichia. It is likely that the alopecia resulted from zinc-induced copper deficiency, which is known to cause alopecia in monkeys. However, no adverse effects were observed in parental mice or mink. No effects on reproduction were reported in rats exposed to 50 mg zinc/kg/day as zinc carbonate; however, increased stillbirths were observed in rats exposed to 250 mg zinc/kg/day.

Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in 'metal fume fever'; also known as 'brass chills', an industrial disease of short duration. [I.L.O.] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas.

Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure.

On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Overexposure to respirable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity, chest infections

Repeated exposures, in an occupational setting, 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 (1/50,000 inch), are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion (exertional dyspnea), increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Other signs or symptoms include altered breath sounds, diminished lung capacity, diminished oxygen uptake during exercise, emphysema and pneumothorax (air in lung cavity) as a rare complication.

Removing workers from possibility of further exposure to dust generally leads to halting the progress of the lung abnormalities. Where worker-exposure potential is high, periodic examinations with emphasis on lung dysfunctions should be undertaken

Dust inhalation over an extended number of years may produce pneumoconiosis. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. It is further classified as being of noncollagenous or collagenous types. Noncollagenous pneumoconiosis, the benign form, is identified by minimal stromal reaction, consists mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible.

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	Not Available	Not Available
zinc oxide	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit) : 500 mg/24 h - mild
	Inhalation (rat) LC50: >1.79 mg/l4 h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: >5000 mg/kg ^[2]	Skin (rabbit) : 500 mg/24 h- mild Skin: no adverse effect observed (not irritating) ^[1]
aluminium oxide	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]
graphite, natural	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2 mg/l4 h ^[1]	Eye (rabbit): non-irritant *
	Oral (rat) LD50: >2000 mg/kg ^[2]	Eye : Not irritating Skin (rabbit): 4 h non-irritant *
		Skin : Not irritating
carbon black	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: >15400 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]

Legend:

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

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For aluminium compounds:

Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex. Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate).

Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability.

For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (μg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 μg per day is considered safe.

Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium.

The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 μg , the calculated systemic intake values for healthy skin are above the 8.6 μg per day that are considered safe for an adult weighing 60 kg. If aluminium-containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account.

Systemic toxicity after repeated exposure

No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium.

When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose.

The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent.

Reproductive and developmental toxicity:

Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity.

High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate. This study was used by JECFA as key study to derive the PTWI.

Genotoxicity

Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.

Carcinogenicity.

The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons.

Neurodegenerative diseases.

Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. Some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.

Contact sensitivity:

It has been suggested that the body burden of aluminium may be linked to different diseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines. The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome.

Aluminium acts not only as an adjuvant, stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitiser causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptensation to be immunogenic and to initiate an immune response. Once inside the skin, the metal ions must bind to proteins to become immunologically reactive. The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT). Nodules were overrepresented in

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	patients with contact allergy to aluminium Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste
ZINC OXIDE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
GRAPHITE, NATURAL	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. * Timcal MSDS
CARBON BLACK	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Inhalation (rat) TCLo: 50 mg/m ³ /6h/90D-1 Nil reported
ALUMINIUM OXIDE & CARBON BLACK	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

12.1. Toxicity

8464 Static Dissipative, Anti-Corrosive Grease	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
zinc oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.58mg/L	2
	EC50	48	Crustacea	0.001-0.014mg/L	2
	EC50	72	Algae or other aquatic plants	0.037mg/L	2
	BCF	336	Fish	4376.673mg/L	4
	NOEC	72	Algae or other aquatic plants	0.00008138mg/L	2
aluminium oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.134mg/L	2
	EC50	48	Crustacea	0.7364mg/L	2
	EC50	72	Algae or other aquatic plants	0.001-0.799mg/L	2
	NOEC	240	Crustacea	0.001-0.1002mg/L	2
graphite, natural	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>100mg/L	2
	NOEC	72	Algae or other aquatic plants	>=100mg/L	2
carbon black	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>10-mg/L	2

Continued...

