Formica Vinyl Doors

Laminex Group Pty Ltd

Chemwatch Hazard Alert Code: **2**

Chemwatch: **4758-41**
Version No: **5.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **20/08/2021**
Print Date: **31/10/2024**
L.GHS.AUS.EN

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

Product Identifier

Product name	Formica Vinyl Doors
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 for interior use.

Details of the manufacturer or 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 (24/7)**
Emergency telephone number(s)	+61 1800 951 288
Other emergency telephone number(s)	+61 3 9573 3188

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

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Germ Cell Mutagenicity 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

Page 1 continued...

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

Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H341	Suspected of causing genetic defects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing dust/fumes.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
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.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		PVC veneer, containing
9002-86-2	<10	polyvinyl chloride
Not Available		adhered to reconstituted wood panel containing
Not Available	>60	softwood particles
Not Available		bonded together with
9011-05-6	<10	urea/ formaldehyde resin
25036-13-9	10-30	melamine/ urea/ formaldehyde resin
Not Available	<10	paper
Not Available		the encapsulated panel will contain traces of

Continued...

CAS No	%[weight]	Name
57-13-6		urea
50-00-0		formaldehyde
822-06-0		hexamethylene diisocyanate
Not Available		dust from sawing and forming operations will contain
Not Available	NotSpec	wood dust softwood
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	If this product comes in contact with eyes: ▸ 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.
Skin Contact	If skin contact occurs: ▸ 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	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ 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

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

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	▸ 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	Brännbar. Kommer att brinna om den antänds. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) and minor amounts of hydrogen cyanide other pyrolysis products typical of burning organic material.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Continued...

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ 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	▸ 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	▸ 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	formaldehyde	Formaldehyde	1 ppm / 1.2 mg/m3	2.5 mg/m3 / 2 ppm	Not Available	Not Available
Australia Exposure Standards	hexamethylene diisocyanate	Hexamethylene diisocyanate	Not Available	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
polyvinyl chloride	Not Available	Not Available
urea/ formaldehyde resin	Not Available	Not Available
melamine/ urea/ formaldehyde resin	Not Available	Not Available
urea	Not Available	Not Available
formaldehyde	20 ppm	Not Available
hexamethylene diisocyanate	Not Available	Not Available
wood dust softwood	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
polyvinyl chloride	E	≤ 0.01 mg/m³
urea/ formaldehyde resin	E	≤ 0.01 mg/m³
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.***	

Continued...

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
melamine/ urea/ formaldehyde resin	D	> 0.01 to ≤ 0.1 mg/m³
urea	E	≤ 0.01 mg/m³
wood dust softwood	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
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

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

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

(a): particle dust respirators, if necessary, combined with an absorption cartridge;
(b): filter respirators with absorption cartridge or canister of the right type;
(c): fresh-air hoods or masks

- Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding.
- Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting.

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

Type of Contaminant:	Air Speed:
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 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)

Within each range the appropriate value depends on:

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

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.

Individual protection measures, such as personal protective equipment

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Continued...

Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber **NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
Body protection	See Other protection below
Other protection	When cutting wear approved dust respirator to avoid inhalation of wood dust created during the cutting process. ‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit. Avoid breathing dust when sawing or grinding. WARNING: Wood dusts have been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS. Wood dusts produce dermatitis and an increased risk of upper respiratory disease. Epidemiological studies in furniture workers show an increased risk of lung, tongue, pharynx and nasal cancer. An excess risk of leukaemia amongst millwrights probably is associated with exposure to various components used in wood preservation. IARC has not limited this finding to any specific type of industry (e.g. furniture manufacturing) or wood dust source (hardwood vs. softwood). IARC s conclusions are based primarily on human carcinogenicity data from studies of various exposed worker populations. The softwood TLV-TWA reflects the apparent low risk for upper respiratory tract involvement amongst workers in the building industry. A separate TLV-TWA, for hard woods, is based on impaired nasal mucociliary function reported to contribute to nasal adenocarcinoma and related hyperplasia found in furniture workers. Allergic reactions are more common from handling green timber, less common for dried hardwood. Impairment of nasal mucociliary function may occur below 5 mg/m3 and may be important in the development of nasal adenocarcinoma amongst furniture workers exposed to hardwoods. Certain exotic hardwoods contain alkaloids which may produce headache, anorexia, nausea, bradycardia and dyspnoea. ACGIH Exposure Standards for Wood dusts

