

Trade Essentials - Whiteboard HMR

Laminex Group Pty Ltd

Chemwatch Hazard Alert Code: 1

Chemwatch: 4031521

Issue Date: 01/11/2019

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Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

L.GHS.AUS.EN

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

Product Identifier

Product name	Trade Essentials - Whiteboard HMR
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	Used for the construction of furniture and cabinets and / or general purpose building boards.
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Details of the supplier of the safety data sheet

Registered company name	Laminex Group Pty Ltd
Address	90-94 Tram Road Doncaster VIC 3108 Australia
Telephone	+61 3 9840 4347
Fax	+61 3 9840 6513
Website	www.laminex.com.au
Email	Sant.quaremba@laminex.com.au

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 2 9186 1132

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

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification ^[1]	Serious Eye Damage/Eye Irritation Category 2A, Skin Corrosion/Irritation Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI


Dust generated from shaping, cutting and sawing operations carried out on this product will contain cured binder/wood particles and may contain wood dust without binder.

Wood dust is a hazardous substance according to the NOHSC criteria.

and "may cause Sensitisation by inhalation and skin contact" (R42/43) and "may cause cancer by inhalation" (R49)

Label elements

Trade Essentials - Whiteboard HMR

Hazard pictogram(s)	
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Signal word	Warning
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Hazard statement(s)

H319	Causes serious eye irritation.
H315	Causes skin irritation.

Precautionary statement(s) Prevention

P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		wood panel containing
Not Available	>85	wood particles
Not Available		bonded together with
9011-05-6	<13	<u>urea/ formaldehyde resin</u>
25036-13-9	<13	<u>melamine/ urea/ formaldehyde resin</u>
8002-74-2	<2	<u>paraffin wax</u>
Not Available		dust from sawing and forming operations will contain
Not Available	NotSpec	<u>wood dust softwood</u>
Not Available	NotSpec	cured binder

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

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

- Water spray or fog.
- Alcohol stable foam.
- Dry chemical powder.
- Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ DO NOT approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<p>Combustible. Will burn if ignited. Combustion products include: carbon dioxide (CO₂) and minor amounts of hydrogen cyanide other pyrolysis products typical of burning organic material.</p>
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	<ul style="list-style-type: none"> ▸ 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	<ul style="list-style-type: none"> ▸ 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	<ul style="list-style-type: none"> ▶ Avoid generating and breathing dust ▶ Avoid contact with skin and eyes. ▶ Wear nominated personal protective equipment when handling. ▶ Use in a well-ventilated area. ▶ Use good occupational work practices. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
Other information	<ul style="list-style-type: none"> ▶ Store away from incompatible materials.

Conditions for safe storage, including any incompatibilities

Suitable container	No restriction on the type of containers. Packing as recommended by manufacturer. Check all material is clearly labelled.
Storage incompatibility	<ul style="list-style-type: none"> ▶ 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	paraffin wax	Paraffin wax (fume)	2 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	wood dust softwood	Wood dust (soft wood)	5 mg/m ³	10 mg/m ³	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
Trade Essentials - Whiteboard HMR	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
urea/ formaldehyde resin	Not Available	Not Available
melamine/ urea/ formaldehyde resin	Not Available	Not Available
paraffin wax	Not Available	Not Available
wood dust softwood	Not Available	Not Available

Occupational Exposure Banding


Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
urea/ formaldehyde resin	E	≤ 0.01 mg/m ³
melamine/ urea/ formaldehyde resin	D	> 0.01 to ≤ 0.1 mg/m ³

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

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <ul style="list-style-type: none"> Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. <ul style="list-style-type: none"> ▶ Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively
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	<p>large, a certain proportion will be powdered by mutual friction.</p> <ul style="list-style-type: none"> ▶ Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace. ▶ If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of: <ul style="list-style-type: none"> (a): particle dust respirators, if necessary, combined with an absorption cartridge; (b): filter respirators with absorption cartridge or canister of the right type; (c): fresh-air hoods or masks <ul style="list-style-type: none"> ▶ Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding. ▶ Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to efficiently remove the contaminant.</p> <table border="1" data-bbox="384 528 1485 696"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <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 ft/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 ft/min)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="384 741 1198 920"> <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 4-10 m/s (800-2000 ft/min) for extraction of crusher dusts generated 2 metres distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 ft/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 ft/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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<p>Personal protection</p>																	
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 																
<p>Skin protection</p>	<p>See Hand protection below</p>																
<p>Hands/feet protection</p>	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. 																
<p>Body protection</p>	<p>See Other protection below</p>																
<p>Other protection</p>	<p>When cutting wear approved dust respirator to avoid inhalation of wood dust created during the cutting process.</p> <ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. <p>Avoid breathing dust when sawing or grinding.</p> <p>WARNING: Wood dusts have been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS.</p> <p>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.</p> <p>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.</p>																

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/m ³	Sensitiser, A4***	May produce asthma
Oak and beech	1 mg/m ³	A1*	May affect pulmonary function
Birch, mahogany, teak, walnut	1 mg/m ³	A2*	May affect pulmonary function
All other species	1 mg/m ³	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 µm in aerodynamic diameter; however, between 61% and 65% of the particles by count measured between 1 and 5 µm 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.

