OneFortyOne MicroPro Treated Timber

OneFortyOne Wood Products

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5589-47**
Version No: **2.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **16/02/2023**
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L.GHS.AUS.EN.E

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

Product Identifier

Product name	OneFortyOne MicroPro Treated Timber
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**OneFortyOne Wood Products**
Address	Jubilee Hwy East Mount Gambier SA 5290 Australia
Telephone	+61 8 8721 2777
Fax	+61 8 8721 2858
Website	http://onefortyone.com/
Email	Nigel.Boyd@onefortyone.com

Emergency telephone number

Association / Organisation	**OneFortyOne Wood Products**
Emergency telephone numbers	+61 8 8721 2777 (Mon-Fri 9am to 5pm)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Not Applicable

Label elements

Hazard pictogram(s)	Not Applicable
Signal word	**Not Applicable**

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Page 1 continued...

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	>95	Timber (softwood), as
Not Available		wood dust softwood
12069-69-1	<2	copper carbonate basic
1309-37-1	<0.3	ferric oxide
51274-00-1	<0.15	ferric hydroxide
7632-00-0	<0.08	sodium nitrite
107534-96-3	<0.02	tebuconazole
1333-86-4	<0.015	carbon black
26530-20-1	<0.008	2-octyl-4-isothiazolin-3-one
55965-84-9	<0.0025	isothiazolinones, mixed
Not Available	<1	additives
Not Available	0.1-0.3	dispersants
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▸ Generally not applicable.
Skin Contact	Brush off dust. Seek medical attention in event if irritation If skin or hair contact occurs: ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If dust is inhaled, remove from contaminated area. ▸ Encourage patient to blow nose to ensure clear passage of breathing. ▸ If irritation or discomfort persists seek medical attention.
Ingestion	Not normally a hazard due to physical form of product. ▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
for copper intoxication:
- Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
- Administer egg white and other demulcents.
- Maintain electrolyte and fluid balances.
- Morphine or meperidine (Demerol) may be necessary for control of pain.
- If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
- Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
- If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.
- It is unlikely that methylene blue would be effective against the occassional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
- Institute measures for impending renal and hepatic failure.

[GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]
- A role for activated charcoals for emesis is, as yet, unproven.
- In severe poisoning CaNa2EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

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

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Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	‣ Use water to wet down wood dusts to reduce the dispersion of dust into the air. ‣ Remove burned or wet dust to open area, after fire is extinguished, as partially burnt or wet dust may spontaneously ignite. ‣ Rake out ashes. ‣ Self-contained breathing apparatus (SCBA) is recommended when fighting fire. Slight hazard when exposed to heat, flame and oxidisers.
Fire/Explosion Hazard	· Wood articles do not normally constitute an explosion hazard. · Wood dusts, however, may constitute an explosion risk where the mean particle size is less than 200 microns, and where as little as 10% of the mixture contains dust less than 80 microns in size. Only weak explosions are likely where the mean particle size exceeds 200 microns. Wood dust is considered to be explosive if ignition of part of a cloud of wood dust results in the propagation of flame through the rest of the cloud. The vigour of flame propagation will vary from dust cloud to dust cloud and not all flammable dusts are equally explosive. · The burning of an unconfined wood dust cloud produces a flash fire. However, if the wood dust is contained within a full or partial enclosure, the pressure build-up can produce a destructive explosion. Its severity will depend on the type and concentration of the dust, particle size distribution, moisture content, the size of the source of ignition and the strength of the enclosure. · Generally, the larger the volume of the exploding dust cloud, the more widespread its effects will be. It is important to ensure that wood dust does not escape from collection systems and be allowed to build up within workrooms. If dust does accumulate, any primary explosion which occurs in a collection unit may stir up dust deposits within the building which houses the plant. Burning particles from the primary explosion can ignite the dust cloud resulting from it, leading to a secondary explosion that is usually more destructive than the first. · Mechanical or abrasive activities which produce wood dust, as a by-product, may present a severe explosion hazard if a dust cloud contacts an ignition source. · Hot humid conditions may result in ***spontaneous combustion*** of accumulated wood dust. · Partially burned or scorched wood dust can explode if dispersed in air. · Wet dusts may ignite spontaneously. · Solid fuels, such as wood, when subjected to a sufficient heat flux, will degrade, gasify and release vapours. There is little or no oxidation involved in this gasification process and thus it is endothermic. This process is referred to as ***forced pyrolysis*** but is sometimes referred to, wrongly, as smoldering combustion. This type of combustion, once initiated, can continue in a low-oxygen environment, even when the fire is in a closed compartment with low oxygen content. · An airborne concentration of 40 grams of dust per cubic meter of air is frequently used as the lower explosive limit (L.E.L) of wood dusts. · Thermal oxidative decomposition may produce vapours and gases including carbon monoxide, aldehydes (including formaldehyde), organic acids, cyanides, polycyclic aromatics, and other volatile organic fragments. Common ignition sources include naked flames, faulty or unsuitable electrics and impact sparks. The sanding or hogging of off-cuts containing metal may produce friction sparks, which can cause sawdust to smoulder and subsequently be fanned into fires or explosions. Use dedicated collection systems for these operations. Consider spark detection and extinguishing devices where there are significant risks. For dry wood dusts: · Moisture Content : less than 5% · Particle size: less than 100 micron · Dust Explosion Class: Kst1 (some wood dust is KSt2) · Minimum Ignition Energy MIE 7-250 mJ · Minimum Cloud Ignition Temperature: 490 deg C. · Minimum Layer Ignition Temperature 310-320 deg C · Minimum Explosible Concentration MEC 40-60 g/m3 · Maximum Pressure Pmax: 9.2 barg (133 psig) · Deflagration Index Kst: 100-150 bar.m-sec Combustion products include: carbon dioxide (CO_2) silicon dioxide (SiO_2) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place. Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ Clean up all spills immediately. ‣ Secure load if safe to do so. ‣ Bundle/collect recoverable product. ‣ Collect remaining material in containers with covers for disposal.
Major Spills	‣ Clean up all spills immediately. ‣ Secure load if safe to do so. ‣ Bundle/collect recoverable product. ‣ Collect remaining material in containers with covers for disposal.

