CHH Shadowclad Ultra

Carter Holt Harvey Plywood

Chemwatch Hazard Alert Code: **1**

Chemwatch: **5274-60**
Version No: **4.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **01/11/2019**
Print Date: **12/09/2022**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	CHH Shadowclad Ultra
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	Exterior cladding.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Carter Holt Harvey Plywood**
Address	173 Captain Springs Road Onehunga Auckland 1061 New Zealand
Telephone	+64 800 326 759
Fax	Not Available
Website	http://chh.com/
Email	info@ecoply.co.nz

Emergency telephone number

Association / Organisation	**Poison Information Centre (New Zealand)**
Emergency telephone numbers	0800 764 766 (24 hours)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

NON-HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

ChemWatch Hazard Ratings

	Min	Max
Flammability	0	
Toxicity	0	
Body Contact	1	
Reactivity	0	
Chronic	0	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Canadian WHMIS Symbols

Poisons Schedule	Not Applicable
Classification [1]	Not Applicable

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Label elements

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

Hazard statement(s)

Not Applicable

**LIMITED EVIDENCE*

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) General

P101	If medical advice is needed, have product container or label at hand.
P102	Keep out of reach of children.
P103	Read carefully and follow all instructions.

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

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	90-95	wood veneer
40798-65-0	<10	phenol/ formaldehyde polymer sodium salt
7727-43-7	<10	barium sulfate
Not Available	<1	impregnation residuals, as
7440-50-8	^	copper
7440-47-3	^	chromium
7440-38-2	^	arsenic
13463-67-7	^	titanium dioxide
Not Available		In use, may generate wood dust softwood
Not Available		THIS REPORT IS FOR TREATED PRODUCT ONLY

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	‣ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations. If this product comes in contact with eyes:

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	▸ Wash out immediately with water. ▸ If irritation continues, seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	Brush off dust. In the event of abrasion or irritation of the skin seek medical attention.
Inhalation	▸ If dust is inhaled, remove from contaminated area. ▸ Encourage patient to blow nose to ensure clear passage of breathing. ▸ If irritation or discomfort persists seek medical attention.
Ingestion	▸ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations. ▸ 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.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid exposure to excessive heat and fire.

Advice for firefighters

Fire Fighting	Wear breathing apparatus plus protective gloves. Equipment should be thoroughly decontaminated after use. Alert Fire Brigade and tell them location and nature of hazard. Use water delivered as a fine spray to control the fire and cool adjacent area.
Fire/Explosion Hazard	- Wood products do not normally constitute an explosion hazard. - Mechanical or abrasive activities which produce wood dust, as a by-product, may present a severe explosion hazard if a dust cloud contacts an ignition source. - Hot humid conditions may result in spontaneous combustion of accumulated wood dust. - Partially burned or scorched wood dust can explode if dispersed in air. Combustible. Will burn if ignited.
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	Pick up. Refer to major spills.
Major Spills	Pick up. Secure load if safe to do so. Bundle/collect recoverable product.

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

SECTION 7 Handling and storage

Precautions for safe handling

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

Conditions for safe storage, including any incompatibilities

Continued...

Suitable container	Supplied in packets. ‣ Generally not applicable.
Storage incompatibility	‣ Keep dry

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

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	barium sulfate	Barium sulphate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	chromium	Chromium (metal)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	arsenic	Arsenic & soluble compounds (as As)	0.05 mg/m3	Not Available	Not Available	(g) Some compounds in these groups are classified as carcinogenic or as sensitisers. Check individual classification details on the safety data sheet for information on classification.
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
barium sulfate	15 mg/m3	170 mg/m3	990 mg/m3
copper	3 mg/m3	33 mg/m3	200 mg/m3
chromium	1.5 mg/m3	17 mg/m3	99 mg/m3
arsenic	1.5 mg/m3	17 mg/m3	100 mg/m3
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
phenol/ formaldehyde polymer sodium salt	Not Available	Not Available
barium sulfate	Not Available	Not Available
copper	100 mg/m3	Not Available
chromium	250 mg/m3	Not Available
arsenic	5 mg/m3	Not Available
titanium dioxide	5,000 mg/m3	Not Available

MATERIAL DATA

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

Exposure controls

Appropriate engineering	‣ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations.