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

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

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Formica Vinyl Doors

Material	CPI
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
PE	C
PE/EVAL/PE	C
PVC	C

Respiratory protection

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

Continued...

SARANEX-23	C
TEFLON	C
VITON	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

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	PVC coated wood-based panel. Newly manufactured board or freshly cut surfaces may have a pine/wood odour

Physical state	Solid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Applicable	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Applicable
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	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

Continued...

Inhaled	New boards or freshly cut surfaces may have a pine/wood/solvent 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. 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. ▸ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations.
Ingestion	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.
Skin Contact	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.
Eye	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).
Chronic	The sealer for the the final topcoat contains toluene diisocyanate, which is s skin sensitiser, but this is expected to be fully reacted. When cutting new sheets there is a miniscule possibility that free isocyanate may be available - care should be taken and correct safety procedures followed to minimise exposure. This manufactured article is considered to have low hazard potential if handling and personal protection recommendations are followed.

Formica Vinyl Doors	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
polyvinyl chloride	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
urea/ formaldehyde resin	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2100 mg/kg[2]	Eye (Rodent - rabbit): 100uL/24H - Severe
	Inhalation (Rat) LC50: >0.167 mg/L4h[2]	Skin (Rodent - rabbit): 500mg/24H - Severe
	Oral (Mouse) LD50; 6361 mg/kg[2]	
melamine/ urea/ formaldehyde resin	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >5000 mg/kg[2]	Not Available
urea	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 8200 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 8471 mg/kg[2]	Skin (Human): 20%/24H - Moderate
		Skin (Human): 22mg/3D (intermittent) - Mild
		Skin: no adverse effect observed (not irritating)[1]
formaldehyde	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 270 mg/kg[2]	Eye (Human): 1ppm/6M - Mild
	Inhalation (Rat) LC50: <463 ppm4h[1]	Eye (Human): 4ppm/5M
	Oral (Rat) LD50: 100 mg/kg[2]	Eye (Rodent - mouse): 3% - Moderate
		Eye (Rodent - rabbit): 10mg - Severe
		Eye (Rodent - rabbit): 37% - Severe
		Eye (Rodent - rabbit): 750ug - Severe
		Eye (Rodent - rabbit): 750ug/24H - Severe
		Skin (Human - man): 1%/2D
		Skin (Human): 0.01% - Severe
		Skin (Human): 150ug/3D (intermittent) - Mild
		Skin (Human): 2%/48H
		Skin (Rodent - mouse): 7% - Moderate
		Skin (Rodent - rabbit): 0.8% - Severe
		Skin (Rodent - rabbit): 2mg/24H - Severe

Continued...

		Skin (Rodent - rabbit): 50mg/24H - Moderate
		Skin (Rodent - rabbit): 540mg - Mild
		Skin (Rodent - rat): 7% - Moderate
		Skin: adverse effect observed (corrosive)[1]

hexamethylene diisocyanate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 593 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Inhalation (Rat) LC50: 0.06 mg/L4h[2]	Skin: adverse effect observed (corrosive)[1]
	Oral (Mouse) LD50; 350 mg/kg[2]	Skin: adverse effect observed (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***