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

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SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	Controls to reduce exposure to dusts include: · Many hazards are associated with wood dust production. Dusts can cause a range of skin, eye, lung and other aliments and complaints. · All work should be carried out in such a way as to minimise the generation of dust. Generally, all dust needs to collected at the point of generation. · Machining should be done with equipment fitted with exhaust extraction. · Hand power tools should be fitted with dust bags and used in well-ventilated areas. · A vacuum cleaner with a high efficiency filter or wet mop should be used to clean work areas. A dry sweeping method should not be used. · Clean inside walls, ceilings, ledges and other surfaces of workrooms regularly to prevent dust accumulating. Use vacuum cleaning equipment with high efficiency filters. Do not use compressed airlines or hand brushing as these will create dust clouds and redistribute the dust. · Clean the workshop machines and tools regularly to prevent dust build-up. · Suspect that a health problem may be related to your workshop if the symptoms improve during holidays or absences from the workshop. · Exposure to wood dust has long been associated with a variety of adverse health effects, including dermatitis, allergic respiratory effects, mucosal and non-allergic respiratory effects, and cancer. · In general, exposure to excessive amounts is considered to have an irritant effect on eyes, nose and throat in addition to pulmonary function. Western red cedar dust has also been shown to cause asthma. · Many tropical timbers are spalted (i.e. black lines are present within the timber). These black lines are caused by fungus. Any timber with fungal spores will grow fungus in a bag. When this timber is worked (by hand or machine) the dust may be toxic. · Medium and high-density fibreboards (MDF) are made using up to 13% formaldehyde resin. Formaldehyde is classified as a probable human carcinogen and may be released during machining. The softwood dust from this product is a sensitiser and may cause allergic dermatitis or asthma.
Other information	‣ Store away from incompatible materials.

Conditions for safe storage, including any incompatibilities

Suitable container	Usually stored in bulk. Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler.
Storage incompatibility	‣ Avoid strong acids, bases. ‣ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	wood dust softwood	Wood dust (soft wood)	5 mg/m3	10 mg/m3	Not Available	Not Available
Australia Exposure Standards	ferric oxide	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric oxide	Rouge dust	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ferric hydroxide	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric hydroxide	Rouge dust	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
copper carbonate basic	5.2 mg/m3	45 mg/m3	270 mg/m3
ferric oxide	15 mg/m3	360 mg/m3	2,200 mg/m3
ferric hydroxide	30 mg/m3	330 mg/m3	2,000 mg/m3
ferric hydroxide	15 mg/m3	360 mg/m3	2,200 mg/m3
ferric hydroxide	24 mg/m3	260 mg/m3	1,600 mg/m3
sodium nitrite	6.4 mg/m3	71 mg/m3	240 mg/m3
carbon black	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
wood dust softwood	Not Available	Not Available
copper carbonate basic	Not Available	Not Available
ferric oxide	2,500 mg/m3	Not Available
ferric hydroxide	2,500 mg/m3	Not Available
sodium nitrite	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
tebuconazole	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
2-octyl-4-isothiazolin-3-one	Not Available	Not Available
isothiazolinones, mixed	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
copper carbonate basic	E	≤ 0.01 mg/m³
sodium nitrite	E	≤ 0.01 mg/m³
tebuconazole	E	≤ 0.01 mg/m³
2-octyl-4-isothiazolin-3-one	E	≤ 0.1 ppm
isothiazolinones, mixed	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

WARNING: Wood dusts have been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS.
Wood dusts produce dermatitis and an increased risk of upper respiratory disease. Epidemiological studies in furniture workers show an increased risk of lung, tongue, pharynx and nasal cancer. An excess risk of leukaemia amongst millwrights probably is associated with exposure to various components used in wood preservation.
IARC has not limited this finding to any specific type of industry (e.g. furniture manufacturing) or wood dust source (hardwood vs. softwood). IARC s conclusions are based primarily on human carcinogenicity data from studies of various exposed worker populations.
The softwood TLV-TWA reflects the apparent low risk for upper respiratory tract involvement amongst workers in the building industry. A separate TLV-TWA, for hard woods, is based on impaired nasal mucociliary function reported to contribute to nasal adenocarcinoma and related hyperplasia found in furniture workers.
Allergic reactions are more common from handling green timber, less common for dried hardwood.
Impairment of nasal mucociliary function may occur below 5 mg/m3 and may be important in the development of nasal adenocarcinoma amongst furniture workers exposed to hardwoods.
Certain exotic hardwoods contain alkaloids which may produce headache, anorexia, nausea, bradycardia and dyspnoea.
ACGIH Exposure Standards for Wood dusts

Species	ACGIH TLV TWA (inhalable fraction)	Notations	TLV Basis
Western red cedar (WRC)	0.5 mg/m3	Sensitiser, A4***	May produce asthma
Oak and beech	1 mg/m3	A1*	May affect pulmonary function
Birch, mahogany, teak, walnut	1 mg/m3	A2*	May affect pulmonary function
All other species	1 mg/m3	A4***	May affect pulmonary function

A1: Confirmed Human Carcinogen *
A2: Suspected Human Carcinogen **
A3 Confirmed Animal Carcinogen
A4 Not Classifiable as a Human Carcinogen ***
A5 Not Suspected as a Human Carcinogen
Australian Exposure Standard: ES: 1 mg/m3 (certain hardwoods as beech and oak)
The majority of the wood-dust mass was reported to be contributed by particles larger than 10 um in aerodynamic diameter; however, between 61% and 65% of the particles by count measured between 1 and 5 um in diameter.
Wood-dust concentrations vary with type of dust extraction, amount of wood removed, and type of sander For electric belt sanders used to sand dowels, total dust concentrations ranged from 0.22 mg/m3 with external dust extraction to 3.74 mg/m3 without extraction, and concentrations of respirable dust ranged from 0.003 mg/m3 with extraction to 0.936 mg/m3 without extraction. Rotary sanders tested with flat wood samples produced total dust concentrations ranging from 0.002 mg/m3 with extraction to 0.699 mg/m3 without extraction; concentrations of respirable dust ranged from 0.001 mg/m3 with extraction to 0.088 mg/m3 without extraction. Comparable decreases in dust concentration were observed when dust extraction was used with electrical orbital sanders.

Exposure controls

Appropriate engineering controls	Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use. Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment. For wood dusts: · Significant accumulations of fine particles of wood dust can also be a fire and explosion hazard in the workplace.Check that the design and installation of dust control equipment incorporates explosion precautions.In particular look at the location of collection equipment and the need for enclosure and/or explosion relief. · Keep floors free and clear from wood chips and dust.Pay particular attention to areas around machines and on or near heating units. · The sanding or hogging of off-cuts containing metal may produce friction sparks, which can cause sawdust to smoulder and subsequently be fanned into fires or explosions. Use dedicated collection systems for these operations. Consider spark detection and extinguishing devices where there are significant risks. · Hot work involving the careless use of welding or flame-cutting equipment has resulted in many incidents. To prevent this, plant should be isolated and thoroughly cleaned before work starts. Use cold cutting methods whenever possible. · Electrical equipment should be sited away from dusty areas. If this is not practicable, ensure it is adequately protected. There are three main types of system for collecting wood waste and as a result reduce the possibility of worker exposure and possible dust explosions. · One or more woodworking machines are exhaust ventilated to a nearby collection unit within the workshop which does not form part of any other exhaust ventilation system. · Many (perhaps all) of the woodworking machines are ventilated to a collection unit, which can be some distance from the machines and may be inside or outside the workshop. · One or more woodworking machines are exhaust ventilated to a nearby collection unit. These units deliver the wood waste into a larger collection unit, usually outside the workshop. This is known as a 'through flow system. Collection units should normally be sited outside, away from areas where there may be people. If units have to be indoors, precautions will depend on the size of the collector; the size and construction of the room it is in; the number of people nearby; and how near they are to walkways and combustible materials. To avoid the risk from secondary explosion or fire, it is essential to enforce good housekeeping practices to prevent the accumulation of wood dust within the building, eg a formal cleaning regime using appropriate vacuums fitted with HEPA-type filters. **For unenclosed sock collectors (<0.5 m3/s capacity)** These would quickly disintegrate if the contents were ignited, but would not produce high explosion pressures or widespread effects. Fire risks may exist so, if unenclosed, do not position them within 3 m of workers, combustible materials or walkways. Alternatively, provide a suitable baffle