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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.

General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

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 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection

Eye and face protection

When sawing, machining or sanding use - Safety glasses with side shields.

Skin protection

See Hand protection below

Hands/feet protection

- Protective gloves eg. Leather gloves or gloves with Leather facing
- Safety footwear

Body protection

See Other protection below

Other protection

No special equipment needed when handling small quantities.
OTHERWISE:
- Overalls.
- Barrier cream.
- Eyewash unit.

Respiratory protection

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

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Appearance	Beige sheet of plywood. THIS CHEMWATCH REPORT IS FOR TREATED PRODUCT ONLY.

Physical state	Manufactured	**Relative density (Water = 1)**	0.5-1.0
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	>200
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Applicable	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not 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	Not normally a hazard due to physical form of product. Generated dust may be discomforting
Ingestion	Ingestion of sawdust may cause nausea, abdominal pain, vomiting or diarrhoea. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments
Skin Contact	The dust is discomforting and mildly abrasive to the skin and may cause drying of the skin, which may lead to contact dermatitis.
Eye	The dust may produce eye discomfort causing transient smarting, blinking
Chronic	Wood dust may cause skin and respiratory sensitisation. ‣ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations. Common chronic responses to wood dust exposures are dermatitis, simple bronchitis and non asthmatic chronic airflow obstruction. Wood is an organic substrate for growth of micro-organisms and fungal spores, these readily become airborne with wood dust and have caused a variety of respiratory infections Various woods, mainly tropical varieties, are able to induce allergies in joiners, carpenters, cabinet makers and model-makers. Allergies of the immediate type (rhino conjunctivitis, bronchial asthma, urticaria), caused by contact with dusts produced during wood-working and those of a delayed type (contact eczema) caused by both the dust and by direct contact with the solid wood, are seen in an occupational setting. Because of the large number of substances found in wood, only a few low molecular weight allergens have been isolated and identified; these are mostly quinone or flavone derivatives. Many of the constituents of wood may also cause primary irritation. Irritation of the skin, eyes and respiratory passages are often distinguished from allergic responses with difficulty. The use of skin tests with wood dusts to confirm suspected allergy must be viewed as suspect because the high concentration of wood components which are sometimes applied, can actually produce new sensitisation in test subjects. It should also be noted that cross-reactions or reactions to groups of similar substances, in other woods and also in other herbaceous plants can also

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occur. The substances in wood responsible for respiratory allergies are probably mostly high molecular weight substances. Wood dusts may induce asthmatic reactions of both the immediate and delayed types, and occasionally, both. Positive results in bronchial provocation tests, are often, but not always, associated with positive results in skin tests and IgE induction. Bronchial provocation tests may produce different results dependent on whether they are carried out with course or fine dusts or with lyophilised aqueous extracts. Very course dust may produce false negatives and very fine dust may produce false positives (irritation). Non-allergenic bronchial and nasal irritation are seen frequently.
Certain exotic woods contain alkaloids which may produce headache, anorexia, nausea, bradycardia and dyspnea. Agents used to treat wood (preservatives, fungicides, stains, glues, pore fillers) may themselves be responsible for allergic reaction. Other allergic reactions may be provoked by liverworts ("Frullania dermatitis"), lichens, fungi (e.g. bronchopulmonary aspergillosis), actinomycetes or other plants which grow on wood. Microorganisms and fungal spores, associated with wood, may become airborne and provoke allergic responses. Other chronic responses associated with exposure to wood dusts include conjunctivitis, simple bronchitis and non-asthmatic chronic airflow obstruction.
Epidemiologic studies in furniture workers show an increased risk of lung, tongue, pharynx and nasal cancer (adenocarcinoma). Workers in timber industries, with a history of exposure to wood dust, have shown increased occurrence of lung, liver and vocal cavity cancer. An excess risk of leukaemia amongst mill-wrights probably is associated with various components used in wood preservation. It is now suggested that sinonasal cancers may be caused by both hardwoods and softwoods (1). The causative agent or agents are unknown although certain aldehydes or their quinone oxidation products have been implicated. Exposure standards for the softwoods reflect the apparent low risk for upper respiratory tract involvement among workers in the building industry. A significantly lower exposure standard for hardwoods is based on impaired nasal mucociliary hyperplasia reported to contribute to nasal adenocarcinoma and related hyperplasia in furniture workers. Exposure standards for both hard and softwoods specifically exclude the issue of occupational asthma and related allergic respiratory response associated with exposure to red cedar dusts and similar woods.
The main components of wood are polysaccharides: cellulose (40-50 wt%) and hemicelluloses (20–35%), while lignin comprises 15–30% of wood mass.3 In addition to these macromolecules, wood contains a small amount of inorganic residues and extractives, which are low molar mass molecules. Extractives include a heterogeneous group of aliphatic and cyclic compounds: terpenes and terpenoids, esters of fatty acids, fatty acids, alcohols, alkanes, simple phenols, stilbenes, lignans, isoflavones, condensed tannins, flavonoids and hydrolyzable tannins. Wood phenolic compounds may possess bioactive functions; in vitro studies suggest that they may act as antioxidants. Due to the close association of lignin and extractives with cellulose and hemicelluloses, low amounts of these compounds commonly exist in hemicellulose or cellulose extracts and can, thus, be considered as “co-passengers” of fibrous materials. While wood extracts are neither presently nor extensively used in food ingredients, they have a long history in food supplement use. Softwood extracts have also received attention in the biomedical field; spruce hemicellulose extract was patented for “use on the treatment of lower urinary tract symptoms and diseases”.
The presence of mycotoxins is unlikely given the production procedure (particularly as there was no significant delay between grinding and extraction). The possibility of fungal contamination on the tree stumps is also unlikely since, firstly, these stumps come from felled wood which is therefore healthy, and secondly, if a fungal contamination were to appear (in the event that the stumps were not collected quickly after the trees were felled), this would essentially be an external contamination which would be eliminated when the stumps were examined before the grinding process.
Radionuclide monitoring checks should be carried out systematically for all batches.

CHH Shadowclad Ultra	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

phenol/ formaldehyde polymer sodium salt	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

barium sulfate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (Mouse) LD50; >3000 mg/kg[2]	

copper	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; 0.733 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; 0.7 mg/kg[2]	

chromium	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >5.41 mg/l4h[1]	Not Available
	Oral (Rat) LD50; >5000 mg/kg[1]	

arsenic	**TOXICITY**	**IRRITATION**
	Oral (Mouse) LD50; 144 mg/kg[2]	Eye: adverse effect observed (irreversible damage)[1]
		Skin: adverse effect observed (irritating)[1]

titanium dioxide	**TOXICITY**	**IRRITATION**
	dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]

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	Inhalation(Rat) LC50; >2.28 mg/l4h[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Oral (Rat) LD50; >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

COPPER	WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever. 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. for copper and its compounds (typically copper chloride): **Acute toxicity:** There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs. No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation. **Repeat dose toxicity:** In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride. **Genotoxicity:** An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen. **Carcinogenicity:** there was insufficient information to evaluate the carcinogenic activity of copper monochloride. Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).
CHROMIUM	Gastrointestinal tumours, lymphoma, musculoskeletal tumours and tumours at site of application recorded. For chrome(III) and other valence states (except hexavalent): For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases. The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells. enter cells The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental, divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of

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	absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter though these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures. The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage. Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms. 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. Tenth Annual Report on Carcinogens: Substance known to be Carcinogenic [*National Toxicology Program: U.S. Dep. of Health and Human Services 2002*]
ARSENIC	Tumorigenic - Carcinogenic by RTECS criteria. Arsenic compounds are classified by the European Union as toxic by inhalation and ingestion and toxic to aquatic life and long lasting in the environment. IARC classify arsenic in drinking water as a confirmed human carcinogen (IARC 1). The main inorganic forms of arsenic relevant for human exposures are pentavalent arsenic (also called arsenate, As(V), or As+5) and trivalent arsenic (also called arsenite, As(III), or As+3). These inorganic species undergoes a series of reduction and oxidative/methylation steps in human liver and other tissues to form tri- and pentavalent methylated metabolites of methylarsonite [MA(III)], methylarsonate [MA(V)], dimethylarsinite [DMA(III)], and dimethylarsinate [DMA(V)]. Some mammalian species also produce trimethylated metabolites, trimethylarsine oxide The distinction between inorganic and organic forms is important because it is generally accepted that the organic species are excreted more quickly from the body and generally considered less toxic, with a relative rank order of As(III) > As(V) >> MA(V), DMA(V) >> arsenobetaine. However, the methylated trivalent metabolites, MA(III) and DMA(III), are significantly more toxic than their pentavalent counterpart and either As(III) or As(V) . In many cases, biomonitoring or environmental occurrence data are reported as total arsenic and do not distinguish between the different species. In those situations, understanding the relevant sources of arsenic is essential to evaluate potential arsenic related health effects, especially those related to inorganic arsenic exposure. **WARNING:** This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**.
TITANIUM DIOXIDE	* IUCLID Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. 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. For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also

Continued...

	exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. **Animal carcinogenicity data** Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
PHENOL/ FORMALDEHYDE POLYMER SODIUM SALT & BARIUM SULFATE & CHROMIUM & TITANIUM DIOXIDE	No significant acute toxicological data identified in literature search.

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

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

SECTION 12 Ecological information

Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
CHH Shadowclad Ultra	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...

phenol/ formaldehyde polymer sodium salt	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
barium sulfate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	72h	Algae or other aquatic plants	>=1.15mg/l	2
	EC50	72h	Algae or other aquatic plants	>1.15mg/l	2
	EC50	48h	Crustacea	32mg/l	4
	LC50	96h	Fish	>3.5mg/l	2
copper	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
	EC50	48h	Crustacea	<0.001mg/L	4
	LC50	96h	Fish	0.005-0.06mg/l	4
	EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
chromium	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	48h	Crustacea	<0.001mg/l	2
	EC50	72h	Algae or other aquatic plants	0.026-0.208mg/L	4
	EC50	48h	Crustacea	<0.001mg/l	2
	LC50	96h	Fish	0.106mg/L	4
	EC50	96h	Algae or other aquatic plants	36mg/L	4
arsenic	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC10(ECx)	48h	Crustacea	0.006mg/l	2
	EC50	48h	Crustacea	0.85mg/l	2
	LC50	96h	Fish	2.8-4.2mg/l	Not Available
	EC50	96h	Algae or other aquatic plants	0.11-0.209mg/l	4
titanium dioxide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
	NOEC(ECx)	504h	Crustacea	0.02mg/l	4
	LC50	96h	Fish	1.85-3.06mg/l	4
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

Although treated, the solid wood will decay on ground contact.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
titanium dioxide	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
titanium dioxide	LOW (BCF = 10)

Mobility in soil

Ingredient	Mobility
titanium dioxide	LOW (KOC = 23.74)

Continued...

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. ▸ Bury residue in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
phenol/ formaldehyde polymer sodium salt	Not Available
barium sulfate	Not Available
copper	Not Available
chromium	Not Available
arsenic	Not Available
titanium dioxide	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
phenol/ formaldehyde polymer sodium salt	Not Available
barium sulfate	Not Available
copper	Not Available
chromium	Not Available
arsenic	Not Available
titanium dioxide	Not Available

SECTION 15 Regulatory information

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

phenol/ formaldehyde polymer sodium salt is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

barium sulfate 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 - Group 1: Carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

copper is found on the following regulatory lists

Continued...

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

chromium 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
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

arsenic 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 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Australian Inventory of Industrial Chemicals (AIIC)
FEI Equine Prohibited Substances List - Banned Substances
FEI Equine Prohibited Substances List (EPSL)
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
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

titanium dioxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (phenol/ formaldehyde polymer sodium salt; barium sulfate; copper; chromium; arsenic)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (phenol/ formaldehyde polymer sodium salt)
Japan - ENCS	No (phenol/ formaldehyde polymer sodium salt; copper; chromium; arsenic)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (phenol/ formaldehyde polymer sodium salt)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (phenol/ formaldehyde polymer sodium salt)
Vietnam - NCI	No (phenol/ formaldehyde polymer sodium salt)
Russia - FBEPH	No (phenol/ formaldehyde polymer sodium salt)
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	01/11/2019
Initial Date	01/12/2017

SDS Version Summary

Continued...

Version	Date of Update	Sections Updated
3.1	22/05/2018	Exposure Standard, Personal Protection (Respirator), Supplier Information, Name
4.1	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