POLYVINYL CHLORIDE	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
UREA/ FORMALDEHYDE RESIN	Somnolence, impaired liver function tests, changes in leucocyte (WBC) count recorded. **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.
UREA	Altered sleep time, change in motor activity, antipsychosis, dyspnea, methaemoglobinaemia, convulsions, lymphomas recorded. Carcinogenic by RTECS criteria. Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies with similar materials using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. For urea: There is little data that relates urea to human health other than its use in dermatology and some more limited applications in clinical medicine. The use of urea (at 10% concentration or less) in ointments and creams to treat dry skin has been widespread, and long term follow-up studies have indicated that the substance is nonallergenic and virtually free from side effects. Among other clinical therapeutic uses, the treatment of inappropriate secretion of antidiuretic hormone (SIADH) should be noted, because its chronic form has involved long term oral administration of large amounts of urea. Most patients have tolerated urea well, although diarrhoea is sometimes reported after ingestion of 60-90 g/day. The possibility exists that infection of H. pylori in human stomach may aggravate local effects by urea because of ammonia generation. **Acute toxicity:** The acute toxicity by urea is well delineated by the oral route. Toxicity is low in mammals other than ruminants, especially cattle, and sheep, in which the rumen micro-organisms contain urease activity and metabolise urea to ammonia at a high rate. In mice and rats, urea is of low toxicity even by the subcutaneous and intravenous route. **Repeated dose toxicity:** No well-conducted repeated dose toxicity studies on urea were located. Chronic toxicity and carcinogenicity screening studies in mice and rats fed with 4500, 9000 or 45000 ppm in diet (up to about 6750 mg/kg body weight/day for mice and about 2250 mg/kg body weight/day for rats) did not uncover any treatment-related toxic syndromes in the various organs studied. Neither was any weight depression noted at terminal necropsy for animals of either sex or species at any dose levels. Thus the NOAELs were about 6750 mg/kg body weight/day for mice and about 2250 mg/kg body weight/day for rats. Repeated dose toxicity studies with rats by skin application over 4 weeks and 25 weeks were conducted using urea ointment at 10%, 20% and 40% concentrations, and no consistent treatment-related toxic effects were found. The ointments were applied on a 20 cm2 area of the back skin; it is concluded that the repeated dose toxicity of urea by dermal route is low. **Reproductive/developmental toxicity:** The studies cited under repeated dose toxicity did not indicate any toxic effects on the reproductive organs of mice and rats. No adequate teratogenicity/developmental toxicity studies of urea with mammals were located. According to one rat study, 50 g/kg body weight/day administered by gavage in two doses 12 hours apart for an average of 14 days did not cause outstanding (external) teratogenicity; the mean birthweight of the newborn was lower but the litter size greater. Injection of urea into the air sack of eggs shows that urea is toxic to the development of chick embryo. No NOAEL can be given for the reproductive/developmental toxicity of urea because appropriate studies are lacking. **Genetic toxicity:** Urea has been negative in several appropriately conducted bacterial mutagenicity tests. Urea caused DNA single strand breaks in mammalian cells in vitro and was clastogenic for mammalian cells in vitro and in vivo but only at concentrations much beyond the physiological range (about 50-100 higher concentrations than found in human blood). The mechanism of genotoxicity is probably non-specific (e.g. difference in osmotic pressure across the cell membrane).
FORMALDEHYDE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

Continued...