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	or deflector plate or enclosure. **For unenclosed sock collectors (0.5–2.5 m3/s capacity)** Ignition of wood dust can lead to a jet of flame at head height, but an explosion is not likely. Where such collectors must remain within the workroom, provide one of the following precautions: Total enclosure within a strong metal cabinet with either an air outlet large enough in area to act as explosion relief or explosion vents. Outlets or vents should preferably discharge to a safe place outside the workroom or, if inside, discharge at least above head height. A baffle or deflector plate made of non-combustible material to direct flames or burning material to a safe place. Ensure the fan can be turned off from a safe place if a fire starts in the filter. A 3 m separation between the filter and regularly occupied locations is likely to be adequate to protect employees. **For unenclosed sock collectors (>2.5 m3/s capacity)** Site these outside or enclose them in a strong cabinet fitted with explosion vents that discharge to a safe place. **For enclosed sock or fabric filter collectors (<0.5 m3/s capacity)** The top of the enclosure may be open as long as it discharges to a safe place, eg above head height. **For enclosed sock or fabric filter collectors (0.5–2.5 m3/s)** Total enclosure within a strong metal cabinet with either an air outlet large enough in area to act as explosion relief or explosion vents. Outlets or vents should preferably discharge to a safe place outside the workroom or, if inside, discharge at least above head height. **For enclosed sock or fabric filter collectors (>2.5 m3/s)** The enclosure should be strong with explosion vents that discharge to a safe place. **Cyclones** Well-made cyclones of less than 0.5 m3/s volume (rare in woodworking) do not usually require explosion relief panels. Larger low-efficiency cyclones usually have large enough air outlets to act as an explosion vent, but the need for additional explosion venting should be assessed. Larger high-efficiency cyclones do not usually have large enough air outlets to act as effective explosion vents and so additional venting will be necessary. Where cyclone air outlets discharge to an after filter, both the cyclone and the after filter will need explosion-relief panels. **Bins or hoppers** Where used to store explosible wood waste, these will require explosion relief appropriate to their volume. They should preferably be outdoors but, if indoors, additional explosion relief may be required on the building itself. There should also be a safe system of work for emptying bins and hoppers. **Interconnected plant** Take precautions to prevent an explosion spreading between interconnected units of plant, such as collectors, cyclones, filters and incinerators. Collectors should discharge their collected wood waste through an explosion choke, eg a rotary valve, or directly into strong metal containers clamped firmly to the discharge outlets. Where rotary valves are intended to act as explosion chokes, they must be certified as explosion protection devices.
Individual protection measures, such as personal protective equipment	
Eye and face protection	When sawing, machining or sanding use: ▸ Safety glasses with side shields. ▸ 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	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. Wear general protective gloves, eg. light weight rubber gloves.
Body protection	See Other protection below
Other protection	· Always wear protective clothing, including shirts with long sleeves and high collars, long trousers, shoes or boots. · Provide vacuum cleaning equipment to remove dust from clothing, where this is a problem. Prevent the use of compressed airlines for this purpose. · Use barrier creams (silicone-free and fatty) before, during and after work. · Always wash hands prior to going to the toilet since some wood dust may irritate the genitals and anus. · Always wash hands prior to eating.

Respiratory protection

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

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

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

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

Respiratory protection not normally required due to the physical form of the product.
Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

	Disposable respirator	Re-usable respirator	Powered respirator

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All woodworking operations eg use of routers, lathes, planers, saws and vertical spindle moulders (VSMs)	Type P2 filter for low residual dust levels for lower risk woods such as pine Type P3 filter for higher residual dust levels such as when sanding (hand , disc, bobbin, pad etc.). Also for all work involving more toxic woods such as hard woods, Western red cedar and MDF	Type P2 filter fitted to either a half mask or full face mask of Class 1 or 2 Type P3 filter fitted to either a half mask or full face mask of Class 2 Note: A combined organic vapour filter Type A (organic), either Class 1 or 2, will provide protection against any formadehyde vapours present from MDF	Lightweight powered hood visor or helmet of Type TH1 equivalent protection to Type P2 filter Lightweight powered visor or helmet with Type TH2 equivalent to Type P3 filter
Changing dust collection bags on simple recirculating dust collectors in the workroom	Type P3 Filter	Type P3 filter fitted to either a half mask or full face mask of Class 2	Lightweight powered visor or helmet of Type TH2 equivalent to Type P3 filter
Entry into dust collection rooms/ vaults Entry into very dusty filter galleries for bag changing Work inside heavily contaminated ducts Ensure none of these are confined spaces (oxygen deficient atmosphere)	Disposable respirators not suitable	Type P3 filter fitted to full face mask of Class 2	Lightweight powered hood, visor or helmet of Type TH2 equivalent to Type P3 filter

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Solid, red coloured wood; insoluble in water. Red

Physical state	Manufactured	**Relative density (Water = 1)**	Not Applicable
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Applicable	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Applicable	**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 Applicable
Vapour density (Air = 1)	Not Applicable	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	Product is considered stable and hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Not normally a hazard due to physical form of product. The dust may be discomforting Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Wood dust may cause nasal dryness, irritation and obstruction of the respiratory system, coughing, wheezing, and sneezing. Inhalation of hardwood dusts may decrease the ability of the nose to clear particles, causing any wood dust in the nose to remain longer in the nasal cavity. Both the type of wood what is being done to the wood to generate the wood dust have a big impact on the dust s hazards. For instance, asthma cases have been reported for workers using western red cedar, and pneumonitis has been associated with redwood dust. Some effects associated with wood dust are thought to be due to molds, bacteria, or pesticides present on the wood or to other materials used during certain woodworking activities (e.g. formaldehyde).

