

# CHH Antisapstain Hylite NCF Treated Pine Timber

## Carter Holt Harvey Building Products

Chemwatch Hazard Alert Code: 2

Chemwatch: 7999-13

Version No: 4.1

Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Initial Date: 22/12/2025

Revision Date: 21/01/2026

Print Date: 25/02/2026

L.GHS.NZL.EN.RISK.E

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

#### Product Identifier

Product name	CHH Antisapstain Hylite NCF Treated Pine Timber
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	Timber for industrial or packaging applications. Use according to manufacturer's directions.
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#### Details of the manufacturer or importer of the safety data sheet

Registered company name	Carter Holt Harvey Building Products
Address	173 Captain Springs Rd Onehunga Auckland 1061 New Zealand
Telephone	0800 746 399
Fax	Not Available
Website	<a href="https://chhwoodproducts.co.nz/">https://chhwoodproducts.co.nz/</a>
Email	info@chhwoodproducts.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

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 <i>*LIMITED EVIDENCE</i>
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	6.3A, 6.4A, 6.7B, 6.1E (respiratory tract irritant) <i>*LIMITED EVIDENCE</i>

#### Label elements

Hazard pictogram(s)	
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Signal word	Warning
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**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) 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.
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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	>99	wood
Not Available	<1	Impregnation Residuals
8001-54-5	^	<u>benzalkonium chloride</u>
68439-50-9	^	<u>alcohols C12-14 ethoxylated</u>
67564-91-4	^	<u>fenpropimorph</u>
60207-90-1	^	<u>propiconazole</u>
55406-53-6	^	<u>3-iodo-2-propynyl butyl carbamate</u>
55965-84-9	^	<u>5-chloro-2-methyl-4-isothiazolin-3-one</u>

**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**

Continued...

**Description of first aid measures**

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Wash out immediately with fresh running water.</li> <li>▶ 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.</li> <li>▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> <li>▶ Generally not applicable.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> <li>▶ Generally not applicable.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor, without delay.</li> <li>▶ Generally not applicable.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ Generally not applicable.</li> </ul>

**Indication of any immediate medical attention and special treatment needed**

Treat symptomatically.

**SECTION 5 Firefighting measures****Extinguishing media**

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

**Special hazards arising from the substrate or mixture**

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

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water courses.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul> <p>Slight hazard when exposed to heat, flame and oxidisers.</p>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Combustible.</li> <li>▶ Slight fire hazard when exposed to heat or flame.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> <li>▶ May emit acrid smoke.</li> <li>▶ Mists containing combustible materials may be explosive.</li> </ul> <p>Combustion products include:</p> <ul style="list-style-type: none"> <li>▶ carbon monoxide (CO)</li> <li>▶ carbon dioxide (CO<sub>2</sub>)</li> </ul> <p>hydrogen iodide nitrogen oxides (NO<sub>x</sub>)</p> <ul style="list-style-type: none"> <li>▶ other pyrolysis products typical of burning organic material.</li> </ul> <p>May emit poisonous fumes. May emit corrosive fumes.</p> <p>Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place.</p> <p>Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.</p>

## 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

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Secure load if safe to do so.</li> <li>▶ Bundle/collect recoverable product.</li> <li>▶ Collect remaining material in containers with covers for disposal.</li> </ul>
<b>Major Spills</b>	<ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Wear protective clothing, safety glasses, dust mask, gloves.</li> <li>▶ Secure load if safe to do so. Bundle/collect recoverable product.</li> <li>▶ Use dry clean up procedures and avoid generating dust.</li> <li>▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use).</li> <li>▶ Water may be used to prevent dusting.</li> <li>▶ Collect remaining material in containers with covers for disposal.</li> <li>▶ Flush spill area with water.</li> </ul> <p>Minor hazard.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Control personal contact with the substance, by using protective equipment as required.</li> <li>▶ Prevent spillage from entering drains or water ways.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal.</li> <li>▶ Wash area and prevent runoff into drains or waterways.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

