



## OneFortyOne Kiln Dried Radiata Pine (Untreated)

### OneFortyOne Wood Products

Chemwatch: 5389-80  
Version No: 2.1.1.1  
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 1

Issue Date: 01/04/2020  
Print Date: 07/04/2020  
L.GHS.AUS.EN

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

### Product Identifier

Product name	OneFortyOne Kiln Dried Radiata Pine (Untreated)
Synonyms	Kiln Dried Timber, Lumber, Scantling
Other means of identification	Not Available

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

Relevant identified uses	Residential, commercial and industrial construction and/or general purpose building and packaging material. Use according to manufacturer's directions.
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### Details of the 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	<a href="http://onefortyone.com/">http://onefortyone.com/</a>
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	<b>NOT APPLICABLE</b>

### 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

Not Applicable

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**SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**

**Substances**

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
Not Available	100	Radiata Pine Kiln Dried Wood
Not Available		in use may generate
Not Available		<u>wood dust softwood</u>

**SECTION 4 FIRST AID MEASURES**

**Description of first aid measures**

<b>Eye Contact</b>	<ul style="list-style-type: none"> <li>Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations.</li> </ul> If this product comes in contact with eyes: <ul style="list-style-type: none"> <li>Wash out immediately with water.</li> <li>If irritation continues, seek medical attention.</li> <li>Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	Brush off dust. In the event of abrasion or irritation of the skin seek medical attention. If skin or hair contact occurs: <ul style="list-style-type: none"> <li>Flush skin and hair with running water (and soap if available).</li> <li>Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>If dust is inhaled, remove from contaminated area.</li> <li>Encourage patient to blow nose to ensure clear passage of breathing.</li> <li>If irritation or discomfort persists seek medical attention.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations.</li> <li>Immediately give a glass of water.</li> <li>First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.</li> </ul>

**Indication of any immediate medical attention and special treatment needed**

Treat symptomatically.

**SECTION 5 FIREFIGHTING MEASURES**

**Extinguishing media**

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

**Special hazards arising from the substrate or mixture**

<b>Fire Incompatibility</b>	Avoid exposure to excessive heat and fire.
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**Advice for firefighters**

<b>Fire Fighting</b>	Wear breathing apparatus plus protective gloves. Equipment should be thoroughly decontaminated after use. Alert Fire Brigade and tell them location and nature of hazard. Use water delivered as a fine spray to control the fire and cool adjacent area.
<b>Fire/Explosion Hazard</b>	- Wood products do not normally constitute an explosion hazard. - 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. Combustible. Will burn if ignited.
<b>HAZCHEM</b>	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**

<b>Minor Spills</b>	Pick up. Refer to major spills.
<b>Major Spills</b>	Pick up. Secure load if safe to do so. Bundle/collect recoverable product.

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

**SECTION 7 HANDLING AND STORAGE**

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Precautions for safe handling

Safe handling	Use gloves when handling product to avoid splinters.
Other information	▶ Keep dry

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Generally not applicable.
Storage incompatibility	▶ Keep dry

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

EMERGENCY LIMITS


Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
OneFortyOne Kiln Dried Radiata Pine (Untreated)	Not Available	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
wood dust softwood	Not Available	Not Available

MATERIAL DATA

for wood dust softwood: Australia Exposure Standards: ES TWA: 5 mg/m3; STEL: 10 mg/m3; Sensitiser

Exposure controls

Appropriate engineering controls	<p>▶ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations. Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air)</td> <td>0.25-0.5 m/s (50-100 f/min)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood - local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood - local control only
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Personal protection																					
Eye and face protection	When sawing, machining or sanding use - Safety glasses with side shields.																				

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<b>Skin protection</b>	See Hand protection below
<b>Hands/feet protection</b>	<ul style="list-style-type: none"> <li>▶ Protective gloves eg. Leather gloves or gloves with Leather facing</li> <li>▶ Safety footwear</li> </ul>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<p>No special equipment needed when handling small quantities.</p> <p><b>OTHERWISE:</b></p> <ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ Barrier cream.</li> <li>▶ Eyewash unit.</li> </ul>

### Respiratory protection

- ▶ Avoid generating and breathing dust.
- ▶ Effective dust extraction and good ventilation is required when using cutting, shaping or sanding tools. Wear a disposable dust mask AS/NZS 1715:2009 class P1 or P2 when machining.