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Ingestion	Not normally a hazard due to physical form of product. The dust may be discomforting Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Not normally a hazard due to physical form of product. The dust may be discomforting Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Eye	Not normally a hazard due to physical form of product. The dust may be discomforting Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	It should be noted that the effects from exposure to this product will depend on several factors including: frequency and duration of use; quantity used; effectiveness of control measures; protective equipment used and method of application. Given that it is impractical to prepare a report which would encompass all possible scenarios, it is anticipated that users will assess the risks and apply control methods where appropriate. [Manufacturer] Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. For copper and its compounds (typically copper chloride): Acute toxicity: There are no reliable acute oral toxicity results available. Animal testing shows that skin in exposure to copper may lead to hardness of the skin, scar formation, exudation and reddish changes. Inflammation, irritation and injury of the skin were noted. Repeat dose toxicity: Animal testing shows that very high levels of copper monochloride may cause anaemia. Genetic toxicity: Copper monochloride does not appear to cause mutations in vivo, although chromosomal aberrations were seen at very high concentrations in vitro. Cancer-causing potential: There was insufficient information to evaluate the cancer-causing activity of copper monochloride. Common chronic responses to wood dust exposures are dermatitis, simple bronchitis and non asthmatic chronic airflow obstruction. Wood is an organic substrate for growth of micro-organisms and fungal spores, these readily become airborne with wood dust and have caused a variety of respiratory infections Various woods, mainly tropical varieties, are able to induce allergies in joiners, carpenters, cabinet makers and model-makers. Allergies of the immediate type (rhino conjunctivitis, bronchial asthma, urticaria), caused by contact with dusts produced during wood-working and those of a delayed type (contact eczema) caused by both the dust and by direct contact with the solid wood, are seen in an occupational setting. Because of the large number of substances found in wood, only a few low molecular weight allergens have been isolated and identified; these are mostly quinone or flavone derivatives. Many of the constituents of wood may also cause primary irritation. Irritation of the skin, eyes and respiratory passages are often distinguished from allergic responses with difficulty. The use of skin tests with wood dusts to confirm suspected allergy must be viewed as suspect because the high concentration of wood components which are sometimes applied, can actually produce new sensitisation in test subjects. It should also be noted that cross-reactions or reactions to groups of similar substances, in other woods and also in other herbaceous plants can also occur. The substances in wood responsible for respiratory allergies are probably mostly high molecular weight substances. Wood dusts may induce asthmatic reactions of both the immediate and delayed types, and occasionally, both. Positive results in bronchial provocation tests, are often, but not always, associated with positive results in skin tests and IgE induction. Bronchial provocation tests may produce different results dependent on whether they are carried out with course or fine dusts or with lyophilised aqueous extracts. Very course dust may produce false negatives and very fine dust may produce false positives (irritation). Non-allergenic bronchial and nasal irritation are seen frequently. Certain exotic woods contain alkaloids which may produce headache, anorexia, nausea, bradycardia and dyspnea. Agents used to treat wood (preservatives, fungicides, stains, glues, pore fillers) may themselves be responsible for allergic reaction. Other allergic reactions may be provoked by liverworts ("Frullania dermatitis"), lichens, fungi (e.g. bronchopulmonary aspergillosis), actinomycetes or other plants which grow on wood. Microorganisms and fungal spores, associated with wood, may become airborne and provoke allergic responses. Other chronic responses associated with exposure to wood dusts include conjunctivitis, simple bronchitis and non-asthmatic chronic airflow obstruction. Epidemiologic studies in furniture workers show an increased risk of lung, tongue, pharynx and nasal cancer (adenocarcinoma). Workers in timber industries, with a history of exposure to wood dust, have shown increased occurrence of lung, liver and vocal cavity cancer. An excess risk of leukaemia amongst mill-wrights probably is associated with various components used in wood preservation. It is now suggested that sinonasal cancers may be caused by both hardwoods and softwoods (1). The causative agent or agents are unknown although certain aldehydes or their quinone oxidation products have been implicated. Exposure standards for the softwoods reflect the apparent low risk for upper respiratory tract involvement among workers in the building industry. A significantly lower exposure standard for hardwoods is based on impaired nasal mucociliary hyperplasia reported to contribute to nasal adenocarcinoma and related hyperplasia in furniture workers. Exposure standards for both hard and softwoods specifically exclude the issue of occupational asthma and related allergic respiratory response associated with exposure to red cedar dusts and similar woods. The main components of wood are polysaccharides: cellulose (40-50 wt%) and hemicelluloses (20–35%), while lignin comprises 15–30% of wood mass.3 In addition to these macromolecules, wood contains a small amount of inorganic residues and extractives, which are low molar mass molecules. Extractives include a heterogeneous group of aliphatic and cyclic compounds: terpenes and terpenoids, esters of fatty acids, fatty acids, alcohols, alkanes, simple phenols, stilbenes, lignans, isoflavones, condensed tannins, flavonoids and hydrolyzable tannins. Wood phenolic compounds may possess bioactive functions; in vitro studies suggest that they may act as antioxidants. Due to the close association of lignin and extractives with cellulose and hemicelluloses, low amounts of these compounds commonly exist in hemicellulose or cellulose extracts and can, thus, be considered as “co-passengers” of fibrous materials. While wood extracts are neither presently nor extensively used in food ingredients, they have a long history in food supplement use. Softwood extracts have also received attention in the biomedical field; spruce

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	hemicellulose extract was patented for “use on the treatment of lower urinary tract symptoms and diseases”. The presence of mycotoxins is unlikely given the production procedure (particularly as there was no significant delay between grinding and extraction). The possibility of fungal contamination on the tree stumps is also unlikely since, firstly, these stumps come from felled wood which is therefore healthy, and secondly, if a fungal contamination were to appear (in the event that the stumps were not collected quickly after the trees were felled), this would essentially be an external contamination which would be eliminated when the stumps were examined before the grinding process. Radionuclide monitoring checks should be carried out systematically for all batches.

OneFortyOne MicroPro Treated Timber	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
wood dust softwood	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
copper carbonate basic	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (Rabbit) LD50; 159 mg/kg[2]	
ferric oxide	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >5000 mg/kg[1]	Not Available
ferric hydroxide	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >10000 mg/kg[2]	Not Available
sodium nitrite	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50: 0.006 mg/L4h[2]	Eye (rabbit): 500 mg/24hr - mild
	Oral (Rat) LD50: 180 mg/kg[2]	
tebuconazole	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >5000 mg/kg[2]	Non-irritating to eyes, skin. *
	Inhalation(Rat) LC50: >0.8 mg/L4h[2]	
	Oral (Mouse) LD50; 2000 mg/kg[2]	
carbon black	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
2-octyl-4-isothiazolin-3-one	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 311 mg/kg[2]	Eye (rabbit): 0.5% non irritant
	Oral (Rat) LD50: 248 mg/kg[2]	Eye (rabbit): 45% conc CORROSIVE
		Eye (rabbit): 5% conc moderate
		Eye(rabbit):100 mg SEVERE
		Eye: adverse effect observed (irreversible damage)[1]
		Skin (rabbit): 45% conc SEVERE
		Skin (rabbit): 500 mg/24 hours
		Skin: adverse effect observed (corrosive)[1]
		Skin: adverse effect observed (irritating)[1]
isothiazolinones, mixed	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >1008 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Inhalation(Rat) LC50: 0.171 mg/l4h[1]	Skin: adverse effect observed (corrosive)[1]
	Oral (Rat) LD50: 53 mg/kg[2]	Skin: adverse effect observed (irritating)[1]
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*	