	WARNING: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [***National Toxicology Program: U.S. Dep. of Health & Human Services 2002***]
HEXAMETHYLENE DIISOCYANATE	Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities. Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages. Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material. For diisocyanates: In general, there appears to be little or no difference between aromatic and aliphatic diisocyanates as toxicants. In addition, there are insufficient data available to make any major distinctions between polymeric (<1000 MW) and monomeric diisocyanates. Based on repeated dose studies in animals by the inhalation route, both aromatic and aliphatic diisocyanates appear to be of high concern for pulmonary toxicity at low exposure levels. Based upon a very limited data set, it appears that diisocyanate prepolymers exhibit the same respiratory tract effects as the monomers in repeated dose studies. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route. Most members of the diisocyanate category have not been tested for carcinogenic potential. Though the aromatic diisocyanates tested positive and the one aliphatic diisocyanate tested negative in one species, it is premature to make any generalizations about the carcinogenic potential of aromatic versus aliphatic diisocyanates. In the absence of more human data, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Diisocyanates are moderate to strong dermal sensitisers in animal studies. Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. For monomers, effects on the respiratory tract (lungs and nasal cavities) were observed in animal studies at exposure concentrations of less than 0.005 mg/L. The experimental animal data available on prepolymeric diisocyanates show similar adverse effects at levels that range from 0.002 mg/L to 0.026 mg/L. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route. **Oncogenicity:** Most members of the diisocyanate category have not been tested for carcinogenic potential. Commercially available Poly-MDI was tested in a 2-year inhalation study in rats. The tested material contained 47% aromatic 4,4'-methylenediphenyl diisocyanate (MDI) and 53% higher molecular weight oligomers. Interim sacrifices at one year showed that males and females in the highest dose group (6 mg/m3) had treatment related histological changes in the nasal cavity, lungs and mediastinal lymph nodes. The incidence and severity of degeneration and basal cell hyperplasia of the olfactory epithelium and Bowman's gland hyperplasia were increased in males at the mid and high doses and in females at the high dose following the two year exposure period. Pulmonary adenomas were found in 6 males and 2 females, and pulmonary adenocarcinoma in one male in the high dose group. However, aliphatic hexamethylene diisocyanate (HDI) was found not to be carcinogenic in a two year repeated dose study in rats by the inhalation route. HDI has not been tested in mice by the inhalation route. Though the oral route is not an expected route of exposure to humans, it should be noted that in two year repeated dose studies by the oral route, aromatic toluene diisocyanate (TDI) and 3,3'-dimethoxy-benzidine-4,4'-diisocyanate (dianisidine diisocyanate, DADI) were found to be carcinogenic in rodents. TDI induced a statistically significant increase in the incidence of liver tumors in rats and mice as well as dose-related hemangiosarcomas of the circulatory system and has been classified by the Agency as a B2 carcinogen. DADI was found to be carcinogenic in rats, but not in mice, with a statistically increase in the incidence of pancreatic tumors observed. **Respiratory and Dermal Sensitization:** Based on the available toxicity data in animals and epidemiologic studies of humans, aromatic diisocyanates such as TDI and MDI are strong respiratory sensitisers. Aliphatic diisocyanates are generally not active in animal models for respiratory sensitization. However, HDI and possibly isophorone diisocyanate (IPDI), are reported to be associated with respiratory sensitization in humans. Symptoms resulting from occupational exposure to HDI include shortness of breath, increased bronchoconstriction reaction to histamine challenges, asthmatic reactions, wheezing and coughing. Two case reports of human exposure to IPDI by inhalation suggest IPDI is a respiratory sensitiser in humans. In view of the information from case reports in humans, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Studies in both human and mice using TDI, HDI, MDI and dicyclohexylmethane-4,4'-diisocyanate (HMDI) suggest cross-reactivity with the other diisocyanates, irrespective of whether the challenge compound was an aliphatic or aromatic diisocyanate. Diisocyanates are moderate to strong dermal sensitisers in animal studies. There seems to be little or no difference in the level of reactivity between aromatic and aliphatic diisocyanates. **Dermal Irritation:** Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. The level of irritation ranged from slightly to severely irritating to the skin. One chemical, hydrogenated MDI (1,1-methylenebis-4-isocyanatocyclohexane), was found to be corrosive to the skin in guinea pigs. For 1,6-hexamethylene diisocyanate: Exposures to HDI are often associated with exposures to its prepolymers, especially to a trimeric biuretic prepolymer of HDI (HDI-BT), which is widely used as a hardener in automobile and airplane paints, and which typically contains 0.5-1% unreacted HDI. There is evidence that diisocyanate prepolymers may induce asthma at the same or greater frequency as the monomers; therefore, there is a need to assess the potential for human exposure to prepolymeric HDI as well as monomeric HDI. 1,6-Hexamethylene diisocyanate is corrosive to the skin and the eye. 1,6-Hexamethylene diisocyanate was found to induce dermal and respiratory sensitization in animals and humans. There is no threshold known for this effect. Inhalation studies with repeated exposures to 1,6-hexamethylene diisocyanate vapor show that the respiratory tract is the target with 1,6-hexamethylene diisocyanate showing primarily upper respiratory tract lesions (nasal cavity). 1,6-Hexamethylene diisocyanate did not show a neurotoxic effect in a combined reproduction/developmental/neurotoxicity study. Life-time inhalation exposure to rats revealed a progression of non-neoplastic respiratory tract lesions, primarily to the nasal cavity, and represented the sequelae of non-specific irritation. Based on the presence of only reversible tissue responses to irritation at the low concentration of 0.005 ppm, this concentration was a NOAEL. No carcinogenic potential in rats was observed after life-time inhalation. 1,6-Hexamethylene diisocyanate showed no mutagenic activity in vitro in bacterial and in mammalian cell test systems.

