Futurebuild LOSP Treated LVL and hyJOIST

Carter Holt Harvey LVL Ltd (Trading as Futurebuild LVL)

Chemwatch Hazard Alert Code: 1

Chemwatch: **4729-83**
Version No: **18.1**
Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Initial Date: **02/08/2006**
Revision Date: **05/12/2024**
Print Date: **09/09/2025**
L.GHS.NZL.EN.RISK.E

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

Product Identifier

Product name	Futurebuild LOSP Treated LVL and hyJOIST
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Used in residential, commercial and industrial construction, and fitments and/or general purpose building.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**Carter Holt Harvey LVL Ltd (Trading as Futurebuild LVL)**
Address	Private Bag 92108, Victoria Street West Auckland 1142 New Zealand
Telephone	+64 800 808 131
Fax	Not Available
Website	https://futurebuild.co.nz/
Email	info@futurebuild.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

Not 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	0	
Toxicity	0	
Body Contact	1	
Reactivity	0	
Chronic	0	

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

Classification [1]	Non hazardous ***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***
Determined by Chemwatch using GHS/HSNO criteria	Not Available ***LIMITED EVIDENCE**

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Label elements

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

Hazard statement(s)

Not Applicable

***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

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	>90	wood veneer
Not Available	<10	impregnation residuals, as
40798-65-0	^	phenol/ formaldehyde polymer sodium salt
107534-96-3	^	tebuconazole
60207-90-1	^	propiconazole
52645-53-1	^	permethrin
55406-53-6	^	3-iodo-2-propynyl butyl carbamate
136-53-8	^	2-ethylhexanoic acid, zinc salt
Not Available		In use, may generate wood dust softwood
Not Available		THIS REPORT IS FOR TREATED PRODUCT ONLY

Legend:	***1. Classified by Chemwatch; 2. Classification drawn from 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	▸ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations. If this product comes in contact with eyes: ▸ Wash out immediately with water. ▸ If irritation continues, seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	Brush off dust. In the event of abrasion or irritation of the skin seek medical attention.
Inhalation	▸ If dust is inhaled, remove from contaminated area. ▸ Encourage patient to blow nose to ensure clear passage of breathing.

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	▸ If irritation or discomfort persists seek medical attention.
Ingestion	▸ Hazard relates to dust released by sawing, cutting, sanding, trimming or other finishing operations. ▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid exposure to excessive heat and fire.

Advice for firefighters

Fire Fighting	Alert Fire Brigade and tell them location and nature of hazard. Use water delivered as a fine spray to control the fire and cool adjacent area.
Fire/Explosion Hazard	Wood products do not normally constitute an explosion hazard. - Mechanical or abrasive activities which produce wood dust, as a by-product, may present a severe explosion hazard if a dust cloud contacts an ignition source. - Hot humid conditions may result in spontaneous combustion of accumulated wood dust. - Partially burned or scorched wood dust can explode if dispersed in air. Combustible. Will burn if ignited.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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

SECTION 7 Handling and storage

Precautions for safe handling

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

Conditions for safe storage, including any incompatibilities

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

+	+	+	+	+	+	+

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

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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)	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

Ingredient	Original IDLH	Revised IDLH
phenol/ formaldehyde polymer sodium salt	Not Available	Not Available
tebuconazole	Not Available	Not Available
propiconazole	Not Available	Not Available
permethrin	Not Available	Not Available
3-iodo-2-propynyl butyl carbamate	Not Available	Not Available
2-ethylhexanoic acid, zinc salt	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls

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

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

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

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

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood - local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the

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	extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Individual protection measures, such as personal protective equipment	
Eye and face protection	When sawing, machining or sanding use - Safety glasses with side shields.
Skin protection	See Hand protection below
Hands/feet protection	‣ Protective gloves eg. Leather gloves or gloves with Leather facing ‣ Safety footwear
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. **OTHERWISE:** ‣ Overalls. ‣ Barrier cream. ‣ Eyewash unit.

Respiratory protection

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)

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

	Disposable respirator	Re-usable respirator	Powered respirator
All woodworking operations eg use of routers, lathes, planers, saws and vertical spindle moulders (VSMs)	Type P2 filter for low residual dust levels for lower risk woods such as pine Type P3 filter for higher residual dust levels such as when sanding (hand , disc, bobbin, pad etc.). Also for all work involving more toxic woods such as hard woods, Western red cedar and MDF	Type P2 filter fitted to either a half mask or full face mask of Class 1 or 2 Type P3 filter fitted to either a half mask or full face mask of Class 2 Note: A combined organic vapour filter Type A (organic), either Class 1 or 2, will provide protection against any formadehyde vapours present from MDF	Lightweight powered hood visor or helmet of Type TH1 equivalent protection to Type P2 filter Lightweight powered visor or helmet with Type TH2 equivalent to Type P3 filter
Changing dust collection bags on simple recirculating dust collectors in the workroom	Type P3 Filter	Type P3 filter fitted to either a half mask or full face mask of Class 2	Lightweight powered visor or helmet of Type TH2 equivalent to Type P3 filter
Entry into dust collection rooms/ vaults Entry into very dusty filter galleries for bag changing Work inside heavily contaminated ducts Ensure none of these are confined spaces (oxygen deficient atmosphere)	Disposable respirators not suitable	Type P3 filter fitted to full face mask of Class 2	Lightweight powered hood, visor or helmet of Type TH2 equivalent to Type P3 filter

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Plywood in all sizes, impregnated with liquid treatment; can give off white spirit odour. THIS CHEMWATCH REPORT IS FOR TREATED PRODUCT ONLY.