WOOD DUST SOFTWOOD	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T

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lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
For wood dusts:
Wood dusts may cause respiratory symptoms including sensitisation and diminished respiratory function and may also be carcinogenic.
OSHA has determined that the health evidence for the toxicity of wood dust cannot be separately distinguished for soft wood and hard wood. A final OSHA ruling however establishes an 8-hour TWA PEL of 2.5 mg/m3 for Western red cedar wood dust, based on its widely recognized ability to cause immune-system-mediated allergic sensitization. Evidence in the record demonstrates the seriousness of this effect.
Wood dust is defined as any wood particles arising from the processing or handling of woods. Hard woods derive from the deciduous broad-leaved flowering species of trees, and soft woods include the coniferous species that do not shed their leaves in the winter. The distinction between hard woods and soft woods is purely botanical. Many so-called "softwoods" are actually hard (i.e., Douglas fir as a softwood is harder than the hardwood birch) and one of the softest woods in existence (balsa) is botanically a hardwood.
Some commentators were of the opinion that many other woods, such as Douglas fir, pine, red and white oak, redwood, walnut, spruce, boxwood, cocobolo, teak, mahogany, and others, should also be designated by OSHA as allergenic in this rulemaking. However, OSHA finds that "it is unlikely that species other than WRC are responsible for large numbers of cases of respiratory allergies".
Other commonly used woods such as oak, birch, redwood, pine, teak, alder, and hemlock, produce pulmonary effects that are less well described than the asthma responses to Western red cedar.
OSHA is establishing a PEL of 5 mg/m3 as an 8-hour TWA and 10 mg/m3 as a 15-minute STEL for hard and soft wood dust, with the exception of Western red cedar. OSHA concludes that promulgation of these exposure limits will substantially reduce the significant risk of material impairment in the form of pulmonary dysfunction (including changes in peak flow, interference with mucociliary clearance, respiratory symptoms, and chronic effects) that is associated with exposure to wood dust at the higher levels that would be permitted in the absence of any limit.
Carcinogenicity The association between occupational exposure to wood dust and various forms of cancer has been explored in many studies and in many countries. In 1987, the International Agency for Research on Cancer (IARC) classified furniture manufacturing in Category I (confirmed human carcinogen) and carpentry in Category 2B (suspected human carcinogen). IARC concludes that there is sufficient evidence in humans for the carcinogenicity of wood dust. (Group 1) Wood dust causes cancer of the nasal cavity and paranasal sinuses and of the nasopharynx. IARC also concludes that there is inadequate evidence in experimental animals for the carcinogenicity of wood dust.
In 1998, IARC issued the results of its detailed analyses of the combined results from 17 studies of nasal cancers and wood dust exposures. These analyses supported IARC s earlier conclusions and led to the following findings:
• Excess sino-nasal cancers were seen primarily in studies of European furniture makers
• The degree of risk was increased in workers with the highest level and length of exposure
• Observed risk levels were lower in studies of U.S. populations, possibly due to differences in the types of exposures that had occurred (e.g., exposures to different types of wood).
Based on its analyses, IARC has concluded that wood dust may cause “adenocarcinomas of the nasal cavities and paranasal sinuses”. This is a specific type of cancer in a specific region in the respiratory tract. IARC did not find sufficient evidence to associate wood dust exposure with other types of cancer of the nasal cavities (e.g., squamous cell carcinomas) or cancers in other parts of the body, such as the oropharynyx, hypopharynx, lung, lymphatic and haematopoietic systems, stomach, colon or rectum.
Dust particles may act as carriers for genotoxic agents. Chromium compounds are often present in oak and beech dusts as they are frequently used in the wood-processing industry, particularly as potassium dichromate in stains as well as fixing agents in wood preservatives. Stained furniture is made largely from oak and beech as they contain enough tannic acid to allow for chemical staining Direct genotoxic effects of wood dust extracts were summarized by IARC (1995).
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Direct genotoxic effects of wood dust extracts were summarized by IARC (1995). Dust particles may act as carriers for genotoxic agents. Chromium compounds are often present in oak and beech dusts as they are frequently used in the wood-processing industry, particularly as potassium dichromate in stains as well as fixing agents in wood preservatives.
Exposure to hexavalent chromium has been associated with the development of sinonasal cancers.
NIOSH (Ex. 8-47) considers both hard and soft wood dust to be potentially carcinogenic in humans; for soft wood dust, NIOSH recommends a separate 6(b) rulemaking (Ex. 8-47, Table N6B). NIOSH concurred, however, with the proposed PEL of 1 mg/m3 TWA for hard wood dust.
Several chemicals were isolated from wood extracts, but only quercetin and delta-3 -carene were shown to be mutagenic (IARC, 1995)
Summary of evidence for nasal and sinus cavity cancers. NIOSH (1987a/Ex. 1-1005) concluded that the literature clearly demonstrates an association between occupational wood dust exposure and nasal cancer. English studies first identified this link by showing a 10- to 20-times-greater incidence of nasal adenocarcinoma among woodworkers in the furniture industry than among other woodworkers and 100 times greater than in the general population. In the United States, three studies have reported a fourfold risk of nasal cancer or adenocarcinoma in furniture workers, and another study noted a similar relationship between nasal cancer and wood dust exposure. One other study failed to find such an association for furniture workers, but did find an increase among logging and timber industry workers.
The association between lung cancer and occupational wood dust exposure is inconclusive, although several epidemiological studies have reported increases in lung cancer among wood-dust-exposed workers. A significant excess of malignant tumours of the bronchus and lung in carpenters and joiners. Only construction workers showed a statistically significant increase in lung cancer rate.
Although the data are conflicting, several epidemiological studies of U.S. workers do report increases in the incidence of Hodgkin's disease among woodworkers. This excess is particularly apparent among carpenters.
Data on the relationship between occupational exposure to wood dust and the development of cancers other than nasal, Hodgkin's disease, or lung cancers are insufficient and inconclusive.
Copper chrome arsenic (CCA) is used widely to treat timber in both industrial and domestic situations. CCA is a water-borne preservative and contains copper, chromium and arsenic salts dissolved in water. Exposure to CCA is considered a potential health risk mainly because some arsenic and chromium compounds are known to cause cancer. It is recommended practice that freshly treated timber is stored at the treatment plant for at least two weeks (and up to 6 weeks) to ensure fixation and surface drying of the CCA. Timber for domestic or playground use should also be surface washed prior to distribution.
Exposure to wood dust has long been associated with a variety of adverse health effects, including dermatitis, allergic respiratory effects, mucosal and non-allergic respiratory effects, and cancer. The toxicity data in animals are limited, particularly with regard to exposure to wood dust alone; there are, however, a large number of studies in humans. There are a large number of case reports, epidemiological studies, and other data on the health effects of wood dust exposure in humans. Dermatitis caused by exposure to wood dusts is common, and can be caused either by chemical irritation, sensitization (allergic reaction), or both of these together. As many as 300 species of trees have been implicated in wood-caused dermatitis.
Allergic respiratory responses are mediated by the immune system, as is also the case with allergic dermatitis. Asthma is the most common response to wood dust exposure, and the allergic nature of such reactions has been demonstrated by the presence of IgE antibodies and positive skin reactions on patch testing. The best-studied of the allergic reactions to wood dust is Western red cedar (WRC) asthma; it is estimated that 5 percent of the workers handling this species are allergic to it.
The symptoms of sensitization are redness, scaling, and itching, which may progress to vesicular dermatitis and, after repeated exposures, to chronic dermatitis. The parts of the body most often affected are the hands, forearms, eyelids, face, neck, and genitals. This form of dermatitis generally appears after a few days or weeks of contact.
The chemicals associated with allergic reactions are generally found in the inner parts of a tree, e.g., the heartwood, and the workers most prone to these reactions are those involved in secondary wood processing (e.g., carpenters, joiners, and finishers).
Cereal flours are used in the wood industry to improve the quality of the glues necessary to produce veneer panels and are a potential source of sensitising substances. Cereal alpha-amylase inhibitors have been previously described as important occupational allergens responsible for baker's asthma. IgE proteins belong to the cereal alpha-amylase inhibitor family have been identified in the sera of several wood workers.
Exposure to microorganisms that grow on wood can also cause potential health effects. Endotoxins from bacteria and allergenic fungi growing on wood are the main biohazards found in wood processing workplaces. Exposure to these biohazards can cause adverse health effects such as organic dust toxic syndrome (ODTS), bronchitis, asthma, extrinsic allergic alveolitis (EAA), and mucous membrane irritation. The fungi predominantly associated with EAA and ODTS are dry spored species such as Aspergillus and Penicillium.