Continued...

	1,6-Hexamethylene diisocyanate showed no clastogenic activity in vivo. 1,6-Hexamethylene diisocyanate has no effect on fertility and post-natal viability through post-natal day 4 in the rat after inhalation up to 0.299 ppm. The overall NOEL was 0.005 ppm. Inhalation of 1,6-hexamethylene diisocyanate during the pregnancy of rats produced maternal effects (nasal turbinate histopathology) at concentrations ³ 0.052 ppm. No developmental toxicity was observed up to 0.308 ppm.
WOOD DUST SOFTWOOD	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. For wood dusts: Wood dusts may cause respiratory symptoms including sensitisation and diminished respiratory function and may also be carcinogenic. OSHA has determined that the health evidence for the toxicity of wood dust cannot be separately distinguished for soft wood and hard wood. A final OSHA ruling however establishes an 8-hour TWA PEL of 2.5 mg/m3 for Western red cedar wood dust, based on its widely recognized ability to cause immune-system-mediated allergic sensitization. Evidence in the record demonstrates the seriousness of this effect. Wood dust is defined as any wood particles arising from the processing or handling of woods. Hard woods derive from the deciduous broad-leaved flowering species of trees, and soft woods include the coniferous species that do not shed their leaves in the winter. The distinction between hard woods and soft woods is purely botanical. Many so-called "softwoods" are actually hard (i.e., Douglas fir as a softwood is harder than the hardwood birch) and one of the softest woods in existence (balsa) is botanically a hardwood. Some commentators were of the opinion that many other woods, such as Douglas fir, pine, red and white oak, redwood, walnut, spruce, boxwood, cocobolo, teak, mahogany, and others, should also be designated by OSHA as allergenic in this rulemaking. However, OSHA finds that "it is unlikely that species other than WRC are responsible for large numbers of cases of respiratory allergies". Other commonly used woods such as oak, birch, redwood, pine, teak, alder, and hemlock, produce pulmonary effects that are less well described than the asthma responses to Western red cedar. OSHA is establishing a PEL of 5 mg/m3 as an 8-hour TWA and 10 mg/m3 as a 15-minute STEL for hard and soft wood dust, with the exception of Western red cedar. OSHA concludes that promulgation of these exposure limits will substantially reduce the significant risk of material impairment in the form of pulmonary dysfunction (including changes in peak flow, interference with mucociliary clearance, respiratory symptoms, and chronic effects) that is associated with exposure to wood dust at the higher levels that would be permitted in the absence of any limit. **Carcinogenicity** The association between occupational exposure to wood dust and various forms of cancer has been explored in many studies and in many countries. In 1987, the International Agency for Research on Cancer (IARC) classified furniture manufacturing in Category I (confirmed human carcinogen) and carpentry in Category 2B (suspected human carcinogen). IARC concludes that there is sufficient evidence in humans for the carcinogenicity of wood dust. (Group 1) Wood dust causes cancer of the nasal cavity and paranasal sinuses and of the nasopharynx. IARC also concludes that there is inadequate evidence in experimental animals for the carcinogenicity of wood dust. In 1998, IARC issued the results of its detailed analyses of the combined results from 17 studies of nasal cancers and wood dust exposures. These analyses supported IARC s earlier conclusions and led to the following