Physical state	Manufactured	**Relative density (Water = 1)**	0.4-0.8

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

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	Based on available data, the classification criteria are not met.
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	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
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	Not normally a hazard due to physical form of product. Generated dust may be discomforting
Ingestion	Ingestion of sawdust may cause nausea, abdominal pain, vomiting or diarrhoea. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments
Skin Contact	The dust is discomforting and mildly abrasive to the skin and may cause drying of the skin, which may lead to contact dermatitis.
Eye	The dust may produce eye discomfort causing transient smarting, blinking

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

	TOXICITY	IRRITATION
Futurebuild LOSP Treated LVL and hyJOIST	Not Available	Not Available
phenol/ formaldehyde polymer sodium salt	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
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**

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	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]

2-ethylhexanoic acid, zinc salt	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Inhalation (Rat) LC50: >5.7 mg/L4h[1]	Skin (Rodent - guinea pig): 18%
	Oral (Rat) LD50: 2043 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

Legend:	***1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances***

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 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.[

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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-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.
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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

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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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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. 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.
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 (CO2) 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): **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

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	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..
PHENOL/ FORMALDEHYDE POLYMER SODIUM SALT & 2-ETHYLHEXANOIC ACID, ZINC SALT	No significant acute toxicological data identified in literature search.
PROPICONAZOLE & PERMETHRIN & 3-IODO-2-PROPYNYL BUTYL CARBAMATE	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]***

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

Continued...

Toxicity

Futurebuild LOSP Treated LVL and hyJOIST

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

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

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

2-ethylhexanoic acid, zinc salt

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	49.3mg/l	2
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

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

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

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
tebuconazole	HIGH	HIGH
permethrin	HIGH	HIGH
3-iodo-2-propynyl butyl carbamate	HIGH	HIGH

Bioaccumulative potential

Continued...

Ingredient	Bioaccumulation
tebuconazole	LOW (LogKOW = 3.7)
permethrin	LOW (LogKOW = 7.4267)
3-iodo-2-propynyl butyl carbamate	LOW (LogKOW = 2.4542)

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)

SECTION 13 Disposal considerations

Waste treatment methods

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

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

Disposal Requirements

Not applicable as substance/ material is non hazardous.

SECTION 14 Transport information

Labels Required

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 Available
tebuconazole	Not Available
propiconazole	Not Available
permethrin	Not Available
3-iodo-2-propynyl butyl carbamate	Not Available
2-ethylhexanoic acid, zinc salt	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
phenol/ formaldehyde polymer sodium salt	Not Available
tebuconazole	Not Available

Continued...

Product name	Ship Type
propiconazole	Not Available
permethrin	Not Available
3-iodo-2-propynyl butyl carbamate	Not Available
2-ethylhexanoic acid, zinc salt	Not Available

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
Not Applicable	Not Applicable

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)

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)

2-ethylhexanoic acid, zinc salt is found on the following regulatory lists

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

Additional Regulatory Information

Not Applicable

Hazardous Substance Location

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

Continued...

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; tebuconazole; propiconazole; permethrin; 3-iodo-2-propynyl butyl carbamate; 2-ethylhexanoic acid, zinc salt)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (phenol/ formaldehyde polymer sodium salt)
Japan - ENCS	No (phenol/ formaldehyde polymer sodium salt)
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; 3-iodo-2-propynyl butyl carbamate; 2-ethylhexanoic acid, zinc salt); No (tebuconazole; propiconazole; permethrin)
Taiwan - TCSI	Yes
Mexico - INSQ	No (phenol/ formaldehyde polymer sodium salt)
Vietnam - NCI	No (phenol/ formaldehyde polymer sodium salt)
Russia - FBEPH	No (phenol/ formaldehyde polymer sodium salt; propiconazole; 2-ethylhexanoic acid, zinc salt)
UAE - Control List (Banned/Restricted Substances)	No (phenol/ formaldehyde polymer sodium salt; 3-iodo-2-propynyl butyl carbamate; 2-ethylhexanoic acid, zinc salt)
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	05/12/2024
Initial Date	02/08/2006

SDS Version Summary

Version	Date of Update	Sections Updated
17.1	23/12/2022	Classification review due to GHS Revision change.
18.1	05/12/2024	Composition / information on ingredients - Ingredients, Identification of the substance / mixture and of the company / undertaking - Supplier Information

Other information

Continued...

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

end of SDS