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	A large number of studies have demonstrated that occupational exposure to wood dust causes both statistically significant and non-significant increases in respiratory symptoms at exposure levels as low as 2 mg/m3. These symptoms range from irritation to bleeding, wheezing, sinusitis, and prolonged colds. In addition, chronic wood dust exposure causes mucociliary stasis (i.e., the absence of effective clearance) in the nose and, in some workers, also causes changes in the nasal mucosa. Several studies have demonstrated decreased pulmonary function among wood-dust-exposed workers, although other studies have not confirmed these findings. One study relates exposure level to ventilatory function. In that study, exposure to concentrations of 2 mg/m3 of WRC dust caused significant decreases in forced vital capacity and forced expiratory volume. Exposures to concentrations above 3 mg/m3 produced eye irritation. Mucosal and non-allergic respiratory effects have also been demonstrated. These changes include nasal dryness, irritation, bleeding, and obstruction; coughing, wheezing, and sneezing; sinusitis; and prolonged colds. These symptoms have been observed even at wood dust concentrations below 4 mg/m3. Workers (carpenters, sawmill workers, woodworkers) exposed from 3 to 24 years to the dust of several different hard woods showed radiologic evidence of pulmonary abnormalities. In all of these workers, mucociliary movement was markedly depressed, leading these authors to conclude that exposure to wood dust in the furniture industry for 10 years or more can impair mucociliary clearance. A respiratory survey in pulp and paper mill workers showed that workers exposed to wood dust at a mean total dust concentration of 0.5 mg/m3 had a slight but statistically significant decrease in pulmonary function values compared with controls. The authors concluded that the chemical preservatives used to treat the wood could also have been responsible for these adverse effects. A further study found that exposure to higher (10+ mg-years/m3), as compared with lower (0 to 2 mg-years/m3), dust concentrations was associated with a statistically significant and higher incidence of decreased pulmonary function. However, dose-response effects were observed only for soft wood (i.e., pine) dusts. Yet another study found no correlation between years of exposure to pine wood dust and pulmonary function. A study of Italian woodworkers showed that the number of wood-dust-exposed workers who had developed anosmia (loss of smell) was significantly higher than in a control group of non-exposed workers. This confirmed was confirmed in other workers exposed to hardwood dusts. Exposure to wood dust can cause chronic obstructive lung disease. Exposure to saw fumes containing terpenes, which is a constituent of wood, also causes chronic obstructive impairment in lung function. Medium density fibre boards (MDF) is widely used in the joinery and furniture industry as well as in building and housing construction. The major constituents of MDF particle boards are pulverised softwood and urea-formaldehyde resin, both of which are recognised as potential health hazards in the working environment. MDF produces very fine dust during processing and the dust particles act as a carrier of absorbed formaldehyde to the lower airways of the lungs. Wood dust and formaldehyde together have been reported to cause respiratory irritation with symptoms of dryness of the throat, rhinitis and eye irritation as well as occupational skin disease. Groups of male guinea pigs were injected intratracheally with suspensions containing 75 mg of sheesham or mango wood dust or of hemp or bagasse fibers, or 20 mg of jute fiber. Lung examination revealed that, at 90 days, Grade I fibrosis of the lungs had occurred in the guinea pigs injected with mango or jute, while those treated with sheesham or hemp had developed Grade II pulmonary fibrosis. In another experiment involving guinea pigs, animals were exposed by inhalation to average respirable dust concentrations of 1143 mg/m3 for 30 minutes/day, 5 days/week for 24 weeks. Histopathological examination showed lung changes, described as moderate to severe, in all exposed guinea pigs. The changes seen included an increase in septal connective tissue components and aggregation of lymphocytes; however, no pulmonary fibrosis or extensive destruction of the parenchymal tissue occurred. The study concluded that exposure to fir bark dust may cause inflammatory changes in the lung. Two studies examined the effect of exposing Syrian golden hamsters to beech wood dust by inhalation, with or without concurrent administration of the known carcinogen diethylnitrosamine (DEN). In Study I was given the DEN doses only (positive control), and the fourth group was given no exposure at all (negative control). Four hamsters exposed to wood dust and DEN exhibited squamous cell papillomas of the trachea, as did three animals in the positive control group and one in the negative control group. No differences in organs other than the respiratory organs were seen between the treated and control groups. In Study II, there were 24 animals in each of four groups. Two groups of animals were exposed to fresh beech wood dust at a mean total dust concentration of 30 mg/m3 for six hours/day, five days/week for 40 weeks. All DEN-exposed hamsters had nasal lesions ranging from hyperplasias and dysplasias to papillomas. In addition, half of all DEN-exposed hamsters developed nasal adenocarcinomas, whether or not they had also been exposed to wood dust. Half of the DEN-exposed animals also had papillomas of the larynx and trachea. In the wood-dust-exposure-only group, two of the animals had nasal lesions, one of which was an unclassifiable malignant nasal tumor and the other of which consisted of focal metaplasia with mild dysplasia. The study concluded that exposure to wood dust did not increase the tumour incidence in DEN-exposed animals but did affect the respiratory tract of all exposed animals. WARNING: Inhalation of wood dust by workers in the furniture and cabinet making industry has been related to nasal cancer [I.L.O. Encyclopedia] Use control measures to limit all exposures.
COPPER CARBONATE BASIC	for copper and its compounds (typically copper chloride): **Acute toxicity:** There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs. No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation. **Repeat dose toxicity:** In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride. **Genotoxicity:** An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen. **Carcinogenicity:** there was insufficient information to evaluate the carcinogenic activity of copper monochloride. Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).
SODIUM NITRITE	Tumorigenic - Carcinogenic by RTECS criteria. Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.

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TEBUCONAZOLE	(aerosol) NOEL (2 y)* for rats, 300 mg/kg diet for dogs, 100 mg/kg " for mice, 20 mg/kg " ADI 0.03 mg/kg b.w. * Toxicity Class WHO III; EPA III * ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]***
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
2-OCTYL-4-ISOTHIAZOLIN-3-ONE	ROHM & HAAS Data ADI: 0.03 mg/kg/day NOEL: 60 mg/kg/day
ISOTHIAZOLINONES, MIXED	In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance. Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration. The European Union has reclassified several formaldehyde-releasing agents (FRAs) such as methylenedimorpholine (MBM), oxazolidine (MBO) and hydroxypropylamine (HPT) as category 1B carcinogens. Previously, formaldehyde itself was classed as a carcinogen – but formaldehyde-releasing agents were not. This is no longer the case. Based on this regulation, formulations for which the maximum theoretical concentration of releasable formaldehyde is more than > 1000 ppm (>0.1%), have to be labelled as carcinogenic. Water mix metalworking fluids are subject to contamination by bacteria and fungi, and the control of this is an essential part of good fluid maintenance. The use of preservatives both within the formulation and tank-side treatment plays a significant contribution in the protection of potentially harmful microbes that could cause health problems for workers. A large proportion of bactericides on the market today are classed as formaldehyde releasing biocides which means that under specific conditions they release small amounts of formaldehyde – this is their mode of action in the presence of bacteria. Although they are effective as a biocide their use may become restricted or unfavourable due to potential changes in legislation. A decision by the ECHA (European Chemicals Agency) was made to re-classify formaldehyde as a category 1b H350 carcinogen and category 2 mutagen in June 2015. It has also been proposed by the ECHA Risk Assessment Committee (RAC) that formaldehyde release biocides should be classified the same as formaldehyde because formaldehyde is released when these substances come into contact under favorable conditions (i.e. interaction with microorganisms). Formaldehyde generators (releasers) are often used as preservatives (antimicrobials, biocides, microbiocides). Formaldehyde may be generated following hydrolysis. The most widely used antimicrobial compounds function by releasing formaldehyde once inside the microbe cell. Some release detectable levels of formaldehyde into the air space, above working solutions, especially when pH has dropped. Many countries are placing regulatory pressure on suppliers and users to replace formaldehyde generators. Formaldehyde generators are a diverse group of chemicals that can be recognised by a small, easily detachable formaldehyde moiety, prepared by reacting an amino alcohol with formaldehyde ("formaldehyde-condensates"), There is concern that when formaldehyde-releasing preservatives are present in a formulation that also includes amines, such as triethanolamine (TEA), diethanolamine (DEA), or monoethanolamine (MEA), nitrosamines can be formed,; nitrosamines are carcinogenic substances that can potentially penetrate skin. One widely-discussed hypothesis states that formaldehyde-condensate biocides, such as triazines and oxazolidines, may cause an imbalance in the microbial flora of in-use metalworking fluids (MWFs).The hypothesis further asserts that this putative microbial imbalance favours the proliferation of certain nontuberculosis mycobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, in a small percentage of susceptible workers. Symptoms of HP include flu-like illness accompanied by chronic dyspnea, i.e., difficult or laboured respiration According to Annex VI of the Cosmetic Directive 76/768/EC, the maximum authorised concentration of free formaldehyde is 0.2% (2000 ppm). In addition, the provisions of Annex VI state that, *All finished products containing formaldehyde or substances in this Annex and which release formaldehyde must be labelled with the warning "contains formaldehyde" where the concentration of formaldehyde in the finished product exceeds 0.05%.* Formaldehyde-releasing preservatives have the ability to release formaldehyde in very small amounts over time. The use of formaldehyde-releasing preservatives ensures that the actual level of free formaldehyde in the products is always very low but at the same time sufficient to ensure absence of microbial growth. The formaldehyde reacts most rapidly with organic and inorganic anions, amino and sulfide groups and electron-rich groups to disrupt metabolic processes, eventually causing death of the organism. 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.
WOOD DUST SOFTWOOD & FERRIC HYDROXIDE & CARBON BLACK & ISOTHIAZOLINONES, MIXED	No significant acute toxicological data identified in literature search.
COPPER CARBONATE BASIC & FERRIC OXIDE & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE & ISOTHIAZOLINONES, MIXED	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
SODIUM NITRITE & ISOTHIAZOLINONES, MIXED	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
2-OCTYL-4-ISOTHIAZOLIN-3-ONE & ISOTHIAZOLINONES, MIXED	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Acute Toxicity	✗	**Carcinogenicity**	✗

Continued...

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

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

SECTION 12 Ecological information

Toxicity

OneFortyOne MicroPro Treated Timber

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

wood dust softwood

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

copper carbonate basic

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	0.0028mg/l	2
EC50	72h	Algae or other aquatic plants	0.0165mg/l	2
EC50	48h	Crustacea	0.001mg/l	2
NOEC(ECx)	504h	Crustacea	<0.001mg/l	2
EC50	96h	Algae or other aquatic plants	0.047mg/l	2

ferric oxide

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

ferric hydroxide

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

sodium nitrite

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	672h	Fish	0.01mg/l	4
EC50	96h	Algae or other aquatic plants	1600mg/l	4
EC50	72h	Algae or other aquatic plants	>100mg/l	2
LC50	96h	Fish	0.00016mg/l	4
EC50	48h	Crustacea	ca.12.51mg/l	1

tebuconazole

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	6.4mg/l	Not Available
EC50	72h	Algae or other aquatic plants	2.09-3.01mg/l	4
EC50	48h	Crustacea	2.1-3.94mg/L	4
NOEC(ECx)	672h	Crustacea	0.000987mg/l	4
EC50	96h	Algae or other aquatic plants	1.45mg/L	4

carbon black

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
EC50	48h	Crustacea	33.076-41.968mg/l	4
NOEC(ECx)	24h	Crustacea	3200mg/l	1

Continued...