findings: • Excess sino-nasal cancers were seen primarily in studies of European furniture makers • The degree of risk was increased in workers with the highest level and length of exposure • Observed risk levels were lower in studies of U.S. populations, possibly due to differences in the types of exposures that had occurred (e.g., exposures to different types of wood). Based on its analyses, IARC has concluded that wood dust may cause “adenocarcinomas of the nasal cavities and paranasal sinuses”. This is a specific type of cancer in a specific region in the respiratory tract. IARC did not find sufficient evidence to associate wood dust exposure with other types of cancer of the nasal cavities (e.g., squamous cell carcinomas) or cancers in other parts of the body, such as the oropharynyx, hypopharynx, lung, lymphatic and haematopoietic systems, stomach, colon or rectum. Dust particles may act as carriers for genotoxic agents. Chromium compounds are often present in oak and beech dusts as they are frequently used in the wood-processing industry, particularly as potassium dichromate in stains as well as fixing agents in wood preservatives. Stained furniture is made largely from oak and beech as they contain enough tannic acid to allow for chemical staining Direct genotoxic effects of wood dust extracts were summarized by IARC (1995). 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). Dust particles may act as carriers for genotoxic agents. Chromium compounds are often present in oak and beech dusts as they are frequently used in the wood-processing industry, particularly as potassium dichromate in stains as well as fixing agents in wood preservatives. Exposure to hexavalent chromium has been associated with the development of sinonasal cancers. NIOSH (Ex. 8-47) considers both hard and soft wood dust to be potentially carcinogenic in humans; for soft wood dust, NIOSH recommends a separate 6(b) rulemaking (Ex. 8-47, Table N6B). NIOSH concurred, however, with the proposed PEL of 1 mg/m3 TWA for hard wood dust. Several chemicals were isolated from wood extracts, but only quercetin and delta-3 -carene were shown to be mutagenic (IARC, 1995) Summary of evidence for nasal and sinus cavity cancers. NIOSH (1987a/Ex. 1-1005) concluded that the literature clearly demonstrates an association between occupational wood dust exposure and nasal cancer. English studies first identified this link by showing a 10- to 20-times-greater incidence of nasal adenocarcinoma among woodworkers in the furniture industry than among other woodworkers and 100 times greater than in the general population. In the United States, three studies have reported a fourfold risk of nasal cancer or adenocarcinoma in furniture workers, and another study noted a similar relationship between nasal cancer and wood dust exposure. One other study failed to find such an association for furniture workers, but did find an increase among logging and timber industry workers. The association between lung cancer and occupational wood dust exposure is inconclusive, although several epidemiological studies have reported increases in lung cancer among wood-dust-exposed workers. A significant excess of malignant tumours of the bronchus and lung in carpenters and joiners. Only construction workers showed a statistically significant increase in lung cancer rate. Although the data are conflicting, several epidemiological studies of U.S. workers do report increases in the incidence of Hodgkin's disease among woodworkers. This excess is particularly apparent among carpenters.

Continued...

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

Continued...