Respiratory protection

Type AX-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	AX-AUS P2	-	AX-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AX-AUS / Class 1 P2	-
up to 100 x ES	-	AX-2 P2	AX-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)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Manufactured pressed board ranging in thickness from 5mm to 40mm, made from wood particles/fibres, bonded together with resin. Newly manufactured board or freshly cut surfaces may have a pine odour.		
Physical state	Manufactured	Relative density (Water = 1)	0.65-0.75
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>220
pH (as supplied)	Not Applicable	Decomposition temperature	Not Applicable
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

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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 Applicable	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Applicable
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (Not Available%)	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	<p>New boards or freshly cut surfaces may have a pine/wood/resin odour which will dissipate with ventilation. When cutting, wood dust will be created which is classified as a Hazardous Substance according to the criteria of NOHSC. Atmosphere should be checked and if necessary suitable arrangements made to reduce the level of vapours in the breathing zone for persons working in the area.</p> <p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>▸ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations.</p>
Ingestion	<p>The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p>
Skin Contact	<p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p>
Eye	<p>Although the material is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).</p>
Chronic	<p>This manufactured article is considered to have low hazard potential if handling and personal protection recommendations are followed.</p>

Trade Essentials - Whiteboard HMR	TOXICITY	IRRITATION
	Not Available	Not Available
urea/ formaldehyde resin	TOXICITY	IRRITATION
	dermal (rat) LD50: >2100 mg/kg ^[2]	Eye (rabbit): 0.1 ul/24h -SEVERE
	Inhalation(Rat) LC50; >0.167 mg/L4h ^[2]	Skin (rabbit): 500 mg/24h-SEVERE
	Oral (Mouse) LD50; 6361 mg/kg ^[2]	
melamine/ urea/ formaldehyde resin	TOXICITY	IRRITATION
	Oral (Rat) LD50; >5000 mg/kg ^[2]	Not Available

paraffin wax	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg/24 hr-mild
	Oral (Rat) LD50; >5000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 500 mg/24 hr-mild
		Skin: no adverse effect observed (not irritating) ^[1]
wood dust softwood	TOXICITY	IRRITATION
	Not Available	Not Available
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

UREA/ FORMALDEHYDE RESIN	<p>NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p> <p>Somnolence, impaired liver function tests, changes in leucocyte (WBC) count recorded.</p>
PARAFFIN WAX	<p>"Hydrocarbon wax" describes a group of solid C20 to C36 paraffinic hydrocarbons which are not absorbed in the gastro-intestinal tract and in small quantity will pass through undigested.</p> <p>The widespread use in cosmetic and in cosmetic surgery over many years demonstrates the low toxicity of refined waxes and many guidelines exist for their safe use Notwithstanding this, there are occasional reports of adverse effects with these products. Subcutaneous deposits often referred to as paraffinoma, have been described frequently following injection of these materials under the skin but these are not normally associated with other progressive changes.</p> <p>Paraffin wax and microcrystalline were each administered orally as a solution in arachis oil to groups of 5 male and 5 female rats at dose levels of 1000 and 5000 g/kg bw. produced no clinical signs of toxicity during the seven day observation period and growth rates were normal. There were no mortalities and no macroscopic changes were observed at autopsy.</p> <p>Three samples of 50% paraffin in petrolatum were tested in repeated, open patch applications to 6 rabbits. Two samples produced erythema in four animals that lasted three days, and one produced erythema in one rabbit that lasted two days. A microcrystalline wax was slightly irritating, to rabbit skin, in a 24 hour occluded patch test.</p> <p>Four 50% solutions of paraffin in petrolatum were each instilled into the eyes of six albino rabbits with no rinse. Eyes were observed for irritation for three days. Two of the samples caused mild irritation in one rabbit on day 1; the other samples were not irritating</p> <p>In a long-term feeding study with Sprague-Dawley rats, no wax-related effects were observed. In a series of 180-day feeding studies in rats that were performed over a period of approximately 15 years (beginning in 1955) on chewing-gum bases containing hydrocarbon wax in proportions varying from 2% to 57% of the gum base, no compound-related effects were observed.</p> <p>Long-term toxicity studies indicated that petroleum-derived paraffin and microcrystalline waxes are non-toxic and non-carcinogenic.</p> <p>Eight slack waxes and eight aromatic hydrocarbon extracts derived from the slack waxes were tested for carcinogenicity after applying these to the skin of mice. The slack waxes showed only a low order of carcinogenicity at 250 days. However by 450 days every sample of slack wax had elicited papillomas and for 5 of them cancers as well. The aromatic extracts on the other hand exhibited a greater potency. At 250 days all but one sample had produced papillomas and 5 samples had produced cancers. At 450 days all but one sample had elicited cancers and all had elicited papillomas. The authors concluded that the carcinogenicity of slack wax can be attributed to the aromatic compounds found in the oils from which the waxes were pressed and which are retained on the waxes as impurities, and is not due to paraffins.</p> <p>Five petrolatum waxes were negative for local and systemic carcinogenicity or toxicity in skin-painting studies in mice and rabbits. However, wax disk implants, but not ground wax implants, were associated with the development of fibrosarcomas at the implantation site in rats.</p> <p>A description of the accumulation of long-chain alkanes (C29, C31, and C33) in a patient who had died of heart disease led the author to conclude that these hydrocarbons were of dietary (plant) origin as judged by the tissue distribution of the alkanes.</p> <p>The EU Scientific Committee for Food (SCF) reviewed the available information on mineral hydrocarbons, which included the petroleum waxes. Their opinion was published in 1995. The SCF reached the following conclusion:</p> <p>There are sufficient data to allow a full Group ADI (Average daily Intake) of 0-20 mg/kg bw for waxes conforming to the following specification: -</p> <ul style="list-style-type: none"> · Highly refined waxes derived from petroleum based or synthetic hydrocarbon feedstocks, with viscosity not less than 11 m³/s (cSt) at 100 deg C · Carbon number not less than 25 at the 5% boiling point · Average molecular weight not less than 500 <p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial</p>

biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.

The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives; The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:

- The adverse effects of these materials are associated with undesirable components, and
- The levels of the undesirable components are inversely related to the degree of processing;
- Distillate base oils receiving the same degree or extent of processing will have similar toxicities;
- The potential toxicity of *residual base oils* is independent of the degree of processing the oil receives.
- The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing.

The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential.

Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.

Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing

Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils).

Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).

Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class (Other Lubricant Base Oils).

Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class (Other Lubricant Base Oils))

Germ cell mutagenicity: The tests performed within the 'in vivo' studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class (Other Lubricant Base Oils)).

Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction.

STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m³ and for systemic effects NOAEL > 980 mg/m³.

Sub-chronic toxicity

90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies).

Repeat dose toxicity:

Oral

NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.

Inhalation

The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m³. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m³.

Dermal

In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.

Toxicity to reproduction:

Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.

Developmental toxicity, teratogenicity:

Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.

The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic

Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small

intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen.

Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.

Highly and Severely Refined Distillate Base Oils

Acute toxicity: Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to > 4 mg/l.

When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating"

Testing in guinea pigs for sensitization has been negative

Repeat dose toxicity: Several studies have been conducted with these oils. The weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil s toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.

- The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,
- The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and
- The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.

Reproductive and developmental toxicity: A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study s authors.

A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed fetuses were found among three litters The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.

Genotoxicity:

In vitro (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.

In vivo (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.

Carcinogenicity: Highly & severely refined base oils are not carcinogens, when given either orally or dermally.

Tumorigenic in rats

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 lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. No significant acute toxicological data identified in literature search.

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 oropharynx, 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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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*.

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.

**UREA/ FORMALDEHYDE
RESIN & MELAMINE/
UREA/ FORMALDEHYDE
RESIN**

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

Trade Essentials - Whiteboard HMR

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	✗
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

Trade Essentials - Whiteboard HMR	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
urea/ formaldehyde resin	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
melamine/ urea/ formaldehyde resin	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
paraffin wax	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 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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
urea/ formaldehyde resin	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
urea/ formaldehyde resin	LOW (LogKOW = -3.4014)

Mobility in soil

Ingredient	Mobility
urea/ formaldehyde resin	HIGH (KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▸ 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.
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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
urea/ formaldehyde resin	Not Available
melamine/ urea/ formaldehyde resin	Not Available
paraffin wax	Not Available
wood dust softwood	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
urea/ formaldehyde resin	Not Available
melamine/ urea/ formaldehyde resin	Not Available
paraffin wax	Not Available
wood dust softwood	Not Available

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture****urea/ formaldehyde resin is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

melamine/ urea/ formaldehyde resin is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

paraffin wax is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

wood dust softwood is found on the following regulatory lists

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (urea/ formaldehyde resin; melamine/ urea/ formaldehyde resin; paraffin wax)

National Inventory	Status
China - IECSC	No (urea/ formaldehyde resin)
Europe - EINEC / ELINCS / NLP	No (urea/ formaldehyde resin; melamine/ urea/ formaldehyde resin)
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (melamine/ urea/ formaldehyde resin)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (urea/ formaldehyde resin; melamine/ urea/ formaldehyde resin)
Vietnam - NCI	Yes
Russia - FBEPH	No (melamine/ urea/ formaldehyde resin)
Legend:	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</p>

SECTION 16 Other information

Revision Date	01/11/2019
Initial Date	09/03/2010

SDS Version Summary

Version	Date of Update	Sections Updated
8.1	18/01/2017	Classification
9.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

Other information

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

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

Definitions and abbreviations

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

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