2-octyl-4-isothiazolin-3-one	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	840h	Fish	0.009mg/L	4
	EC50	96h	Algae or other aquatic plants	0.15mg/l	2
	LC50	96h	Fish	0.041-0.104mg/l	4
	EC50	48h	Crustacea	0.057-0.178mg/L	4
isothiazolinones, mixed	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	48h	Algae or other aquatic plants	0.00049mg/l	2
	EC50	72h	Algae or other aquatic plants	0.0063mg/l	2
	LC50	96h	Fish	0.129mg/l	2
	EC50	96h	Algae or other aquatic plants	0.0357mg/l	2
	EC50	48h	Crustacea	0.007mg/l	2
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

For copper:
Atmospheric Fate - Copper is unlikely to accumulate in the atmosphere due to a short residence time for airborne copper aerosols. Airborne coppers, however, may be transported over large distances. Air Quality Standards: no data available.
Aquatic Fate: Toxicity of copper is affected by pH and hardness of water. Total copper is rarely useful as a predictor of toxicity. In natural sea water, more than 98% of copper is organically bound and in river waters a high percentage is often organically bound, but the actual percentage depends on the river water and its pH.
Ecotoxicity: Copper accumulates significantly in the food chain. The toxic effect of copper in the aquatic biota depends on the bio-availability of copper in water which, in turn, depends on its physico-chemical form (i.e. speciation). Bioavailability is decreased by complexation and adsorption of copper by natural organic matter, iron and manganese hydrated oxides, and chelating agents excreted by algae and other aquatic organisms. Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper. Silicate, iron, manganese and EDTA may reduce bioavailability.
For copper: Ecotoxicity - Significant effects are expected on various species of microalgae, some species of macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Copper is moderately toxic to crab and their larvae and is highly toxic to gastropods (mollusks, including oysters, mussels and clams). In fish, the acute lethal concentrations of copper depends both on test species and exposure conditions. Waters with high concentrations of copper can have significant effects on diatoms and sensitive invertebrates, notably cladocerans (water fleas). Most taxonomic groups of macroalgae and invertebrates will be severely affected.
For Copper: Typical foliar levels of copper are: Uncontaminated soils (0.3-250 mg/kg) ; Contaminated soils (150-450 mg/kg) ; Mining/smelting soils (6.1-25 mg/kg80 mg/kg300 mg/kg).
Terrestrial Fate: Plants - Generally, vegetation reflects soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned. Crops are often more sensitive to copper than the native flora. Soil: In soil, copper levels are raised by application of fertilizer, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Chronic and or acute effects on sensitive species occur as a result of human activities such as copper fertilizer addition and addition of sludge. When soil levels exceed 150 mg Cu/kg, native and agricultural species show chronic effects. Soils in the range 500-1000 mg Cu/kg act in a strongly selective fashion allowing the survival of only copper-tolerant species and strains. At 2000 Cu mg/kg, most species cannot survive. By 3500 mg Cu/kg, areas are largely devoid of vegetation cover. The organic content of the soil appears to be a key factor affecting the bioavailability of copper. On normal forest soils, non-rooted plants such as mosses and lichens show higher copper concentrations. The fruiting bodies and mycorrhizal sheaths of soil fungi associated with higher plants in forests often accumulate copper to much higher levels than plants at the same site.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
sodium nitrite	LOW	LOW
tebuconazole	HIGH	HIGH
2-octyl-4-isothiazolin-3-one	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
sodium nitrite	LOW (LogKOW = 0.0564)
tebuconazole	HIGH (LogKOW = 5.4673)
2-octyl-4-isothiazolin-3-one	LOW (LogKOW = 2.561)

Mobility in soil

Ingredient	Mobility
sodium nitrite	LOW (KOC = 23.74)
tebuconazole	LOW (KOC = 20660)
2-octyl-4-isothiazolin-3-one	LOW (KOC = 2120)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. For wood wastes including wood dusts: Various public policies encourage the utilisation of waste wood for heat and energy production. Generation of heat using combustion technologies such as grate-fired boilers, fluidised bed combustion and cement kilns. Energy production technologies have been developed which are able to utilise mixed biomass to create energy. Common technologies include steam turbines, gasification and pyrolysis. The main issue preventing the utilisation of wood wastes is overcoming contamination, especially contamination by chemicals such as wood preservatives. However, technologies are being developed to overcome such issues, which may be viable for some of the larger industries wishing to use waste wood for manufacturing or energy production. When considering options for minimising waste, the waste hierarchy of “reduce, reuse, recycle” is a common feature across jurisdictions. The

Continued...

	hierarchy expresses a preference to achieve sustainable outcomes by reducing the amount of waste that is generated, reusing what cannot be reduced and recycling what cannot be reused, with disposal as the last option. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
wood dust softwood	Not Available
copper carbonate basic	Not Available
ferric oxide	Not Available
ferric hydroxide	Not Available
sodium nitrite	Not Available
tebuconazole	Not Available
carbon black	Not Available
2-octyl-4-isothiazolin-3-one	Not Available
isothiazolinones, mixed	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
wood dust softwood	Not Available
copper carbonate basic	Not Available
ferric oxide	Not Available
ferric hydroxide	Not Available
sodium nitrite	Not Available
tebuconazole	Not Available
carbon black	Not Available
2-octyl-4-isothiazolin-3-one	Not Available
isothiazolinones, mixed	Not Available

SECTION 15 Regulatory information

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

wood dust softwood is found on the following regulatory lists

Not Applicable

copper carbonate basic is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ferric oxide is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ferric hydroxide is found on the following regulatory lists

Continued...

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

sodium nitrite is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans

tebuconazole is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

carbon black is found on the following regulatory lists

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

2-octyl-4-isothiazolin-3-one is found on the following regulatory lists

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

isothiazolinones, mixed is found on the following regulatory lists

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (tebuconazole; isothiazolinones, mixed)
Canada - DSL	No (tebuconazole)
Canada - NDSL	No (copper carbonate basic; ferric oxide; sodium nitrite; tebuconazole; carbon black; 2-octyl-4-isothiazolin-3-one; isothiazolinones, mixed)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (isothiazolinones, mixed)
Japan - ENCS	No (isothiazolinones, mixed)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (tebuconazole; isothiazolinones, mixed)
Taiwan - TCSI	Yes
Mexico - INSQ	No (copper carbonate basic; isothiazolinones, mixed)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	16/02/2023
Initial Date	16/02/2023

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	16/02/2023	Physical and chemical properties - Appearance

Other information

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

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

Definitions and abbreviations

Continued...

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