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### Information on basic physical and chemical properties

<b>Appearance</b>	Dried Timber; insoluble in water. THIS REPORT IS FOR UNTREATED PRODUCT ONLY.		
<b>Physical state</b>	Manufactured	<b>Relative density (Water = 1)</b>	Not Available
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Applicable
<b>Initial boiling point and boiling range (°C)</b>	Not Applicable	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	Not Applicable	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Applicable	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Applicable	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Applicable
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Applicable
<b>Vapour pressure (kPa)</b>	Not Applicable	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Applicable
<b>Vapour density (Air = 1)</b>	Not Applicable	<b>VOC g/L</b>	Not Applicable

## SECTION 10 STABILITY AND REACTIVITY

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

## SECTION 11 TOXICOLOGICAL INFORMATION

### Information on toxicological effects

<b>Inhaled</b>	Not normally a hazard due to physical form of product. Generated dust may be discomforting
<b>Ingestion</b>	Ingestion of sawdust may cause nausea, abdominal pain, vomiting or diarrhoea. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments
<b>Skin Contact</b>	The dust is discomforting and mildly abrasive to the skin and may cause drying of the skin, which may lead to contact dermatitis.
<b>Eye</b>	The dust may produce eye discomfort causing transient smarting, blinking
<b>Chronic</b>	<p>Wood dust may cause skin and respiratory sensitisation.</p> <ul style="list-style-type: none"> <li>▶ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations.</li> </ul> <p>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</p>

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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 coarse or fine dusts or with lyophilised aqueous extracts. Very coarse 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.

<b>OneFortyOne Kiln Dried Radiata Pine (Untreated)</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<b>wood dust softwood</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<b>Legend:</b>		
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		

<b>WOOD DUST SOFTWOOD</b>	<p>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).</p> <p>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.</p> <p>Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.</p> <p>No significant acute toxicological data identified in literature search.</p> <p>For wood dusts:</p> <p>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/m<sup>3</sup> 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.</p> <p>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.</p> <p>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".</p> <p>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.</p> <p>OSHA is establishing a PEL of 5 mg/m<sup>3</sup> as an 8-hour TWA and 10 mg/m<sup>3</sup> 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.</p> <p><b>Carcinogenicity</b> 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 1 (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.</p> <p>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:</p> <ul style="list-style-type: none"> <li>• Excess sino-nasal cancers were seen primarily in studies of European furniture makers</li> <li>• The degree of risk was increased in workers with the highest level and length of exposure</li> <li>• 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).</li> </ul> <p>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 oropharynx, hypopharynx, lung, lymphatic and haematopoietic systems, stomach, colon or rectum.</p> <p>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</p>
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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). 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/m<sup>3</sup> 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 sensitizing 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*.

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/m<sup>3</sup>. 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/m<sup>3</sup> of WRC dust caused significant decreases in forced vital capacity and forced expiratory volume. Exposures to concentrations above 3 mg/m<sup>3</sup> 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/m<sup>3</sup>. 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/m<sup>3</sup> 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/m<sup>3</sup>), as compared with lower (0 to 2 mg-years/m<sup>3</sup>), 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/m<sup>3</sup> 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).



## OneFortyOne Kiln Dried Radiata Pine (Untreated)

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/m<sup>3</sup> 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.

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

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

## SECTION 12 ECOLOGICAL INFORMATION

## Toxicity

OneFortyOne Kiln Dried Radiata Pine (Untreated)	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

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

The solid wood will decay on ground contact.

## Persistence and degradability

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

## Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

## Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

## SECTION 13 DISPOSAL CONSIDERATIONS

## Waste treatment methods

Product / Packaging disposal	
	<ul style="list-style-type: none"> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Management Authority for disposal.</li> <li>▶ Bury residue in an authorised landfill.</li> </ul>

## SECTION 14 TRANSPORT INFORMATION

## Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

**SECTION 15 REGULATORY INFORMATION****Safety, health and environmental regulations / legislation specific for the substance or mixture****WOOD DUST SOFTWOOD IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Not Applicable

**National Inventory Status**

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	Yes
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

**SECTION 16 OTHER INFORMATION**

<b>Revision Date</b>	01/04/2020
<b>Initial Date</b>	01/04/2020

**SDS Version Summary**

Version	Issue Date	Sections Updated
2.1.1.1	01/04/2020	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Appearance, Chronic Health, Disposal, Engineering Control, Environmental, Exposure Standard, Fire Fighter (extinguishing media), Fire Fighter (fire/explosion hazard), Fire Fighter (fire fighting), Fire Fighter (fire incompatibility), First Aid (eye), First Aid (inhaled), First Aid (skin), First Aid (swallowed), Handling Procedure, Ingredients, Instability Condition, Personal Protection (other), Personal Protection (Respirator), Personal Protection (eye), Personal Protection (hands/feet), Spills (major), Spills (minor), Storage (storage incompatibility), Storage (storage requirement), Storage (suitable container), Transport

**Other information**

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

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

**Definitions and abbreviations**

PC—TWA: Permissible Concentration-Time Weighted Average  
 PC—STEL: Permissible Concentration-Short Term Exposure Limit  
 IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit.  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 OSF: Odour Safety Factor  
 NOAEL :No Observed Adverse Effect Level  
 LOAEL: Lowest Observed Adverse Effect Level  
 TLV: Threshold Limit Value  
 LOD: Limit Of Detection  
 OTV: Odour Threshold Value  
 BCF: BioConcentration Factors  
 BEI: Biological Exposure Index

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