	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.
POLYVINYL CHLORIDE & UREA & FORMALDEHYDE & HEXAMETHYLENE DIISOCYANATE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
POLYVINYL CHLORIDE & FORMALDEHYDE & HEXAMETHYLENE DIISOCYANATE & WOOD DUST SOFTWOOD	No significant acute toxicological data identified in literature search.
UREA/ FORMALDEHYDE RESIN & MELAMINE/ UREA/ FORMALDEHYDE RESIN & FORMALDEHYDE & HEXAMETHYLENE DIISOCYANATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
HEXAMETHYLENE DIISOCYANATE & 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.

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

Formica Vinyl Doors

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

polyvinyl chloride

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

Continued...

	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

urea	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	ErC50	72h	Algae or other aquatic plants	24541.9mg/l	2
	EC50	72h	Algae or other aquatic plants	24541.9mg/l	2
	EC50	48h	Crustacea	3910mg/L	4
	LC50	96h	Fish	4.65-8.48mg/l	4
	NOEC(ECx)	5040h	Fish	>=1.71mg/l	2

formaldehyde	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	1.034-1.984mg/l	4
	EC50	48h	Crustacea	3.26mg/l	4
	LC50	96h	Fish	0.727-9.193mg/L	4
	EC50	96h	Algae or other aquatic plants	0.375-0.579mg/l	4
	NOEC(ECx)	96h	Algae or other aquatic plants	0.005mg/l	4

hexamethylene diisocyanate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>77.4mg/l	2
	EC0(ECx)	24h	Crustacea	<0.33mg/l	1
	LC50	96h	Fish	22mg/l	1

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***

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
polyvinyl chloride	LOW	LOW
urea/ formaldehyde resin	LOW	LOW
urea	LOW	LOW
formaldehyde	LOW (Half-life = 14 days)	LOW (Half-life = 2.97 days)
hexamethylene diisocyanate	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
polyvinyl chloride	LOW (LogKOW = 1.6233)
urea/ formaldehyde resin	LOW (LogKOW = -3.4014)
urea	LOW (BCF = 10)
formaldehyde	LOW (LogKOW = 0.35)
hexamethylene diisocyanate	LOW (LogKOW = 3.1956)

Mobility in soil

Ingredient	Mobility
polyvinyl chloride	LOW (Log KOC = 23.74)
urea/ formaldehyde resin	HIGH (Log KOC = 1)
urea	LOW (Log KOC = 4.191)

Continued...

Ingredient	Mobility
formaldehyde	HIGH (Log KOC = 1)
hexamethylene diisocyanate	LOW (Log KOC = 5864)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
polyvinyl chloride	Not Available
urea/ formaldehyde resin	Not Available
melamine/ urea/ formaldehyde resin	Not Available
urea	Not Available
formaldehyde	Not Available
hexamethylene diisocyanate	Not Available
wood dust softwood	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
polyvinyl chloride	Not Available
urea/ formaldehyde resin	Not Available
melamine/ urea/ formaldehyde resin	Not Available
urea	Not Available
formaldehyde	Not Available
hexamethylene diisocyanate	Not Available
wood dust softwood	Not Available

SECTION 15 Regulatory information

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

polyvinyl chloride is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Continued...

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)

urea is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

formaldehyde is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans

hexamethylene diisocyanate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

wood dust softwood is found on the following regulatory lists

Not Applicable

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (polyvinyl chloride; urea/ formaldehyde resin; melamine/ urea/ formaldehyde resin; urea; formaldehyde; hexamethylene diisocyanate)
China - IECSC	No (urea/ formaldehyde resin)
Europe - EINEC / ELINCS / NLP	No (polyvinyl chloride; 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	All chemical substances in this product have been designated as TSCA Inventory ‘Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (urea/ formaldehyde resin; melamine/ urea/ formaldehyde resin)
Vietnam - NCI	Yes
Russia - FBEPH	No (melamine/ urea/ formaldehyde resin)
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	20/08/2021
Initial Date	04/07/2011

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
5.1	20/08/2021	Classification change due to full database hazard calculation/update.

Continued...

Other information

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

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

Definitions and abbreviations

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

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances