Shadowclad Ultra LOSP Treated Plywood

Carter Holt Harvey Plywood Ltd

Chemwatch Hazard Alert Code: **2**

Chemwatch: **7999-21**
Version No: **2.1**
Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Initial Date: **23/12/2025**
Revision Date: **23/12/2025**
Print Date: **29/01/2026**
L.GHS.NZL.EN.RISK.E

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

Product Identifier

Product name	Shadowclad Ultra LOSP Treated Plywood
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	Suitable for use in residential, commercial and industrial construction, as well as in fitments and general building uses. Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**Carter Holt Harvey Plywood Ltd**
Address	173 Captain Springs Rd Onehunga Auckland 1061 New Zealand
Telephone	+64 800 746 399
Fax	Not Available
Website	CHHPly.co.nz
Email	info@chhply.co.nz

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone number(s)	Not Available
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Not regulated for transport of Dangerous Goods.

Chemwatch Hazard Ratings

	Min	Max
Flammability	1	
Toxicity	1	
Body Contact	2	
Reactivity	1	
Chronic	2	

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

Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 2 ***LIMITED EVIDENCE***
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

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Determined by Chemwatch using GHS/HSNO criteria	6.3A, 6.4A, 6.7B, 6.1E (respiratory tract irritant) ***LIMITED EVIDENCE**

Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H351	Suspected of causing cancer.

***LIMITED EVIDENCE**

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

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.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.

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.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
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.
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.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

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

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

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CAS No	%[weight]	Name
Not Available	80-85	wood veneer
40798-65-0	<11	phenol/ formaldehyde polymer sodium salt
Not Available	<9	Treatment residuals may include
8052-41-3.	^	white spirit
107534-96-3	^	tebuconazole
60207-90-1	^	propiconazole
52645-53-1	^	permethrin
55406-53-6	^	3-iodo-2-propynyl butyl carbamate
557-09-5	^	zinc octoate
Not Available	<1	Coating comprises
7727-43-7	^	barium sulfate
13463-67-7	^	C.I. Pigment White 6
2451-62-9	^	triglycidyl isocyanurate
68186-94-7	^	C.I. Pigment Black 26

Legend:	***1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
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 or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
For chronic or short term repeated exposures to pyrethrum and synthetic pyrethroids:

- Mammalian toxicity of pyrethrum and synthetic pyrethroids is low, in part because of poor bioavailability and a large first pass extraction by the liver.
- The most common adverse reaction results from the potent sensitising effects of pyrethrins.
- Clinical manifestations of exposure include contact dermatitis (erythema, vesiculation, bullae); anaphylactoid reactions (pallor, tachycardia, diaphoresis) and asthma. [***Ellenhorn Barceloux***]
- In cases of skin contact, it has been reported that topical application of Vitamin E Acetate (alpha-tocopherol acetate) has been found to have high therapeutic value, eliminating almost all skin pain associated with exposure to synthetic pyrethroids. [***Incitec***]

SECTION 5 Firefighting measures

Extinguishing media

- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

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- 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	▸ Combustible solid which burns but propagates flame with difficulty; it is estimated that most organic dusts are combustible (circa 70%) - according to the circumstances under which the combustion process occurs, such materials may cause fires and / or dust explosions. ▸ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions). ▸ Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion. Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn rapidly and fiercely if ignited - particles exceeding this limit will generally not form flammable dust clouds; once initiated, however, larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. ▸ In the same way as gases and vapours, dusts in the form of a cloud are only ignitable over a range of concentrations; in principle, the concepts of lower explosive limit (LEL) and upper explosive limit (UEL) are applicable to dust clouds but only the LEL is of practical use; - this is because of the inherent difficulty of achieving homogeneous dust clouds at high temperatures (for dusts the LEL is often called the "Minimum Explosible Concentration", MEC). ▸ When processed with flammable liquids/vapors/mists,ignitable (hybrid) mixtures may be formed with combustible dusts. Ignitable mixtures will increase the rate of explosion pressure rise and the Minimum Ignition Energy (the minimum amount of energy required to ignite dust clouds - MIE) will be lower than the pure dust in air mixture. The Lower Explosive Limit (LEL) of the vapour/dust mixture will be lower than the individual LELs for the vapors/mists or dusts. ▸ A dust explosion may release of large quantities of gaseous products; this in turn creates a subsequent pressure rise of explosive force capable of damaging plant and buildings and injuring people. ▸ Usually the initial or primary explosion takes place in a confined space such as plant or machinery, and can be of sufficient force to damage or rupture the plant. If the shock wave from the primary explosion enters the surrounding area, it will disturb any settled dust layers, forming a second dust cloud, and often initiate a much larger secondary explosion. All large scale explosions have resulted from chain reactions of this type. ▸ Dry dust can be charged electrostatically by turbulence, pneumatic transport, pouring, in exhaust ducts and during transport. ▸ Build-up of electrostatic charge 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. ▸ All movable parts coming in contact with this material should have a speed of less than 1-meter/sec. ▸ A sudden release of statically charged materials from storage or process equipment, particularly at elevated temperatures and/ or pressure, may result in ignition especially in the absence of an apparent ignition source. ▸ One important effect of the particulate nature of powders is that the surface area and surface structure (and often moisture content) can vary widely from sample to sample, depending of how the powder was manufactured and handled; this means that it is virtually impossible to use flammability data published in the literature for dusts (in contrast to that published for gases and vapours). ▸ Autoignition temperatures are often quoted for dust clouds (minimum ignition temperature (MIT)) and dust layers (layer ignition temperature (LIT)); LIT generally falls as the thickness of the layer increases. Combustion products include: ▸ carbon monoxide (CO) ▸ carbon dioxide (CO_2) hydrogen iodide metal oxides ▸ other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. ▸ Powdered Phenolic resin is a combustible dust and this means that it is capable of forming flammable and explosive dust clouds in air. Such dust clouds can be sensitive to low energy ignition. Combustion can also propagate along a powder trail of settled dust, or result in repeated explosions as more dust is disturbed and rises into the air. ▸ The presence of dust external to plant items creates a potential hazard in that a secondary explosion could occur in the event of a flame or burning material being ejected due to a primary explosion within plant equipment. ▸ The severity of explosions by ignition of dust clouds is often much greater than that of vapour or gas mixtures and in industrial situations the potential exists for substantial damage to structures and harm to personnel. ▸ For Phenol Formaldehyde* powders the explosion severity is 3.9 based upon an explosion severity rating. Similarly flammability rating for Phenol Formaldehyde* powders (relative sensitivity of dusts to ignition) is 9.3 based upon a severity rating. *[Empirical scale based upon standard Pittsburgh coal dust being 1.0] ▸ Guidance as how to safely handle combustible dust can be obtained from including but not limited to AS/NZ standard 4745:2004 (Code of Practice for handling Combustible Dusts)and US National Fire Protection Association Standard 654. It is

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	highly recommended that these standards be consulted prior to assessing and addressing the risks that can be encountered, other reference standards are (including but not limited to): AS/NZS 30000: 2000 Electrical installations AS/NZS 2381.1: 1999 Electrical equipment for explosive atmospheres.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ Clean up waste regularly and abnormal spills immediately. ▸ Avoid breathing dust and contact with skin and eyes. ▸ Wear protective clothing, gloves, safety glasses and dust respirator. ▸ Use dry clean up procedures and avoid generating dust. ▸ Vacuum up or sweep up. **NOTE:** Vacuum cleaner must be fitted with an exhaust micro filter (H-Class HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). H-Class HEPA filtered industrial vacuum cleaners should NOT be used on wet materials or surfaces. ▸ Dampen with water to prevent dusting before sweeping. ▸ Place in suitable containers for disposal.
Major Spills	Moderate hazard. ▸ **CAUTION**: Advise personnel in area. ▸ Alert Emergency Services and tell them location and nature of hazard. ▸ Control personal contact by wearing protective clothing. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Recover product wherever possible. ▸ **IF DRY:** Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. **IF WET:** Vacuum/shovel up and place in labelled containers for disposal. ▸ **ALWAYS:** Wash area down with large amounts of water and prevent runoff into drains. ▸ If contamination of drains or waterways occurs, advise Emergency Services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid skin contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to come in direct contact with human skin or eyes. ▸ **DO NOT** allow material to come in contact with exposed food or food contact surfaces. ▸ Suitable PPE must be worn at all times. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. ▸ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) ▸ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. ▸ Establish good housekeeping practices. ▸ Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds. ▸ Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in.(0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. ▸ Do not use air hoses for cleaning. ▸ Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used. ▸ Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition.

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	▸ Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance. ▸ Do not empty directly into flammable solvents or in the presence of flammable vapors. ▸ The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges. Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source. ▸ Do NOT cut, drill, grind or weld such containers. ▸ In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.
Other information	Store in the dark. ▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry area protected from environmental extremes. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. For major quantities: ▸ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams}. ▸ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Glass container is suitable for laboratory quantities ▸ Polyethylene or polypropylene container. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents

+	x	+	o	+	+	o

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
New Zealand Workplace Exposure Standards (WES)	white spirit	Stoddard solvent (White spirits)	100 ppm / 525 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	3-iodo-2-propynyl butyl carbamate	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	3-iodo-2-propynyl butyl carbamate	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	zinc octoate	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	zinc octoate	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	barium sulfate	Barium sulphate respirable dust	1 mg/m3	5 mg/m3	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	C.I. Pigment White 6	Titanium dioxide ultrafine dust	0.2 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	C.I. Pigment White 6	Titanium dioxide respirable dust	2.5 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	triglycidyl isocyanurate	Triglycidyl isocyanurate (TGIC)	0.08 mg/m3	Not Available	Not Available	Not Available

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	C.I. Pigment Black 26	Manganese fume, dust and compounds, as Mn	0.2 mg/m3	Not Available	Not Available	oto - Ototoxin
New Zealand Workplace Exposure Standards (WES)	C.I. Pigment Black 26	Manganese fume, dust and compounds, as Mn respirable dust	0.02 mg/m3	Not Available	Not Available	oto - Ototoxin

MATERIAL DATA

Exposure controls

Appropriate engineering controls

Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation.
HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours.
Barrier protection or laminar flow cabinets should be considered for laboratory scale handling.
A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg.
When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology.
Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies.
Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required.
Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved. Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. 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, 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 (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, 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.)

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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 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.
The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated.
The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of:
10; high efficiency particulate (HEPA) filters or cartridges
10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator.
25-50; a full face-piece negative pressure respirator with HEPA filters
50-100; tight-fitting, full face-piece HEPA PAPR
100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode.

Individual protection measures, such as personal protective equipment

Eye and face protection

When handling very small quantities of the material eye protection may not be required.
For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs:
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- Face shield. Full face shield may be required for supplementary but never for primary protection of eyes.

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	▸ 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
Hands/feet protection	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▸ Double gloving should be considered. ▸ PVC gloves. ▸ Change gloves frequently and when contaminated, punctured or torn. ▸ Wash hands immediately after removing gloves. ▸ Protective shoe covers. [AS/NZS 2210] ▸ Head covering. Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present. ▸ polychloroprene. ▸ nitrile rubber. ▸ butyl rubber. ▸ fluorocaoutchouc. ▸ polyvinyl chloride. Gloves should be examined for wear and/ or degradation constantly.
Body protection	See Other protection below
Other protection	▸ For quantities up to 500 grams a laboratory coat may be suitable. ▸ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ▸ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ▸ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ▸ Eye wash unit.

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	▸ Ensure there is ready access to an emergency shower. ▸ For Emergencies: Vinyl suit

Respiratory protection

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

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

· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.
Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.
P2 is a respiratory filter rating under various international standards, Filters at least 94% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Plywood Manufacture from wood veneers, clear in colour with one face powder coated. Potential for light chemical odour from preservative and solvents; insoluble in water.

Physical state	Manufactured	**Relative density (Water = 1)**	Not Available
Odour	Light chemical	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	>200
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	>200
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not 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

Continued...

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	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ 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

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.
Eye	This material causes serious eye irritation.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms

Continued...

	can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. Chronic poisoning by natural pyrethrins may result in convulsion, tetanic paralysis, rapid and uneven heart beat, liver and kidney damage, or death. The natural pyrethrins may produce hypersensitivity, especially following previous sensitising exposure. In general, repeated exposures over 2 or 3 years are required to elicit a response and involve exposure to pyrethrum rather than its individual components (including pyrethrins). The sesquiterpene lactone (pyrethrosin) and the pyrethrum glycoproteins account for the immediate and delayed hypersensitivity seen in guinea pigs following a single injection of ground chrysanthemum in Freud's adjuvant. Mild erythematic vesicular dermatitis (with papules), pruritus, localized oedema (particularly of the face, lips and eyelids), rhinitis, tachycardia, pallor and sweating are the most common syndromes. An initial skin sensitisation can progress to marked dermal oedema and skin cracking. Pyrethrum dermatitis appears to increase in hot weather or under conditions were heavy perspiration is produced. The active ingredients of pyrethrum (except pyrethrin II) are inactive in patch tests. Those patients allergic to ragweed pollen are particularly sensitive to pyrethrin. Rats fed on a diet of pyrethrins for 5000 ppm for 2 years showed some signs of tissue damage including liver lesions, bile duct proliferation and focal necrosis of the liver cells. A no-effect level of 1000 ppm found in animal experiments correspond to a daily dose of 3600 mg/man.

Shadowclad Ultra LOSP Treated Plywood	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
phenol/ formaldehyde polymer sodium salt	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
white spirit	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3000 mg/kg[1]	Eye (Human): 100ppm - Mild
	Inhalation (Rat) LC50: >5.5 mg/l4h[1]	Eye (Rodent - rabbit): 500mg/24H - Moderate
	Oral (Rat) LD50: >5000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
tebuconazole	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >5000 mg/kg[2]	Not Available
	Inhalation (Rat) LC50: >0.8 mg/L4h[2]	
	Oral (Mouse) LD50; 2000 mg/kg[2]	
propiconazole	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >4000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >5.8 mg/L4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 550 mg/kg[1]	
permethrin	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 1750 mg/kg[2]	Skin (Rodent - rabbit): 500mg/24H - Mild
	Oral (Rat) LD50: 383 mg/kg[2]	Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
3-iodo-2-propynyl butyl carbamate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye: adverse effect observed (irreversible damage)[1]
	Inhalation (Rat) LC50: 0.63 mg/l4h[1]	Skin (Human): 0.3%/48H
	Oral (Rat) LD50: 1056 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
zinc octoate	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >5.08 mg/l4h[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Mouse) LD50; 2370 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
barium sulfate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; >3000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

Continued...

C.I. Pigment White 6	**TOXICITY**	**IRRITATION**
	dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >2.28 mg/l4h[1]	Skin (Human): 300ug/3D (intermittent) - Mild
	Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
triglycidyl isocyanurate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 100mg - Severe
	Inhalation (Rat) LC50: 0.65 mg/L4h[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: <100 mg/kg[2]	Skin: adverse effect observed (irritating)[1]
C.I. Pigment Black 26	**TOXICITY**	**IRRITATION**
	Not Available	Eye: no adverse effect observed (not irritating)[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***

WHITE SPIRIT	white spirit, as CAS RN 8052-41-3 For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.
TEBUCONAZOLE	(aerosol) NOEL (2 y)* for rats, 300 mg/kg diet for dogs, 100 mg/kg " for mice, 20 mg/kg " ADI 0.03 mg/kg b.w. * Toxicity Class WHO III; EPA III * Side effects of antiestrogens include hot flashes, osteoporosis, breast atrophy, vaginal dryness, and vaginal atrophy. In addition, they may cause depression and reduced libido. The antiestrogen withdrawal response is a paradoxical improvement in breast cancer caused by discontinuation of antiestrogen therapy for breast cancer. It has been documented rarely with the selective estrogen receptor modulators (SERMs) tamoxifen and raloxifene. The phenomenon indicates that these agents can somehow result in stimulation of breast cancer tumor progression under certain circumstances. One proposed theory for the mechanism is that the sensitivity of breast cells to estrogens shifts with estrogen deprivation, and upon antiestrogen withdrawal, endogenous estrogen acts in the manner of high-dose estrogen therapy in the breast to inhibit breast cancer growth and induce breast cancer cell death. The antioestrogen withdrawal syndrome is analogous to but less common and well-known than the antiandrogen withdrawal syndrome, a phenomenon in which paradoxical improvement in prostate cancer occurs upon discontinuation of antiandrogen therapy. aromatase inhibitors (AIs) are commonly used in breast cancer treatment, but they can cause various side effects, primarily due to estrogen depletion. Common side effects include musculoskeletal problems like joint pain (arthralgia), muscle stiffness, and bone loss leading to osteoporosis and fractures. Additionally, women may experience menopausal symptoms such as hot flashes, vaginal dryness, and sexual dysfunction. Other potential side effects include fatigue, insomnia, and an increased risk of cardiovascular events. ther Common Side Effects: Fatigue: Many patients report feeling tired or weak. Insomnia: Sleep disturbances are also common. Cardiovascular Events: While less frequent, there's a slightly increased risk of heart problems, especially in women with pre-existing conditions. Weight Gain: AIs can contribute to weight gain. Mood Changes: Some women experience depression or mood swings. Less Common but Serious Side Effects: Liver Problems: AIs can occasionally affect liver function. Skin Reactions: Skin rashes and other skin changes can occur. Cardiovascular Risks: Some studies suggest an increased risk of heart attack or stroke. Cytochrome P450 (CYP) enzyme modulators can cause adverse effects, primarily through drug-drug interactions. Specifically, CYP inhibitors can increase the concentration of other drugs in the body, leading to toxicity or adverse reactions, while CYP inducers can decrease drug concentrations, potentially causing therapeutic failure. CYP inhibitors, particularly those affecting CYP3A4, can cause a range of adverse effects due to their impact on drug metabolism. These effects include increased risk of toxicity from other drugs, arrhythmias like torsades de pointes, rhabdomyolysis, and even potentially fatal outcomes

Continued...

epending on the specific CYP inhibitor and the other drugs involved, adverse effects can include gastrointestinal disorders, liver damage, and neurological problems.
CYP inhibitors can reduce the metabolism of other drugs, leading to higher concentrations in the bloodstream and potentially increasing the risk of side effects and toxicity
People metabolize drugs differently, and factors like age, sex, genetics, and other medical conditions can influence how CYP inhibitors affect them.
Incidents of liver injury or failure among modern antifungal medicines are very low to non-existent. However, some can cause allergic reactions in people.[
There are also many drug interactions. Patients must read in detail the enclosed data sheet(s) of any medicine. For example, the azole antifungals such as ketoconazole or itraconazole can be both substrates and inhibitors of the P-glycoprotein, which (among other functions) excretes toxins and drugs into the intestines.] Azole antifungals also are both substrates and inhibitors of the cytochrome P450 family CYP3A4,[] causing increased concentration when administering, for example, calcium channel blockers, immunosuppressants, chemotherapeutic drugs, benzodiazepines, tricyclic antidepressants, macrolides and SSRIs.[35]
Before oral antifungal therapies are used to treat nail disease, a confirmation of the fungal infection should be made.[
Approximately half of suspected cases of fungal infection in nails have a non-fungal cause.[The side effects of oral treatment are significant and people without an infection should not take these drugs.[
Azoles are the group of antifungals which act on the cell membrane of fungi. They inhibit the enzyme 14-alpha-sterol demethylase, a microsomal CYP, which is required for biosynthesis of ergosterol for the cytoplasmic membrane. This leads to the accumulation of 14-alpha-methylsterols resulting in impairment of function of certain membrane-bound enzymes and disruption of close packing of acyl chains of phospholipids, thus inhibiting growth of the fungi. Some azoles directly increase permeability of the fungal cell membrane.
vntifungal resistance is a subset of antimicrobial resistance, that specifically applies to fungi that have become resistant to antifungals. Resistance to antifungals can arise naturally, for example by genetic mutation or through aneuploidy. Extended use of antifungals leads to development of antifungal resistance through various mechanisms.
Some fungi (e.g. Candida krusei and fluconazole) exhibit intrinsic resistance to certain antifungal drugs or classes, whereas some species develop antifungal resistance to external pressures. Antifungal resistance is a One Health concern, driven by multiple extrinsic factors, including extensive fungicidal use, overuse of clinical antifungals, environmental change and host factors.]
Unlike resistance to antibacterials, antifungal resistance can be driven by antifungal use in agriculture. Currently there is no regulation on the use of similar antifungal classes in agriculture and the clinic.
The emergence of Candida auris as a potential human pathogen that sometimes exhibits multi-class antifungal drug resistance is concerning and has been associated with several outbreaks globally. The WHO has released a priority fungal pathogen list, including pathogens with antifungal resistance
conazoles are azole antifungals used in agricultural and pharmaceutical products. Exposure to conazole fungicides leads to several toxic endpoints, including reproductive and endocrine. The results of animal experiments have shown that various conazole fungicides at high doses affect the structure and functions of reproductive organs. In males, adverse effects of conazole fungicides are manifested in the testes, prostate, sperm viability, fertility and sexual behaviour. Reduced testis weight, testis atrophy and reduced or absent sperm production were frequently observed.
In female genitalia, structural changes in the ovaries and uterus have been observed. The extent of the changes depends on the dose and duration of treatment. Triazoles affected the expression of multiple genes involved in steroid hormone metabolism and modulate enzyme activity of multiple cytochrome P450 (CYP) and other metabolic enzymes in mammalian liver and other tissues. Conazole fungicides act as endocrine disruptors. Conazoles have been reported to reduce oestradiol and testosterone production and to increase progesterone concentration, indicating the inhibition of enzymes involved in the conversion of progesterone to testosterone. The reproductive effects are consistent with impairment of testosterone homeostasis. The disruption in steroid homeostasis is a common mode of action, leading to abnormal reproductive development and diminished reproductive function. At high doses, azole fungicides affect reproductive organs and fertility in several species.
The relatively poor selectivity of the agricultural azoles for the fungal CYP51 over the human homolog raises the concern that exposure to azole fungicide residues might disrupt sterol biosynthesis and other endogenous downstream cytochrome P450 metabolic systems, such as human steroidogenesis and phase I metabolism of xenobiotics in the liver.
agricultural azoles have the general ability to inhibit the P450 enzymes in steroidogenesis, albeit with different potencies. Since the introduction of large-scale use of azole antifungals increasing evidence of hepatotoxicity and associated hepatic tumors has been reported with liver tissue concentrations of ketoconazole and itraconazole being reported as 22- and 10-fold higher, respectively, than plasma levels (58), indicating azole toxicity in the liver being more acute than in other tissues.
Rapidly multiplying cancer cells synthesize greater amounts of cholesterol to build their membranes.
Sterol 14alpha-demethylase (CYP51) is potentially is a specific drug target because of its role in the production of cholesterol in animals Sterol biosynthesis is an essential metabolic pathway in most eukaryotes and in some bacteria. Sterols (cholesterol in humans, sitosterol in plants, ergosterol and its C24-alkylated derivatives in fungi and protozoa) are required components of eukaryotic membranes, where they control fluidity and permeability and modulate functions of membrane-bound enzymes, receptors, and ion channels. These sterols also serve as precursors for multiple regulatory molecules that are crucial for cell division, growth, and development.
Sterol biosynthesis is the target for many drugs. Statins, as inhibitors of HMG-CoA reductase (EC.1.1.1.34), act upstream in the pathway at the step of mevalonate production and serve as major cholesterol-lowering drugs in humans, while azoles, as inhibitors of fungal and protozoan cytochrome P450 sterol 14a-demethylase (CYP51, EC.1.14.13.70), act downstream in the pathway at its postsqualene portion and are widely used as antimicrobial agent.
Human CYP51 [<35% amino acid sequence identity with fungal and <25% identity with protozoan orthologs (9)] has also been considered as a drug target.
Due to the side effects of statins, alternative drugs that target cholesterol biosynthesis in humans more specifically would be highly desirable, and CYP51, the lanosterol demethylase, is a target of interest in that several more distal (post-lanosterol) steps in the cholesterol synthesis pathway are known to be associated with hereditary diseases when they are attenuated (40). For instance, a loss of the sterol 7-reductase (DHCR7) is associated with a severe disease, Smith-Lemli-Opitz syndrome (41). Deficiency in the 3ß-hydroxysteroid dehydrogenase (NSDHL) is responsible for congenital hemidysplasia, ichthyosiform nevus, and limb defects (CHILD) syndrome (42). Mutations in the ?7,?8-sterol isomerase (EBP) are associated with Conradi-Hünermann-Happle syndrome, or X-linked dominant chondrodysplasia punctate type 2 (CDPX2) (40), and mutations in sterol 5-

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desaturase (SC5D) cause lathosterolosis (43). Compared with these problems, attenuating lanosterol 14-demethylation seems very favourable.
It is now generally accepted that cancer cells have elevated levels of cholesterol in lipid rafts and contain more lipid rafts than their normal cell line counterparts (26, 27, 29, 31, 44). Enhanced expression of enzymes of the cholesterol pathway has been reported in many cancer cell types (31, 45–47). Furthermore, certain types of cancer exhibit CYP51 gene amplification (https://www.cbioportal.org). It is likely that the earlier attempts to develop human CYP51 inhibitors were unsuccessful because they concentrated on substrate analogs, which had rather low inhibitory potency (10) and could not compete in efficiency with statins.
in comparison with orthologs from other biological kingdoms, human CYP51 has a broader substrate profile, displays faster catalytic rates, and is resistant to inhibition with the azole drugs and drug candidates that target CYP51s of microbial pathogens.
in comparison with orthologs from other biological kingdoms, human CYP51 has a broader substrate profile, displays faster catalytic rates, and is resistant to inhibition with the azole drugs and drug candidates that target CYP51s of microbial pathogens.
However, screening a variety of commercial and experimental inhibitors of microbial CYP51 orthologs revealed that most of them (including all clinical antifungals) weakly inhibit human CYP51 activity,
Present in all animals, plants, fungi, in some protozoa and bacteria, the CYP51 protein located in the inner face of the endoplasmic reticulum is a membrane monospanning enzyme Its N-terminus includes an amphipathic helix, which links the catalytic subunit to the lipid bilayer.
Sterol synthesis is a very ancient pathway. After the appearance of molecular oxygen in the atmosphere, squalene-2,3-epoxide is formed and then cyclized to steroid precursors, such as lanosterol. Under the oxidative removal of methyl groups by CYP51, these precursors were transformed into ergosterol, which is critical in membrane permeability and fluidity in the fungal kingdom.
Cytochrome P450s (P450s, CYP) are an abundant hemease superfamily. As the first group of enzymes ranked as “superfamily,” cytochrome P450s play an important role in the primary as well as secondary metabolic pathway.
These members are important for catalyzing the oxidative process of various organic substrates, and play a critical role during heterogeneous metabolism and steroid conversion in biological kingdoms.
Unlike other CYP enzymes, CYP51 has a strong specificity. It only catalyzes the demethylation of a very narrow range of substrates, including lanoserol, obtusifoliol, 24,25-dihydrolanosterol, 24-methylenedihydrolanosterol and 4 beta-desmethyllanosterol. The CYP51-involved catalytic reaction consists of three steps, each of which requires one molecule of oxygen and two molecules of NADPH-sourced reduction equivalent. The first two steps are typical cytochrome P450 monooxygenation processes, during which the 14a methyl is converted to methyl alcohol and further converted to methyl aldehyde. And in the last step, the aldehyde group is transformed into formic acid and detached, accompanied with the synthesis of the delta-14, 15 double bond.
The 14a-demethylase is the only invariant P450 present in all sterol biosynthetic pathways, suggesting that all sterol 14a-demethylases share a common prokaryotic ancestor CYP51s are widely distributed in the fungal kingdom. However, in different species of fungi, there are still differences in the types and subtypes, as shown in the phylogenetic tree.
As potential anticancer agents, human CYP51 inhibitors are expected to have generally the same mode of action as statins in vivo, yet present certain important advantages. First, their specificity for the biosynthesis of cholesterol would help to avoid side effects of statins due to inhibition of other metabolic pathways. Second, as with systemic clinical antifungal azoles, which kill pathogenic cells that invade multiple organs and tissues, they should have broad tissue distribution. Finally, because all mammalian CYP51 enzymes share very high amino acid sequence identity (e.g., human/mouse 88%, human/dog 96%), animal in vivo models should produce highly relevant outcomes in preclinical trials. Potent inhibitors of human CYP51 may also have advantages as alternative medications for treatment of other cholesterol-related human diseases. The dual occupancy may be important in the mechanism of human CYP51 inhibition.
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	Rapidly multiplying cancer cells synthesize greater amounts of cholesterol to build their membranes. Sterol 14alpha-demethylase (CYP51) is potentially is a specific drug target because of its role in the production of cholesterol in animals. It is now generally accepted that cancer cells have elevated levels of cholesterol in lipid rafts and contain more lipid rafts than their normal cell line counterparts. Enhanced expression of enzymes of the cholesterol pathway has been reported in many cancer cell types. Furthermore, certain types of cancer exhibit CYP51 gene amplification. It is likely that the earlier attempts to develop human CYP51 inhibitors were unsuccessful because they concentrated on substrate analogs, which had rather low inhibitory potency and could not compete in efficiency with statins. As potential anticancer agents, human CYP51 inhibitors are expected to have generally the same mode of action as statins in vivo, yet present certain important advantages. First, their specificity for the biosynthesis of cholesterol would help to avoid side effects of statins due to inhibition of other metabolic pathways. Second, as with systemic clinical antifungal azoles, which kill pathogenic cells that invade multiple organs and tissues, they should have broad tissue distribution. Finally, because all mammalian CYP51 enzymes share very high amino acid sequence identity (e.g., human/mouse 88%, human/dog 96%), animal in vivo models should produce highly relevant outcomes in preclinical trials. Potent inhibitors of human CYP51 may also have advantages as alternative medications for treatment of other cholesterol-related human diseases. The dual occupancy may be important in the mechanism of human CYP51 inhibition.
PROPICONAZOLE	No sensitisation in guinea pigs * ADI 0.04 mg/kg b.w. * Toxicity Class WHO III NOEL for dogs 50 ppm (1.9 mg/kg b.w. daily) *
PERMETHRIN	Oral (rat) LD50: 430-4000 mg/kg * Oral (mouse) LD50: 540-2960 mg/kg * cis/trans ratio: 40:60 cis/trans ratio: 20:80 ADI: 0.05 mg/kg for nominal cis-trans 40:60 and 25:75 isomers only The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
3-IODO-2-PROPYNYL BUTYL CARBAMATE	for carbamates: Carbamates are effective insecticides by virtue of their ability to inhibit acetylcholinesterase (AChE) (EC 3.1.1.7) in the nervous system. They can also inhibit other esterases. The carbamylation of the enzyme is unstable, and the regeneration of AChE is relatively rapid compared with that from a phosphorylated enzyme. Thus, carbamate pesticides are less dangerous with regard to human exposure than organophosphorus pesticides. The ratio between the dose required to produce death and the dose required to produce minimum symptoms of poisoning is substantially larger for carbamate compounds than for organophosphorus compounds. A dose-effect relationship exists between the dose, the severity of symptoms, and the degree of cholinesterase (ChE) inhibition. Because most carbamates have a low volatility, inhalation studies are mainly carried out using a dust or mist. In these studies, the toxicity is highly dependent on the size of the particles or droplets and, therefore, difficult to evaluate. The acute dermal toxicity of carbamates is generally low to moderate. From controlled human studies, it is clear that poisoning symptoms can be seen a few minutes after exposure, and can last for a few hours. Thereafter, recovery starts and within hours, the symptoms disappear, and the ChE activity in erythrocytes and plasma returns to normal, because the carbamate is rather rapidly metabolised and the metabolites excreted. The appearance of these metabolites in the urine may be used for biological monitoring. Apart from the symptoms indicative of ChE poisoning, other signs and symptoms induced by certain carbamates have been described, such as skin and eye irritation, hyperpigmentation, and influence on the function of testes (slight increase of sperm abnormalities). These signs and symptoms were found in a few studies and should be confirmed before it can be stated that they were induced by carbamates. Epidemiological studies with persons primarily exposed to carbamates are not available. Carbamates produce slight to moderate skin and eye irritation, depending on the vehicle used, duration of contact, and on whether the substance is applied to the abraded or intact skin. From the available data, it cannot be excluded that some of the carbamates will have a slight to moderate sensitization potential. Short- and long-term toxicity studies have been carried out. Some carbamates are very toxic and others are less toxic in long- term studies. From these studies, it is evident that, apart from the anticholinesterase activity, the following changes can be found: an influence on the haemopoietic system, an influence on the functioning of, and, at higher dosages, degeneration of, the liver and kidneys, and degeneration of testes. These abnormalities in the different organ systems depend on the animal strain and on the chemical structure of the carbamate. A clear influence on the nervous system, functional as well as histological, was found, particularly in non-laboratory animals such as pigs. A considerable number of reproduction and teratogenicity studies have been carried out with different carbamates and various animal species. Different types of abnormalities were found, i.e., increase in mortality, disturbance of the endocrine system, and effects on the hypophysis and its gonadotrophic function. These effects were mainly seen at high dose levels. Generally, the fetal effects included an increase in mortality, decreased weight gain in the first weeks after birth, and induction of early embryonic death. All these effects can be summarized as embryotoxic effects. Certain carbamates also induce teratogenic effects, mainly at high dose levels applied by stomach tube. When the same dose level was administered with the diet, no effects were seen. Some carbamates induce mutagenic effects, others are negative. In general, the methyl carbamates are negative in mammalian tests, while compounds such as carbendazim, benomyl, and the 2 thiophanate derivatives showed a positive effect with very high dose levels in certain systems. The benzimidazole moiety may act as a base analogue for DNA and as a spindle poison. They are antimitotic agents and cause mitotic arrest, mitotic delay, and a low incidence of chromosome damage. Sometimes, the results are contradictory or cannot be reproduced, but positive results for point mutation and chromosome aberrations are well documented. These benzimidazole derivatives can be considered as weak mutagenic compounds. Carcinogenicity studies with benzimidazole derivatives showed either positive or equivocal results. Added to the fact that certain mutagenicity studies also give positive results, it cannot be excluded that these compounds may have carcinogenic or promotor properties. Carbamate pesticides may be converted to ***N*** -nitroso compounds. This was demonstrated in a great number of ***in vivo*** nitrosation studies in which high levels of the carbamates were administered to animals in combination with high levels of nitrite. These ***N*** - nitroso compounds have to be considered as mutagenic and carcinogenic. However, the amount of nitroso compounds that can be expected to result from dietary intake of carbamate pesticide residues is negligible in comparison with nitroso-precursors that occur naturally in food and drinking-water. The metabolic fate of carbamates is basically the same in plants, insects, and mammals. Carbamates are usually easily absorbed through the skin, mucous membranes, and respiratory and gastrointestinal tracts, but there are exceptions. Generally, the metabolites are less toxic than the parent compounds. However, in certain cases, the metabolites are just as toxic or even more toxic than the parent carbamate. In most mammals, the metabolites are mainly excreted rather rapidly in the urine. The dog seems to be different in this respect. Accumulation takes place in certain cases, but is of minor importance because of the rapid metabolism. The first step in the metabolism of carbamates is hydrolysis to carbamic acid, which decomposes to carbon dioxide (CO_2) and the corresponding amine. The rate of hydrolysis by esterases is faster in mammals than in plants and insects. The organs in which residues have been reported are the liver, kidneys, brain, fat, and muscle. The half-life in the rat is of the order of 3 - 8 h. From the limited data available, it seems that the excretion of carbamates via urine is also rapid in man, and that the metabolic pathways in man are the same as those in the rat for 3-iodo-2-propynyl butyl carbamate (IPBC):

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	Acute toxicity: Acceptable acute toxicity studies with IPBC indicate low toxicity except eye irritation. In a primary eye irritation study in rabbi. IPBC technical was severely irritating to the eyes of white rabbits, with corneal opacity and corneal vascularization reported in unwashed eyes by day 21 post-treatment. The technical grade of IPBC was slightly irritating to the skin of white rabbits. In a dermal sensitization study in Guinea pigs IPBC technical, at a concentration of 0.32%, produced no evidence of sensitization in male and female Guinea pigs. **Subchronic toxicity:** In a subchronic oral toxicity study, male and female Sprague-Dawley rats received IPBC technical by gavage for 13 weeks at doses of 0, 20, 50, and 125 mg/kg/day. At the 125 mg/kg/day dose level, body weight gain was decreased by 19% in male rats for weeks 1-13 of the study, and by 12% in female rats over the same period. Absolute liver weight was increased by 20% in male rats at the 125 mg/kg/day dose, and by 31% in female rats at this dose level. Liver to body weight ratio was significantly increased by approximately 31% in both male and female rats at the 125 mg/kg/day dose level, while kidney to body weight ratio in female rats was increased 18% at the 125 mg/kg/day dose level. The systemic NOEL was considered to be 20 mg/kg/day, while the systemic LEL was considered to be 50 mg/kg/day, based on increased liver to body weight ratio. In a subchronic dermal toxicity study, male and female Sprague-Dawley rats (10/sex/dose) received dermal doses of 50, 200, and 500 mg/kg/day IPBC technical grade (97.5%) to the shaved skin for five days a week, six hours per day. At the 500 mg/kg/day dose, decreased body weight (4-6%) and weight gain (11%) were observed in male rats, but not in female rats. In female rats, significant increases in haemoglobin, haematocrit, and eosinophils were observed at the 500 mg/kg/day dose level. Reticulocytes as a percentage of red cells were decreased in the 50 and 200 mg/kg/day dose groups but not at the 500 mg/kg/day dose level. Females in this study showed inhibition of plasma cholinesterase at 500 mg/k/day test article, which may have been the result of either direct liver toxicity or inhibition of cholinesterase itself. Based upon the results of this study, the systemic NOEL is 200 mg/kg/day, the systemic LEL is 500 mg/kg/day for male and female rats. **Carcinogenicity:** In a 2-year chronic toxicity/carcinogenicity study, technical grade IPBC (98.68% ai) was administered to male and female Sprague Dawley rats (50/sex/group) at dose levels of 0, 20, 40, and 80 mg/kg/day. There were no statistically significant increases in tumor incidences in male rats. The incidence of mammary gland fibroadenoma and combined fibroadenoma/carcinoma in female rats was significantly increased at the 20 mg/kg/day dose level but there was no dose-related trend. **Developmental and reproductive toxicity:** The developmental toxicity of IPBC was assessed in pregnant Sprague-Dawley rats on gestation days six through 15 by oral administration of the test chemical at doses of 0, 20, 50, and 125 mg/kg/day. Maternal toxicity as reduced body weight gain during dosing was observed at the 125 mg/kg/day dose level. Developmental toxicity consisted of an increased incidence of skeletal abnormalities at the 125 mg/kg/day dose level. The maternal toxicity NOEL was determined to be 50 mg/kg/day, and the maternal toxicity LEL was determined to be 125 mg/kg/day, based on reduced body weight gain. The developmental toxicity NOEL was determined to be 50 mg/kg/day, and the developmental toxicity LEL was determined to be 125 mg/kg/day, based on incompletely ossified frontal skull bones and pelvic girdles. A 2-generation reproductive toxicity study was conducted in male and female Sprague- Dawley rats. IPBC technical was administered over two generations at doses of 0, 120, 300, and 750 ppm (0, 6, 15, and 37.5 mg/kg/day). Reduced body weight and food consumption was observed for P1 and F1 males during the premating period at the 37.5 mg/kg/day dose. A decreased mean live birth index was reported for P1 and F1 generations without an effect on viability and development of pups. No adverse effects on reproductive indices or mating performance were observed at any dose level. The parental toxicity NOEL was determined to be 15 mg/kg/day, and the parental toxicity LEL was determined to be 37.5 mg/kg/day, based on decreased body weight and food consumption during premating for P1 and F1 males, and decreased mean live birth index for the P1 and F1 generations. The reproductive toxicity Noel was determined to be 37.5 mg/kg/day, and the reproductive toxicity LEL was determined to be >37.5 mg/kg/day. **Mutagenicity:** In a mutagenicity study, IPBC technical was tested for the ability to cause mutations in ***Salmonella typhimurium*** strains TA 1535, TA 1537, TA 1538, TA 98, and TA 100. In the five strains used, IPBC was found to be non-mutagenic in the presence or absence of metabolic activation at the concentrations tested, 1-1000 jig/plate . In a micronucleus assay in mice, IPBC at doses of 200, 600, and 2000 mg/kg did not induce any significant increase of the PCE containing micronuclei from the treated mice when compared to that of the vehicle control mice. In two independent unscheduled DNA synthesis (UDS) assays in primary rat hepatocytes, eight doses of IPBC ranging from 3.0 to 13.5 ug/ml did not cause an appreciable increase in mean net nuclear grain counts. Doses >13.5 ug/ml were cytotoxic, supporting the conclusion that IPBC induced cytotoxicity but no genotoxicity in this assay. **Metabolism:** Based on the metabolite identification data, a scheme for metabolism of IPBC was proposed. According to this scheme, IPBC undergoes reductive dehalogenation followed by dealkylation to form the URM-9 and URM-10 metabolites. In addition, de-carboxylation following reductive dehalogenation yields carbon dioxide. Various other metabolites formed from dehalogenation are glucuronidated and constitute minor metabolites of IPBC..
ZINC OCTOATE	For aliphatic fatty acids (and salts) Acute oral (gavage) toxicity: The acute oral LD50 values in rats for both were greater than >2000 mg/kg bw Clinical signs were generally associated with poor condition following administration of high doses (salivation, diarrhoea, staining, piloerection and lethargy).There were no adverse effects on body weight in any study In some studies, excess test substance and/or irritation in the gastrointestinal tract was observed at necropsy. Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length According to several OECD test regimes the animal skin irritation studies indicate that the C6-10 aliphatic acids are severely irritating or corrosive, while the C12 aliphatic acid is irritating, and the C14-22 aliphatic acids generally are not irritating or mildly irritating. Human skin irritation studies using more realistic exposures (30-minute,1-hour or 24-hours) indicate that the aliphatic acids have sufficient, good or very good skin compatibility. Animal eye irritation studies indicate that among the aliphatic acids, the C8-12 aliphatic acids are irritating to the eye while the C14-22 aliphatic acids are not irritating. Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes. Dermal absorption: The in vitro penetration of C10, C12, C14, C16 and C18 fatty acids (as sodium salt solutions) through rat skin decreases with increasing chain length. At 86.73 ug C16/cm2 and 91.84 ug C18/cm2, about 0.23% and less than 0.1% of the C16 and C18 soap solutions is absorbed after 24 h exposure, respectively. Sensitisation: No sensitisation data were located.

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Repeat dose toxicity:
Repeated dose oral (gavage or diet) exposure to aliphatic acids did not result in systemic toxicity with NOAELs greater than the limit dose of 1000 mg/kg bw. .
Mutagenicity
Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo
Carcinogenicity
No data were located for carcinogenicity of aliphatic fatty acids.
Reproductive toxicity
No effects on fertility or on reproductive organs, or developmental effects were observed in studies on aliphatic acids and the NOAELs correspond to the maximum dose tested. The weight of evidence supports the lack of reproductive and developmental toxicity potential of the aliphatic acids category.

Given the large number of substances in this category, their closely related chemical structure, expected trends in physical chemical properties, and similarity of toxicokinetic properties, both mammalian and aquatic endpoints were filled using read-across to the closest structural analogue, and selecting the most conservative supporting substance effect level.
Structure-activity relationships are not evident for the mammalian toxicity endpoints.That is, the low mammalian toxicity of this category of substances limits the ability to discern structural effects on biological activity. Regardless, the closest structural analogue with the most conservative effect value was selected for read across. Irritation is observed for chain lengths up to a cut-off" at or near 12 carbons).
Metabolism:
The aliphatic acids share a common degradation pathway in which they are metabolized to acetyl-CoA or other key metabolites in all living systems. Common biological pathways result in structurally similar breakdown products, and are, together with the physico-chemical properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.
Differences in metabolism or biodegradability of even and odd numbered carbon chain compounds or saturated/ unsaturated compounds are not expected; even-and odd-numbered carbon chain compounds, and the saturated and unsaturated compounds are naturally occurring and are expected to be metabolized and biodegraded in the same manner.
The acid and alkali salt forms of the homologous aliphatic acid are expected to have many similar physicochemical and toxicological properties when they become bioavailable; therefore,data read across is used for those instances where data are available for the acid form but not the salt, and vice versa. In the gastrointestinal tract, acids and bases are absorbed in the undissociated (non-ionised) form by simple diffusion or by facilitated diffusion. It is expected that both the acids and the salts will be present in (or converted to) the acid form in the stomach. This means that for both aliphatic acid or aliphatic acid salt,the same compounds eventually enter the small intestine, where equilibrium, as a result of increased pH, will shift towards dissociation (ionised form).
Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated
Note that the saturation or unsaturation level is not a factor in the toxicity of these substances and is not a critical component of the read across process..
Toxicokinetics:
The turnover of the [14C] surfactants in the rat showed that there was no significant difference in the rate or route of excretion of 14C given by intraperitoneal or subcutaneous administration. The main route of excretion was as 14CO2 in the expired air at 6 h after administration. The remaining material was incorporated in the body. Longer fatty acid chains are more readily incorporated than shorter chains. At ca. 1.55 and 1.64 mg/kg bw, 71% of the C16:0 and 56% of the C18:0 was incorporated and 21% and 38% was excreted as 14CO2, respectively.

Glycidyl fatty acid esters (GEs), one of the main contaminants in processed oils, are mainly formed during the deodorisation step in the refining process of edible oils and therefore occur in almost all refined edible oils. GEs are potential carcinogens, due to the fact that they readily hydrolyze into the free form glycidol in the gastrointestinal tract, which has been found to induce tumours in various rat tissues. Therefore, significant effort has been devoted to inhibit and eliminate the formation of GEs
GEs contain a common terminal epoxide group but exhibit different fatty acid compositions. This class of compounds has been reported in edible oils after overestimation of 3-monochloropropane-1,2-diol (3-MCPD) fatty acid esters analysed by an indirect method , 3-MCPD esters have been studied as food processing contaminants and are found in various food types and food ingredients, particularly in refined edible oils. 3-Monochloropropane-1,2-diol (3-MCPD) and 2-monochloropropane-1,3-diol (2-MCPD) are chlorinated derivatives of glycerol (1,2,3-propanetriol). 3- and 2-MCPD and their fatty acid esters are among non-volatile chloropropanols, Glycidol is associated with the formation and decomposition of 3- and 2-MCPD. It forms monoesters with fatty acids (GE) during the refining of vegetable oils. Chloropropanols are formed in HVP during the hydrochloric acid-mediated hydrolysis step of the manufacturing process. In food production, chloropropanols form from the reaction of endogenous or added chloride with glycerol or acylglycerol.
Although harmful effects on humans and animals have not been demonstrated, the corresponding hydrolysates, 3-MCPD and glycidol, have been identified as rodent genotoxic carcinogens, ultimately resulting in the formation of kidney tumours (3-MCPD) and tumours at other tissue sites (glycidol). Therefore, 3-MCPD and glycidol have been categorised as ‘‘possible human carcinogens (group 2B) and "probably carcinogenic to humans (group 2A), respectively, by the International Agency for Research on Cancer (IARC).
Diacylglyceride (DAG) based oils produced by one company were banned from the global market due to "high levels" of GEs. Several reports have also suggested that a bidirectional transformation process may occur not only between glycidol and 3-MCPD but also their esterified forms in the presence of chloride ions. The transformation rate of glycidol to 3-MCPD was higher than that of 3-MCPD to glycidol under acidic conditions in the presence of chloride ion.
Precursors of GEs in refined oils have been identified as partial acylglycerols, that is, DAGs and monoacylglycerides (MAGs); however, whether they also originate from triacylglycerides (TAGs) is still a topic of controversial debates. Several authors noted that pure TAGs were stable during heat treatment (such as 235 deg C) for 3 h and were therefore not involved in the formation of GEs. However, experimental results have shown that small amounts of GEs are present in a heat-treated oil model consisting of almost 100% TAGs. The formation of GEs from TAGs can be attributed to the pyrolysis of TAGs to DAGs and MAGs. In contrast, 3-MCPD esters in refined oils can be obtained from TAG . Presently, the mechanism for the formation of GE intermediates and the relationship between GEs and 3-MCPD esters are still unknown.

Fatty acid salts are of low acute toxicity. Their skin and eye irritation potential is chain length dependent and decreases with increasing chain length - they are poorly absorbed through the skin nor are they skin sensitisers. The available repeated dose toxicity data demonstrate the low toxicity of the fatty acids and their salts. Also, they are not considered to be mutagenic,

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	genotoxic or carcinogenic, and are not reproductive or developmental toxicants. Accidental ingestion of fatty acid salt containing detergent products is not expected to result in any significant adverse health effects. This assessment is based on toxicological data demonstrating the low acute oral toxicity of fatty acid salts and the fact that not a single fatality has been reported in the UK following accidental ingestion of detergents containing fatty acid salts. Also in a report published by the German Federal Institute for Health Protection of Consumers and Veterinary Medicine, detergent products were not mentioned as dangerous products with a high incidence if poisoning. The estimated total human exposure to fatty acid salts, from the different exposure scenarios for the handling and use of detergent products containing fatty acid salts, showed a margin of exposure (MOE) of 258,620. This extremely large MOE is large enough to be reassuring with regard to the relatively small variability of the hazard data on which it is based. Also, in the UK, the recommended dietary fatty acid intake by the Department of Health is about 100 g of fatty acids per day or 1.7 g (1700 mg) of fatty acids per kilogram body weight per day. This exposure is several orders of magnitude above that resulting from exposure to fatty acid salts in household cleaning products. Based on the available data, the use of fatty acid salts in household detergent and cleaning products does not raise any safety concerns with regard to consumer For fatty acids, C8-10, zinc salts (CAS RN: 91051-00-2) Repeat dose toxicity: Under the test conditions, NOEL of the test material in rats was determined to be 3,000 ppm (approximately equivalent to 234 mg/kg/day in male rats and 243 mg/kg/day in female rats) An inhalation study was conducted to evaluate the low-level exposures together with occasional intense exposures of ultrafine test material particles in guinea pigs. Under the test conditions, Exposures to 2.7 mg/ m3, using the same 3 hr/ day, 5 day time frame, did not alter any parameters measured Under the test conditions, the short peaks occurring during normal low-level exposures can induce rapid pulmonary functional changes and greater extent of pulmonary damage and oedema. Genetic toxicity: in vivo A study was conducted to determine the potential genotoxicity of the test material using chromosomal aberration assay. Non-inbred female white rats were exposed to the test material aerosols at a concentration of 0.5 and 0.1 mg/m3 continuously for 5 months. Statistically significant increase in the frequency of damaged cells, primarily hyperdiploid cells were observed at both tested concentration. Toxicity to reproduction: A study was conducted to evaluate the reproductive toxicity potential of test material in rats for two generations. Under the test conditions, administration of test material to adult male and female rats throughout maturation, mating, gestation and early lactation resulted in significant effects on adults and offspring at 30 and 15 mg/kg/d. Although effects were seen at 7.5 mg/kg/d, these were considered to be toxicologically non significant and is therefore considered to be the "No Observed Adverse Effect Level" (NOAEL). * REACh Dossier
C.I. PIGMENT WHITE 6	Substance has been investigated as a mutagen, tumorigen and primary irritant. 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 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.

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	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.
TRIGLYCIDYL ISOCYANURATE	* TGIC Full Public Report: NICNAS (Australia) April 1994; ** [Manufacturer] 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. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative. for 1,2-butylene oxide (ethyloxirane): Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic When 14C-labelled a -TGIC was administrated to rabbits by stomach tube, no parent drug was detected in plasma. Plasma concentration of metabolites were lower compared to those observed following i.v. administration. In a study in mice at least 17% of the administered dose was absorbed within 24 hours. TGIC was distributed to the liver, stomach and testes (the only tissues studied). Eight hours after treatment no free TGIC was detected. **Metabolism:** TGIC metabolism seem to involve hydrolysis catalysed by microsomal epoxide hydrolase. In a recently conducted study, induction of epoxide hydrolase activity in rat livers was associated with increased hydrolysis of TGIC. However, this study only examined oral and intraperitoneal administration and did not consider dermal and inhalational exposure. alpha -TGIC was metabolised in vitro by rat liver, but not lung, microsomal preparations by an NADPH-independent pathway. Epoxide hydrolysis metabolites were detected in the microsomal incubations, and cyclohexene oxide, a known inhibitor of microsomal epoxide hydrolases, inhibited alpha -TGIC metabolism. Epoxide hydrolase activity in some human tissues may be higher than in rodent tissues. There is considerable variation in epoxide hydrolase activity between tissues and also significant (approximately 100-fold) interindividual variation of epoxide hydrolase activity in humans. **Excretion:** When 14C-labelled a -TGIC was administrated to rabbits by stomach tube, no parent drug was detected in plasma. Twenty-four-hour urinary recovery of radioactivity was about 30%. When humans were given i.v. administration of a -TGIC in doses up to 500 mg/m2 (= 15 mg/kg bw, calculated by using a standard weight of 60 kg and a standard surface area of 1.85 m2), less than 1 % alpha -TGIC is recovered in 24-hour urine. In a phase I anticancer clinical trial with alpha -TGIC, i.v. infusion of 2000 mg/m2 (= 62 mg/kg bw, calculated) administered over 2-3 hours was measured during the study to correspond to a plasma concentration of about 1mg/ml. When 14C-labelled a -TGIC is administrated to rabbits by i.v. infusion, twenty-four-hour urinary recovery of parent drug is < 1%, while urinary recovery of 14C total radioactivity is 60 to 70%. **Half-life:** Rapid plasma elimination (t½< 5min.) and total body clearance (about 5 litres/min.) are observed following i.v. administration of alpha -TGIC to humans in doses up to 500 mg/m2 (= 15 mg/kg, calculated). When 14C-labelled alpha -TGIC is administrated to rabbits by i.v. infusion, plasma disappearance of parent drug is very rapid (t½< 5min.), while metabolites in the plasma are eliminated at a much slower rate (t½> 60 min.). **Toxicological mechanisms:** The cytotoxicity of TGIC is probably related to the alkylating capacity of the epoxide moieties . alpha -TGIC was shown to alkylate a model compound 4-(p-nitrobenzyl)pyridine. E. coli strains defective in UV repair function were much more sensitive to a -TGIC than were the nondefective strains, suggesting that DNA may be the target of drug action. However, no in vitro interaction between a -TGIC and DNA or its components could be detected under physiological conditions by using a variety of biochemical and physicochemical techniques. Two in vivo studies have demonstrated that TGIC is capable of covalently binding to DNA in mouse liver, stomach and testis tissues following oral administration and in rat liver tissue following intraperitoneal or oral administration. **Reproductive and developmental effects:** In a dominant lethal test, technical grade TGIC (in arachid oil) was administered by single gavage at doses of 0, 160 and 480 mg/kg b.w. to groups of 20 male Tif MAGf(SPF) mice. These mice were mated over three periods of 6 days to 40 female mice per dose group. The female mice were replaced at the end of each period. Females were killed on day 14 of gestation and the

Continued...

numbers of live and dead foetuses and foetal resorptions were noted. Females mated to males given 480 mg/kg of TGIC during the first period showed a significant increase in the number of embryonic deaths, compared with the negative control. No increase was seen in the females mated in the second and third periods at the same dose, nor in the females in the other treated groups.
In a second dominant lethal test, technical grade TGIC (in peanut oil) was administered by single gavage at doses of 0, 138, 275 and 550 mg/kg b.w. to groups of 20 male ICR mice. These mice were mated over three periods of 5 days to 40 female mice per dose group. The female mice were replaced at the end of each period. No significant difference was observed in the number of embryonic deaths in test groups compared to the negative control.
In a third dominant lethal test, 10% TGIC in powder coating (doses of 0, 100, 1000, or 1700 mg/m3) was administered by whole body inhalational exposure to dust for six hours per day for five consecutive days to 30 male CD-1 mice per group. Following treatment each male was mated to two virgin females for eight weekly periods with the females being replaced at the end of each period. No increase in embryonic deaths was observed except in the positive control group and therefore TGIC did not induce heritable dominant lethal mutations under the conditions of the experiments.
In a fourth dominant lethal test, technical grade TGIC (doses of 0, 2.5, 10, or 50 mg/m3) was administered by whole body inhalational exposure to dust for six hours per day for five consecutive days to 30 male CD-1 mice per group. Following treatment each male was mated to two virgin females for eight weekly periods with the females being replaced at the end of each period. TGIC did not induce heritable dominant lethal mutations. There was a slight increase in the number of non-viable implants and early resorptions in the third mating week, but this was not statistically significant. The study showed reduced fertility in males at 10 and 50 mg/m3 as seen by reduced number of males impregnating females in some of the mating weeks and a non-significant ten percent reduction in testes weight in the 50 mg/m3 group. The reductions in fertility were consistent with an effect on mature sperm, maturing spermatids and Type B spermatogonia at the 50 mg/m3 level and with Type B spermatogonia at the 10 mg/m3 level.
In a 13-week toxicity/fertility study, groups of 10 male rats were given diets containing 0, 10, 30, or 100 ppm (0, 0.5, 1.5 or 5 mg/kg b.w., calculated) TGIC. This study followed a preliminary 19-day range-finding investigation in which signs of toxicity were observed in animals administered diets containing 160 or 640 ppm TGIC. In the full study after 64 days of treatment, each male was placed with two females until mating occurred. The females were then allocated to two subgroups (caesarean or normal delivery) on day 19 of pregnancy. Females from the caesarean group were killed on day 20 of pregnancy and the ovaries and uterus examined . The other group was allowed to deliver normally and the pups were examined for clinical signs and development. Between 22 and 25 days postpartum, the females in the normal delivery group were sacrificed and examined. In males at autopsy, all organs were examined in the high-dose and control groups. No exposure-related clinical effects or death were observed. Body weight gain was slightly lower over the first 6 weeks in animals from the 100 ppm test group. A dose-related reduction in the number of spermatozoa was noted but the spermatozoa viability was unchanged. No exposure-related infertility was noted in males, and no effects on embryonic and pup development were observed in the offspring. The highest concentration used in this study was not a maximum tolerated dose.
Mutagenic and genotoxic effects:
In vitro tests
Technical grade TGIC (dissolved in DMSO) was positive in two Ames tests using five strains (TA1535, TA1538, TA1537, TA98 and TA100) of Salmonella typhimurium when tested both with and without metabolic activation systems.
When tested in mammalian cells, technical grade TGIC (dissolved in DMSO) was positive in the mouse lymphoma assay with and without metabolic activation systems, in chromosome aberration and sister chromatid exchange tests in Chinese hamster ovary cells with and without metabolic activation systems, and for unscheduled DNA synthesis in rat hepatocytes. Technical grade TGIC dissolved in DMSO also tested positive in a chromosome aberration assay in Chinese hamster lung cells without metabolic activation systems but was negative with metabolic activation. A reduction in the response to treatment was noted in the mouse lymphoma assay and in the two assays in Chinese hamster ovary cells when metabolic activation was present. TGIC was negative in two cell transformation assays in mouse embryo fibroblasts.
In human cells, technical grade TGIC tested negative in a chromosomal aberrations assay in human lymphocyte cultures (TGIC doses between 0.063 and 10 mg/ml) and for unscheduled DNA synthesis in human fibroblast cultures (TGIC doses between 2.7 and 400 mg/ml) without metabolic activation systems.
In vivo tests.
Technical grade TGIC was shown to be clastogenic in a nucleus anomaly test in Chinese hamsters. TGIC (in arachid oil) was administered by gavage to groups of three animals of each sex at dose levels of 0, 140, 280 or 560 mg/kg b.w./day for two days. The animals were sacrificed 24 hours after the second dose and femoral bone marrow samples were taken. One thousand bone marrow cells were scored per animal. Nuclear anomalies for the intermediate and high dose groups were significantly different from the negative control.
Two studies were conducted to determine the ability of technical grade TGIC to induce sister chromatid exchanges (SCEs) in the bone marrow cells of Chinese hamsters. In each study, TGIC was suspended in arachid oil and administered by gavage. Twenty-five cells per animal were scored for SCEs. In one study, four animals of each sex per dose were treated with TGIC at dose levels of 0, 35, 70 and 140 mg/kg b.w.. In this study, no increases in the number of SCEs were observed. In the second study, groups of two animals per sex were treated with TGIC at doses of 0, 140, 280 and 560 mg/kg b.w. and a dose-related positive effect was observed.
Four oral and four inhalational studies have been conducted to determine chromosomal aberrations in mouse germ cells in animals exposed to technical grade TGIC in vivo.
In tree chromosomal aberration studies, male mice were dosed orally with TGIC by gavage on five consecutive days in doses ranging between 0 and 350 mg/kg b.w. Chromosomal aberrations in the spermatogonia were observed in a dose-related manner starting at about 30 mg/kg b.w. Cytotoxicity was first observed at 57.5 mg/kg b.w.
In another chromosomal aberration study, groups of 15 male mice were given TGIC (in arachid oil) by gavage at dose levels of 0, 32 or 96 mg/kg b.w. on days 0, 2, 3, 5 and 9. Animals were killed 3 days after the final dose and tested for chromosomal aberrations in their spermatocytes. The results of this study were negative.
In an inhalational chromosomal aberration study, groups of 10 male CD-1 mice were whole body exposed to technical grade TGIC at concentrations of 0, 2.5, 10 and 50 mg/m3 for 6 hours/day for five days. The particle size range of TGIC was 2.5 to 3.5 µm. Animals were killed six hours after the end of the last exposure. The results of this study were inconclusive mainly because statistical analysis could not include the 10 and 50 mg/m3 groups due to a small number of animals in these groups with a sufficient number of scorable cells (>50 per animal). No statistically significant aberrations were found in the 2.5 mg/m3 group. The study suggests TGIC was cytotoxic to spermatogonial cells at doses of 10 and 50 mg/m3. However, the cytotoxic ratios were not calculated.
In a second inhalational chromosomal aberration study, groups of 10 CD-1 male mice were whole body exposed to atmospheres containing 0, 100, 1000 or 1700 mg/m3 powder coating containing 10 % TGIC for 6 hours/ day for five days. The animals were

Continued...

	killed six hours after the last exposure period. The test material significantly increased the number of chromosomal aberrations in spermatogonial cells of the animals exposed to 1700 mg/m3 powder coating but as for the above mentioned study, the number of animals with enough scorable cells was very low. **Carcinogenic effects:** 2.5% TGIC did not promote skin tumour formation in CF-1 mice painted twice weekly for 26 weeks. Four groups of 24 mice of each sex had a tumour initiating agent applied dermally to their shaved backs. Three weeks later (and twice weekly for the next 26 weeks), these mice were painted with either 2.5% TGIC, 2.5% of a tumour promoting agent, solvent or received no secondary treatment. After 27 weeks, the mice were killed and the skin from the treated areas was examined microscopically. Only mice who received the tumour promoting agent developed skin tumours. Of those mice exposed to TGIC, one female showed severe acanthosis and two males showed ulceration.
PHENOL/ FORMALDEHYDE POLYMER SODIUM SALT & BARIUM SULFATE & C.I. PIGMENT BLACK 26	No significant acute toxicological data identified in literature search.
PROPICONAZOLE & PERMETHRIN & 3-IODO-2-PROPYNYL BUTYL CARBAMATE & TRIGLYCIDYL ISOCYANURATE	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.
PROPICONAZOLE & PERMETHRIN	***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]***
PERMETHRIN & C.I. PIGMENT WHITE 6	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.

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

Shadowclad Ultra LOSP Treated Plywood

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

phenol/ formaldehyde polymer sodium salt

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

white spirit

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	0.277mg/l	2
LC50	96h	Fish	0.14mg/l	2
NOEC(ECx)	720h	Fish	0.02mg/l	2

tebuconazole

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	2.09-3.01mg/l	4
EC50	48h	Crustacea	2.1-3.94mg/L	4
NOEC(ECx)	672h	Crustacea	0.001mg/L	4
EC50	96h	Algae or other aquatic plants	1.45mg/L	4
LC50	96h	Fish	6.4mg/l	Not Available

Continued...

propiconazole	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.001mg/L	4
	EC50	48h	Crustacea	3.354-4.902mg/L	4
	EC50	96h	Algae or other aquatic plants	1.29mg/l	4
	NOEC(ECx)	48h	Fish	<0.001mg/L	4
	LC50	96h	Fish	5.3mg/l	Not Available
permethrin	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	<0.001mg/L	4
	EC50	48h	Crustacea	<0.001mg/L	4
	EC50	96h	Algae or other aquatic plants	0.068mg/L	4
	NOEC(ECx)	72h	Fish	<0.001mg/L	4
3-iodo-2-propynyl butyl carbamate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.022mg/L	2
	EC50	48h	Crustacea	0.04mg/L	5
	NOEC(ECx)	0.5h	Fish	<0.001mg/L	4
	LC50	96h	Fish	0.05-0.089mg/L	4
zinc octoate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	0.105mg/L	2
	EC10(ECx)	168h	Algae or other aquatic plants	0.003mg/L	2
	LC50	96h	Fish	0.112mg/L	2
barium sulfate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>1.15mg/l	2
	EC50	48h	Crustacea	32mg/L	2
	NOEC(ECx)	72h	Algae or other aquatic plants	>=1.15mg/l	2
	LC50	96h	Fish	>3.5mg/l	2
C.I. Pigment White 6	**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)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
	LC50	96h	Fish	1.85-3.06mg/l	4
triglycidyl isocyanurate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	72h	Algae or other aquatic plants	>29<30mg/l	2
	EC50	72h	Algae or other aquatic plants	>29<30mg/l	2
	LC50	96h	Fish	>77mg/l	2
C.I. Pigment Black 26	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	18mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	504h	Fish	0.52mg/l	2
	LC50	96h	Fish	0.05mg/l	2
Legend:	***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data***				

DO NOT discharge into sewer or waterways.

Persistence and degradability

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
tebuconazole	HIGH	HIGH
permethrin	HIGH	HIGH
3-iodo-2-propynyl butyl carbamate	HIGH	HIGH
zinc octoate	LOW	LOW
C.I. Pigment White 6	HIGH	HIGH
triglycidyl isocyanurate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
white spirit	HIGH (LogKOW = 5.01)
tebuconazole	LOW (LogKOW = 3.7)
permethrin	LOW (LogKOW = 7.4267)
3-iodo-2-propynyl butyl carbamate	LOW (LogKOW = 2.4542)
zinc octoate	LOW (LogKOW = 3.0334)
C.I. Pigment White 6	LOW (BCF = 10)
triglycidyl isocyanurate	LOW (LogKOW = 1.2052)

Mobility in soil

Ingredient	Mobility
tebuconazole	LOW (Log KOC = 20660)
permethrin	LOW (Log KOC = 178400)
3-iodo-2-propynyl butyl carbamate	LOW (Log KOC = 365.3)
zinc octoate	LOW (Log KOC = 25.62)
C.I. Pigment White 6	LOW (Log KOC = 23.74)
triglycidyl isocyanurate	LOW (Log KOC = 10)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material) ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.
The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.
Only dispose to the environment if a tolerable exposure limit has been set for the substance.
Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 Transport information

Labels Required

Continued...

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (UN): 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. Maritime transport in bulk according to IMO instruments

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
phenol/ formaldehyde polymer sodium salt	Not Applicable
white spirit	Not Applicable
tebuconazole	Not Applicable
propiconazole	Not Applicable
permethrin	Not Applicable
3-iodo-2-propynyl butyl carbamate	Not Applicable
zinc octoate	Not Applicable
barium sulfate	Not Applicable
C.I. Pigment White 6	Not Applicable
triglycidyl isocyanurate	Not Applicable
C.I. Pigment Black 26	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
phenol/ formaldehyde polymer sodium salt	Not Applicable
white spirit	Not Applicable
tebuconazole	Not Applicable
propiconazole	Not Applicable
permethrin	Not Applicable
3-iodo-2-propynyl butyl carbamate	Not Applicable
zinc octoate	Not Applicable
barium sulfate	Not Applicable
C.I. Pigment White 6	Not Applicable
triglycidyl isocyanurate	Not Applicable
C.I. Pigment Black 26	Not Applicable

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002512	Additives Process Chemicals and Raw Materials Carcinogenic Group Standard 2020

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

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

New Zealand Inventory of Chemicals (NZIoC)

Continued...

white spirit is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

tebuconazole is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods

propiconazole is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods

permethrin is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods

3-iodo-2-propynyl butyl carbamate is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

zinc octoate is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods
New Zealand Workplace Exposure Standards (WES)

barium sulfate is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

C.I. Pigment White 6 is found on the following regulatory lists

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

triglycidyl isocyanurate is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

C.I. Pigment Black 26 is found on the following regulatory lists

Continued...

New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

Additional Regulatory Information

Not Applicable

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantities
Not Applicable	Not Applicable

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
Not Applicable	Not Applicable	Not Applicable	Not Applicable	Not Applicable

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (tebuconazole)
Canada - DSL	No (tebuconazole; propiconazole; permethrin)
Canada - NDSL	No (phenol/ formaldehyde polymer sodium salt; white spirit; tebuconazole; propiconazole; permethrin; 3-iodo-2-propynyl butyl carbamate; zinc octoate; barium sulfate; C.I. Pigment White 6; triglycidyl isocyanurate; C.I. Pigment Black 26)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (phenol/ formaldehyde polymer sodium salt)
Japan - ENCS	No (phenol/ formaldehyde polymer sodium salt; C.I. Pigment Black 26)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (phenol/ formaldehyde polymer sodium salt)
USA - TSCA	TSCA Inventory 'Active' substance(s) (phenol/ formaldehyde polymer sodium salt; white spirit; 3-iodo-2-propynyl butyl carbamate; zinc octoate; barium sulfate; C.I. Pigment White 6; triglycidyl isocyanurate; C.I. Pigment Black 26); No (tebuconazole; propiconazole; permethrin)
Taiwan - TCSI	Yes
Mexico - INSQ	No (phenol/ formaldehyde polymer sodium salt)
Vietnam - NCI	No (phenol/ formaldehyde polymer sodium salt; zinc octoate)
Russia - FBEPH	No (phenol/ formaldehyde polymer sodium salt; propiconazole; zinc octoate; C.I. Pigment Black 26)
UAE - Control List (Banned/Restricted Substances)	No (phenol/ formaldehyde polymer sodium salt; white spirit; 3-iodo-2-propynyl butyl carbamate; zinc octoate; barium sulfate; C.I. Pigment White 6; triglycidyl isocyanurate; C.I. Pigment Black 26)
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	23/12/2025
Initial Date	23/12/2025

Continued...

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	23/12/2025	Hazards identification - 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- 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