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EC10	72	Algae or other aquatic plants	>10-mg/L	2
NOEC	96	Fish	>=1-mg/L	2

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

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

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

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

Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms.

When released to dry soil most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. Environmental processes may also be important in changing solubilities.

Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects.

A metal ion is considered infinitely persistent because it cannot degrade further.

The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.

The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.

Environmental processes may enhance bioavailability.

For zinc and its compounds:

Environmental fate:

Zinc is capable of forming complexes with a variety of organic and inorganic groups (ligands). Biological activity can affect the mobility of zinc in the aquatic environment, although the biota contains relatively little zinc compared to the sediments. Zinc bioconcentrates moderately in aquatic organisms; bioconcentration is higher in crustaceans and bivalve species than in fish. Zinc does not concentrate appreciably in plants, and it does not biomagnify significantly through terrestrial food chains.

However biomagnification may be of concern if concentration of zinc exceeds 1632 ppm in the top 12 inches of soil.

Zinc can persist in water indefinitely and can be toxic to aquatic life. The threshold concentration for fish is 0.1 ppm. Zinc may be concentrated in the aquatic food chain; it is concentrated over 200,000 times in oysters. Copper is synergistic but calcium is antagonistic to zinc toxicity in fish. Zinc can accumulate in freshwater animals at 5 -1,130 times the concentration present in the water. Furthermore, although zinc actively bioaccumulates in aquatic systems, biota appears to represent a relatively minor sink compared to sediments. Steady-state zinc bioconcentration factors (BCFs) for 12 aquatic species range from 4 to 24,000. Crustaceans and fish can accumulate zinc from both water and food. A BCF of 1,000 was reported for both aquatic plants and fish, and a value of 10,000 was reported for aquatic invertebrates. The order of enrichment of zinc in different aquatic organisms was as follows (zinc concentrations in µg/g dry weight appear in parentheses): fish (25), shrimp (50), mussel (60), periphyton (260), zooplankton (330), and oyster (3,300). The high enrichment in oysters may be due to their ingestion of particulate matter containing higher concentrations of zinc than ambient water. Other investigators have also indicated that organisms associated with sediments have higher zinc concentrations than organisms living in the aqueous layer. With respect to bioconcentration from soil by terrestrial plants, invertebrates, and mammals, BCFs of 0.4, 8, and 0.6, respectively, have been reported. The concentration of zinc in plants depends on the plant species, soil pH, and the composition of the soil.

Plant species do not concentrate zinc above the levels present in soil.

In some fish, it has been observed that the level of zinc found in their bodies did not directly relate to the exposure concentrations. Bioaccumulation of zinc in fish is inversely related to the aqueous exposure. This evidence suggests that fish placed in environments with lower zinc concentrations can sequester zinc in their bodies.

The concentration of zinc in drinking water may increase as a result of the distribution system and household plumbing. Common piping materials used in distribution systems often contain zinc, as well as other metals and alloys. Trace metals may enter the water through corrosion products or simply by the dissolution of small amounts of metals with which the water comes in contact.

Reactions with materials of the distribution system, particularly in soft low-pH waters, very often have produced concentrations of zinc in tap water much greater than those in the raw or treated waters at the plant of origin. Zinc gives water a metallic taste at low levels. Overexposures to zinc also have been associated with toxic effects. Ingestion of zinc or zinc-containing compounds has resulted in a variety of systemic effects in the gastrointestinal and hematological systems and alterations in the blood lipid profile in humans and animals. In addition, lesions have been observed in the liver, pancreas, and kidneys of animals.

Environmental toxicity of zinc in water is dependent upon the concentration of other minerals and the pH of the solution, which affect the ligands that associate with zinc.

Zinc occurs in the environment mainly in the +2 oxidation state. Sorption is the dominant reaction, resulting in the enrichment of zinc in suspended and bed sediments. Zinc in aerobic waters is partitioned into sediments through sorption onto hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their concentrations, pH, redox potential (Eh), salinity, nature and concentrations of complexing ligands, cation exchange capacity, and the concentration of zinc. Precipitation of soluble zinc compounds appears to be significant only under reducing conditions in highly polluted water. Generally, at lower pH values, zinc remains as the free ion. The free ion (Zn²⁺) tends to be adsorbed and transported by suspended solids in unpolluted waters.

Zinc is an essential nutrient that is present in all organisms. Although biota appears to be a minor reservoir of zinc relative to soils and sediments, microbial decomposition of biota in water can produce ligands, such as humic acids, that can affect the mobility of zinc in the aquatic environment through zinc precipitation and adsorption.

The relative mobility of zinc in soil is determined by the same factors that affect its transport in aquatic systems (i.e., solubility of the compound, pH, and salinity)

The redox status of the soil may shift zinc partitioning. Reductive dissolution of iron and manganese (hydr)oxides under suboxic conditions release zinc into the aqueous phase; the persistence of suboxic conditions may then lead to a repartitioning of zinc into sulfide and carbonate solids. The mobility of zinc in soil depends on the solubility of the speciated forms of the element and on soil properties such as cation exchange capacity, pH, redox potential, and chemical species present in soil; under anaerobic conditions, zinc sulfide is the controlling species.

Since zinc sulfide is insoluble, the mobility of zinc in anaerobic soil is low. In a study of the effect of pH on zinc solubility: When the pH is <7, an inverse relationship exists between the pH and the amount of zinc in solution. As negative charges on soil surfaces increase with increasing pH, additional sites for zinc adsorption are activated and the amount of zinc in solution decreases. The active zinc species in the adsorbed state is the singly charged zinc hydroxide species (i.e., Zn(OH)⁺). Other investigators have also shown that the mobility of zinc in soil increases at lower soil pH under oxidizing conditions and at a lower cation exchange capacity of soil. On the other hand, the amount of zinc in solution generally increases when the pH is >7 in soils high in organic matter. This is a result of the release of organically complexed zinc, reduced zinc adsorption at higher pH, or an increase in the concentration of chelating agents in soil. For calcareous soils, the relationship between zinc solubility and pH is nonlinear. At a high pH, zinc in solution is precipitated as Zn(OH)₂, zinc carbonate (ZnCO₃), or calcium zincate. Clay and metal oxides are capable of sorbing zinc and tend to retard its mobility in soil. Zinc was more mobile at pH 4 than at pH 6.5 as a consequence of sorption

Zinc concentrations in the air are relatively low, except near industrial sources such as smelters. No estimate for the atmospheric lifetime of zinc is available at this time, but the fact that zinc is transported long distances in air indicates that its lifetime in air is at least on the order of days. There are few data regarding the speciation of zinc released to the atmosphere. Zinc is removed from the air by dry and wet deposition, but zinc particles with small diameters and low densities suspended in the atmosphere travel long distances from emission sources.

For aluminium and its compounds and salts:

Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals. Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.

Environmental fate:

Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.

Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.

As an element, aluminium cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminium in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminium can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminium in the environment will depend on the ligands present and the pH.

The trivalent aluminium ion is surrounded by six water molecules in solution. The hydrated aluminium ion, [Al(H₂O)₆]³⁺, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H₂O)₅(OH)]²⁺, [Al(H₂O)₄(OH)₂]⁺). The speciation of aluminium in water is pH dependent. The hydrated trivalent aluminium ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)₂⁺ and Al(OH)₂⁺, while the solid Al(OH)₃ is most prevalent between pH 5.2 and 8.8.

The soluble species Al(OH)₄⁻ is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminium hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)₃, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminium is precipitated as poorly crystallised clay mineral species.

Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminium hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.

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Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.

The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.

Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.

Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead bioluted.

Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.

The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic.

Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.

Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.

Ecotoxicity:**Freshwater species pH >6.5**

Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar*) - 106 mg/L; Chronic NOEC (8-28 d): 7 spp, NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.

Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L

Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L

Freshwater species pH <6.5 (all between pH 4.5 and 6.0)

Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L

Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 mg/L (absolute range 0.40-5.2 mg/L)

Alga: 1 sp NOEC growth 2.0 mg/L

Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.

The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4-5.4, with a maximum toxicity occurring around pH 5.0-5.2. The inorganic single unit aluminium species (Al(OH)₂⁺) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H⁺ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.

Drinking Water Standards:

aluminium: 200 ug/l (UK max.)

200 ug/l (WHO guideline)

chloride: 400 mg/l (UK max.)

250 mg/l (WHO guideline)

fluoride: 1.5 mg/l (UK max.)

1.5 mg/l (WHO guideline)

nitrate: 50 mg/l (UK max.)

50 mg/l (WHO guideline)

sulfate: 250 mg/l (UK max.)

Soil Guideline: none available.

Air Quality Standards: none available.

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
zinc oxide	LOW (BCF = 217)

12.4. Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Applicable	Not Applicable	Not Applicable
PBT Criteria fulfilled?	Not Applicable	Not Applicable	Not Applicable

12.6. Other adverse effects

No data available

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SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ 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.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

	<p>NOT REGULATED by Ground ADR Special Provision 375 NOT REGULATED by Air IATA Special Provision A197 NOT REGULATED by Sea IMDG per 2.10.2.7 NOT REGULATED by ADN Special Provision 274 (The provision of 3.1.2.8 apply)</p>
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Land transport (ADR)

14.1. UN number	3077												
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)												
14.3. Transport hazard class(es)	<table border="0"> <tr> <td>Class</td> <td>9</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	9	Subrisk	Not Applicable								
Class	9												
Subrisk	Not Applicable												
14.4. Packing group	III												
14.5. Environmental hazard	Environmentally hazardous												
14.6. Special precautions for user	<table border="0"> <tr> <td>Hazard identification (Kemler)</td> <td>90</td> </tr> <tr> <td>Classification code</td> <td>M7</td> </tr> <tr> <td>Hazard Label</td> <td>9</td> </tr> <tr> <td>Special provisions</td> <td>274 335 375 601</td> </tr> <tr> <td>Limited quantity</td> <td>5 kg</td> </tr> <tr> <td>Tunnel Restriction Code</td> <td>3 (-)</td> </tr> </table>	Hazard identification (Kemler)	90	Classification code	M7	Hazard Label	9	Special provisions	274 335 375 601	Limited quantity	5 kg	Tunnel Restriction Code	3 (-)
Hazard identification (Kemler)	90												
Classification code	M7												
Hazard Label	9												
Special provisions	274 335 375 601												
Limited quantity	5 kg												
Tunnel Restriction Code	3 (-)												

Air transport (ICAO-IATA / DGR)

14.1. UN number	3077														
14.2. UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. * (contains zinc oxide)														
14.3. Transport hazard class(es)	<table border="0"> <tr> <td>ICAO/IATA Class</td> <td>9</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>9L</td> </tr> </table>	ICAO/IATA Class	9	ICAO / IATA Subrisk	Not Applicable	ERG Code	9L								
ICAO/IATA Class	9														
ICAO / IATA Subrisk	Not Applicable														
ERG Code	9L														
14.4. Packing group	III														
14.5. Environmental hazard	Environmentally hazardous														
14.6. Special precautions for user	<table border="0"> <tr> <td>Special provisions</td> <td>A97 A158 A179 A197</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>956</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>400 kg</td> </tr> <tr> <td>Passenger and Cargo Packing Instructions</td> <td>956</td> </tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td> <td>400 kg</td> </tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td> <td>Y956</td> </tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td> <td>30 kg G</td> </tr> </table>	Special provisions	A97 A158 A179 A197	Cargo Only Packing Instructions	956	Cargo Only Maximum Qty / Pack	400 kg	Passenger and Cargo Packing Instructions	956	Passenger and Cargo Maximum Qty / Pack	400 kg	Passenger and Cargo Limited Quantity Packing Instructions	Y956	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G
Special provisions	A97 A158 A179 A197														
Cargo Only Packing Instructions	956														
Cargo Only Maximum Qty / Pack	400 kg														
Passenger and Cargo Packing Instructions	956														
Passenger and Cargo Maximum Qty / Pack	400 kg														
Passenger and Cargo Limited Quantity Packing Instructions	Y956														
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G														

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3077
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)

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14.3. Transport hazard class(es)	IMDG Class	9
	IMDG Subrisk	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-A, S-F
	Special provisions	274 335 966 967 969
	Limited Quantities	5 kg

Inland waterways transport (ADN)

14.1. UN number	3077	
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)	
14.3. Transport hazard class(es)	9	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Classification code	M7
	Special provisions	274; 335; 375; 601
	Limited quantity	5 kg
	Equipment required	PP, A***
	Fire cones number	0

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

ZINC OXIDE(1314-13-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

Europe ADN - European Agreement concerning the International Carriage of Dangerous Goods by Inland Waterways

Europe EC Inventory

Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD

Europe European Agreement concerning the International Carriage of Dangerous Goods by Road

Europe European Customs Inventory of Chemical Substances

European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI - Chemwatch Standard Format

European Union (EU) Transport of Dangerous Goods by Road - Dangerous Goods List

International Air Transport Association (IATA) Dangerous Goods Regulations

International Maritime Dangerous Goods Requirements (IMDG Code)

Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2019 (English)

United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

ALUMINIUM OXIDE(1344-28-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Europe EC Inventory

Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD

Europe European Customs Inventory of Chemical Substances

European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

UK Workplace Exposure Limits (WELs)

GRAPHITE, NATURAL(7782-42-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Europe EC Inventory

Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD

Europe European Customs Inventory of Chemical Substances

European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

CARBON BLACK(1333-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

Europe EC Inventory

Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD

Europe European Customs Inventory of Chemical Substances

European Chemical Agency (ECHA) Classification & Labelling Inventory - Chemwatch Harmonised classification

European List of Notified Chemical Substances - ELINCS - 6th publication - COM(2003) 642, 29.10.2003

European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

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

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

UK Workplace Exposure Limits (WELs)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2015/830; Regulation (EC) No 1272/2008 as updated through ATPs.

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

Continued...

8464 Static Dissipative, Anti-Corrosive Grease

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (aluminium oxide; carbon black; graphite, natural)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (graphite, natural)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Thailand - TECI	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	18/03/2020
Initial Date	12/02/2018

Full text Risk and Hazard codes

H351	Suspected of causing cancer.
H373	May cause damage to organs through prolonged or repeated exposure.
H400	Very toxic to aquatic life.

SDS Version Summary

Version	Issue Date	Sections Updated
6.8.1.1.1	20/06/2019	Classification, Environmental, Fire Fighter (fire/explosion hazard), Physical Properties, Storage (storage incompatibility)

Other information

Ingredients with multiple cas numbers

Name	CAS No
zinc oxide	1314-13-2, 175449-32-8
aluminium oxide	1344-28-1, 1011245-20-7, 1022097-81-9, 107462-07-7, 107874-14-6, 1097999-44-4, 1197416-35-5, 122784-35-4, 1234495-70-5, 1239586-42-5, 12522-88-2, 127361-04-0, 12737-16-5, 131689-14-0, 1346644-15-2, 135152-65-7, 1355357-83-3, 135667-70-8, 138361-58-7, 148619-39-0, 152743-26-5, 153858-98-1, 157516-29-5, 163581-50-8, 165390-91-0, 170448-81-4, 190401-78-6, 200295-99-4, 205316-36-5, 209552-43-2, 230616-05-4, 252756-35-7, 253606-46-1, 253606-47-2, 253606-45-0, 268724-08-9, 39354-49-9, 457654-46-5, 488831-46-5, 521982-71-8, 53809-96-4, 54352-04-4, 546141-61-1, 663170-52-3, 67853-35-4, 67894-14-8, 67894-42-2, 68189-68-4, 68389-42-4, 68389-43-5, 74871-10-6, 76363-81-0, 84149-21-3, 90669-62-8, 916225-60-0, 960377-08-6, 11092-32-3

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.

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average

PC – STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit,

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL: No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

8464 Static Dissipative, Anti-Corrosive Grease

OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index

Reason For Change

A-1.01 - Update to the emergency phone number information.