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

## SECTION 7 Handling and storage

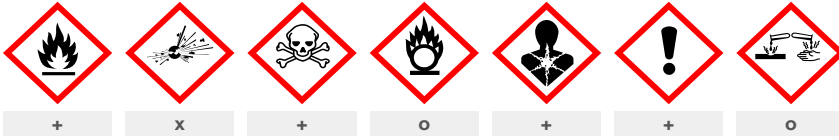
### Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ Avoid skin contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ <b>DO NOT</b> allow material to come in direct contact with human skin or eyes.</li> <li>▶ <b>DO NOT</b> allow material to come in contact with exposed food or food contact surfaces.</li> <li>▶ Suitable PPE must be worn at all times.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
<b>Other information</b>	<p>Store in the dark.</p> <ul style="list-style-type: none"> <li>▶ Store away from incompatible materials.</li> </ul>

### Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<p>Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler.</p> <ul style="list-style-type: none"> <li>▶ Glass container is suitable for laboratory quantities</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid reaction with oxidising agents</li> </ul>

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X — Must not be stored together

O — 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)	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

**MATERIAL DATA**

Airborne particulate or vapour must be kept to levels as low as is practicably achievable given access to modern engineering controls and monitoring hardware. Biologically active compounds may produce idiosyncratic effects which are entirely unpredictable on the basis of literature searches and prior clinical experience (both recent and past).

**Exposure controls**

<b>Appropriate engineering controls</b>	<p>Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use.</p> <p>Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.</p> <p>Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation.</p> <p>HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours.</p> <p>Barrier protection or laminar flow cabinets should be considered for laboratory scale handling.</p> <p>A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg.</p> <p>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.</p> <p>Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies.</p> <p>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.</p> <p>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.</p>										
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	<p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table>	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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	<p>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</p>										

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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

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

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.
- ▶ 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].

No special equipment required due to the physical form of the product.

#### Skin protection

See Hand protection below

#### Hands/feet protection

- ▶ Wear chemical protective gloves, e.g. PVC.
- ▶ Wear safety footwear or safety gumboots, e.g. Rubber
- ▶ 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.

No special equipment required due to the physical form of the product.

#### 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.
- ▶ Ensure there is ready access to an emergency shower.
- ▶ For Emergencies: Vinyl suit
- ▶ Overalls.
- ▶ P.V.C apron.
- ▶ Barrier cream.
- ▶ Skin cleansing cream.
- ▶ Eye wash 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(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

Respiratory protection not normally required due to the physical form of the product.

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

<b>Appearance</b>	Clear on air dried sawn timber in all sizes, envelope treatment with liquid preservative to give temporary protection against sap stain and mould. With potential for mild chemical odour; insoluble in water.		
<b>Physical state</b>	Manufactured	<b>Relative density (Water = 1)</b>	Not Available
<b>Odour</b>	Slight chemical	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	>200
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature (°C)</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Applicable
<b>Initial boiling point and boiling range (°C)</b>	Not Applicable	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	Not Available	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Applicable	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Applicable	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Applicable
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Applicable	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Applicable
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available
<b>Heat of Combustion (kJ/g)</b>	Not Available	<b>Ignition Distance (cm)</b>	Not Available
<b>Flame Height (cm)</b>	Not Available	<b>Flame Duration (s)</b>	Not Available
<b>Enclosed Space Ignition Time Equivalent (s/m<sup>3</sup>)</b>	Not Available	<b>Enclosed Space Ignition Deflagration Density (g/m<sup>3</sup>)</b>	Not Available

## SECTION 10 Stability and reactivity

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

## CHH Antisapstain Hylite NCF Treated Pine Timber

### SECTION 11 Toxicological information

#### Information on toxicological effects

<b>a) Acute Toxicity</b>	Based on available data, the classification criteria are not met.				
<b>b) Skin Irritation/Corrosion</b>	There is sufficient evidence to classify this material as skin corrosive or irritating.				
<b>c) Serious Eye Damage/Irritation</b>	There is sufficient evidence to classify this material as eye damaging or irritating				
<b>d) Respiratory or Skin sensitisation</b>	Based on available data, the classification criteria are not met.				
<b>e) Mutagenicity</b>	Based on available data, the classification criteria are not met.				
<b>f) Carcinogenicity</b>	There is sufficient evidence to classify this material as carcinogenic				
<b>g) Reproductivity</b>	Based on available data, the classification criteria are not met.				
<b>h) STOT - Single Exposure</b>	There is sufficient evidence to classify this material as toxic to specific organs through single exposure				
<b>i) STOT - Repeated Exposure</b>	Based on available data, the classification criteria are not met.				
<b>j) Aspiration Hazard</b>	Based on available data, the classification criteria are not met.				
<b>Inhaled</b>	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.				
<b>Ingestion</b>	The material has <b>NOT</b> been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.				
<b>Skin Contact</b>	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 is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.				
<b>Eye</b>	This material causes serious eye irritation.				
<b>Chronic</b>	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.				
<b>CHH Antisapstain Hylite NCF Treated Pine Timber</b>	<table border="1" style="width: 100%;"> <thead> <tr> <th style="width: 50%;">TOXICITY</th> <th style="width: 50%;">IRRITATION</th> </tr> </thead> <tbody> <tr> <td style="text-align: center;">Not Available</td> <td style="text-align: center;">Not Available</td> </tr> </tbody> </table>	TOXICITY	IRRITATION	Not Available	Not Available
TOXICITY	IRRITATION				
Not Available	Not Available				

## CHH Antisapstain Hylite NCF Treated Pine Timber

<b>benzalkonium chloride</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 1560 mg/kg <sup>[2]</sup>	Eye (Human): 50ug - Severe
	Oral (Rat) LD50: 240 mg/kg <sup>[2]</sup>	Eye (Primate - monkey): 2mg/24H - Severe
		Eye (Rodent - rabbit): 100ug
		Eye (Rodent - rabbit): 10mg - Mild
		Eye (Rodent - rabbit): 1mg/24H - Severe
		Skin (Human - woman): 0.1% - Moderate
		Skin (Human): 1%/24H
		Skin (Human): 1%/48H - Moderate
		Skin (Human): 150ug/3D (intermittent) - Mild
		Skin (Human): 3%/24H - Mild
		Skin (Human): 5pph/24H - Moderate
	Skin (Human): 6%/2W	
	Skin (Rodent - rabbit): 50mg/24H - Moderate	
<b>alcohols C12-14 ethoxylated</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>fenpropimorph</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >4000 mg/kg <sup>[2]</sup>	Not Available
	Inhalation (Rat) LC50: 2.9 mg/l4h <sup>[2]</sup>	
	Oral (Rat) LD50: >1400 mg/kg <sup>[2]</sup>	
<b>propiconazole</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >4000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation (Rat) LC50: >5.8 mg/L4h <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: 550 mg/kg <sup>[1]</sup>	
<b>3-iodo-2-propynyl butyl carbamate</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irreversible damage) <sup>[1]</sup>
	Inhalation (Rat) LC50: 0.63 mg/l4h <sup>[1]</sup>	Skin (Human): 0.3%/48H
	Oral (Rat) LD50: 1056 mg/kg <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>5-chloro-2-methyl-4-isothiazolin-3-one</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >1008 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irreversible damage) <sup>[1]</sup>
	Inhalation (Rat) LC50: 1.23 mg/l4h <sup>[2]</sup>	Skin (Human - woman): 0.01%
	Oral (Rat) LD50: 53 mg/kg <sup>[2]</sup>	Skin (Human): 0.01% - Severe
		Skin (Human): 0.1%/48H
		Skin: adverse effect observed (corrosive) <sup>[1]</sup>
		Skin: adverse effect observed (irritating) <sup>[1]</sup>

**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

**CHH Antisapstain Hylite NCF Treated Pine Timber**

conazoles areazole 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

Continued...

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.

#### BENZALKONIUM CHLORIDE

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.

#### For alkyldimethylbenzylammonium chlorides (ADMBAC):

Alkyldimethylbenzylammonium chlorides (ADMBAC) are included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC with the following classification: C8-18 ADMBAC are classified as Harmful (Xn) with the risk phrases R21/22 (Harmful in contact with skin and if swallowed) and Corrosive (C) with R34 (Causes burns) and (N) with R50 (Very toxic to aquatic organisms).

**Acute toxicity:** Absorption of these alkyldimethylbenzylammonium (ADMBAC) cationic surfactants through the skin is anticipated to be low. Different homologues of ADMBAC showed a moderate acute toxicity in experiments with rats and mice. The relationship between alkyl chain length and the acute toxicity of various ADMBAC homologues (C8 to C19) has been studied in mice. The studies indicated that chain lengths above C16 had a markedly lower acute toxicity and that even-numbered alkyl chain homologues appeared to be less toxic than odd-numbered carbon chains. It was suggested that the decrease in toxicity above C16 was due to a decreased water-solubility.

**Irritation studies:** ADMBAC is a skin irritant in animals at concentrations above 0.1% ). A nonspecified ADMBAC caused skin irritation and minor to moderate eye irritation at 0.625 and 1.25% concentrations. Inflammation of the eye and deterioration of vision occurred 3 days after change of soaking solution for a soft contact lens to a solution containing C8-18 ADMBAC.

**Sensitisation:** The sensitisation potential of ADMBAC has been examined in an experiment including 2,295 patients with suspected allergic contact dermatitis. Some of the patients (5.5%) showed positive reactions after exposure to 0.1% ADMBAC. These results were surprising as ADMBAC was not suspected to be a sensitiser. The high irritating potential of ADMBAC, even at low concentrations, could be an explanation of the observed results as the patch test reactions may have been false positives. However, another group of 2,806 patients with eczema was patch tested with 0.1% ADMBAC, and 2.13% of these patients appeared to be sensitised. Skin sensitisation was noted in patients patch tested with ADMBAC in aqueous solutions at 0.07 to 0.1% surfactant. However, there was no incidence of skin sensitisation in a population of normal individuals tested with 0.1% ADMBAC. This indicates that individuals with diseased skin may be at risk for sensitisation to ADMBAC.

**Genetic toxicity:** C16 ADMBAC did not induce transformation of the cells in an in vitro bioassay for carcinogenesis by using cultures of Syrian golden hamster embryo cells. The mutagenic potential of this surfactant was also examined by using Salmonella typhimurium strains - no mutagenic effects were seen). In other short-term genotoxicity assays (Salmonella/microsome assay) and rec-assay (bacterial DNA repair test) C16 ADMBAC was tested for ability to cause DNA damage in bacteria. None of the data indicated any mutagenic effects.

**Carcinogenicity:** Lifetime studies of ADMBAC were conducted in mice and rabbits that were treated with 8.5 to 17% surfactant dissolved in acetone or methanol. ADMBAC was applied repeatedly to the skin and ADMBAC caused ulceration, inflammations and scars in many animals, but no tumours.

**Developmental toxicity:** No embryotoxic activity was detected when C18 ADMBAC was applied topically to pregnant rats during the period of major organogenesis (day 6-15) at doses up to 6.6%, which was sufficient to cause adverse maternal reactions. Intravaginal instillation of ADMBAC (single doses up to 200 mg/kg) to pregnant rats on day one of the gestation caused abnormal foetal development and embryotoxicity

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljoministeriet (Danish Environmental Protection Agency)

#### For quaternary ammonium compounds (QACs):

Quaternary ammonium compounds (QACs) are cationic surfactants. They are synthetic organically tetra-substituted ammonium compounds, where the R substituents are alkyl or heterocyclic radicals. A common characteristic of these synthetic compounds is that one of the R's is a long-chain hydrophobic aliphatic residue

The cationic surface active compounds are in general more toxic than the anionic and non-ionic surfactants. The positively-charged cationic portion is the functional part of the molecule and the local irritation effects of QACs appear to result from the quaternary ammonium cation.

Due to their relative ability to solubilise phospholipids and cholesterol in lipid membranes, QACs affect cell permeability which may lead to cell death. Further QACs denature proteins as cationic materials precipitate protein and are accompanied by generalised tissue irritation.

It has been suggested that the experimentally determined decrease in acute toxicity of QACs with chain lengths above C16 is due to decreased water solubility.

In general it appears that QACs with a single long-chain alkyl groups are more toxic and irritating than those with two such substitutions,

The straight chain aliphatic QACs have been shown to release histamine from minced guinea pig lung tissue. However, studies with benzalkonium chloride have shown that the effect on histamine release depends on the concentration of the solution. When cell suspensions (11% mast cells) from rats were exposed to low concentrations, a decrease in histamine release was seen. When exposed to high concentrations the opposite result was obtained.

In addition, QACs may show curare-like properties (specifically benzalkonium and cetylpyridinium derivatives, a muscular paralysis with no involvement of the central nervous system. This is most often associated with lethal doses. Parenteral injections in rats, rabbits and dogs have resulted in prompt but transient limb paralysis and sometimes fatal paresis of the respiratory muscles. This effect seems to be transient.

From human testing of different QACs the generalised conclusion is obtained that all the compounds investigated to date exhibit similar toxicological properties.

**Long term/repeated exposure:**

**Inhalation:** A group of 196 farmers (with or without respiratory symptoms) were evaluated for the relationship between exposure to QACs (unspecified, exposure levels not given) and respiratory disorders by testing for lung function and bronchial responsiveness to histamine. After histamine provocation statistically significant associations were found between the prevalence of mild bronchial responsiveness (including asthma-like symptoms) and the use of QACs as disinfectant. The association seems even stronger in people without respiratory symptoms.

for acid mists, aerosols, vapours

Data from assays for genotoxic activity *in vitro* suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events *in vivo* in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH *in vivo* differ from exposures *in vitro* in that, *in vivo*, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than *in vitro*.

**ALCOHOLS C12-14  
ETHOXYLATED**

\* BASF Canada \*\* [Henkel CCINFO 1450373]

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing

Overall, alcohol alkoxyates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length  $C \geq 6$  may also contain shorter alkyl chains  $C < 6$ . It is not practical to quantify the proportion of shorter  $C < 6$  chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO<sub>2</sub>). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO<sub>2</sub>). The metabolism of C12 AE yields PEG, carboxylic acids, and CO<sub>2</sub> as metabolites. The LD<sub>50</sub> values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein *in vitro* and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form

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hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information *in vivo* and *in vitro* demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

**Skin absorption:** Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm<sup>2</sup>/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm<sup>2</sup>/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that

of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

**Metabolism:** The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

**Acute toxicity:** Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure.

Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

**Irritation:** The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

**Repeat dose toxicity:** Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats.

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

**Mutagenicity:** Mutagenicity studies have been conducted for several category members. All *in vitro* and *in vivo* studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

**Reproductive toxicity:** Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower

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molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGME is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

**Developmental toxicity:** The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

**FENPROPI MORPH**

ADI 0.003 mg/kg \* NOEL for rats 0.3, mice 3.0, dogs 3.2 mg/kg b.w. daily \* No carcinogenicity observed \*

The literature suggests that some morpholine fungicides demonstrate potential teratogenicity. Fenpropimorph has been associated with anasarca (excessive tissue fluid) in rats and cleft palate in rats and rabbits; tridemorph has been associated with cleft palate in rodents. The malformations and increase in postimplantation loss observed with dodemorph-acetate are considered serious responses. Furthermore, these responses in the rabbit occur at dose levels that do not demonstrate any maternal toxicity. The sensitivity of the foetus coupled with the seriousness of the effect indicates a high level of concern for prenatal toxicity.

The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.

Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

**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) \*

**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 antimetabolic 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 promoter properties. Carbamate pesticides may be converted to *N*-nitroso compounds. This was demonstrated in a great number of *in*

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*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<sub>2</sub>) 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 rabbit. 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/kg/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 pre-mating 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 pre-mating 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 µg/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 µg/ml did not cause an appreciable increase in mean net nuclear grain counts. Doses >13.5 µg/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.

## 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE

Considered to be the major sensitiser in Kathon CG (1) (1). Bruze et al - Contact Dermatitis 20: 219-39, 1989  
Based on laboratory and animal testing, exposure to the material may result in irreversible effects and mutations in humans. In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance.

Continued...

## CHH Antisapstain Hylite NCF Treated Pine Timber

	<p>Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration. Formaldehyde generators (releasers) are often used as preservatives. The maximum authorised concentration of free formaldehyde is 0.2% and must be labelled with the warning sign "contains formaldehyde" where the concentration exceeds 0.05%. The use of formaldehyde-releasing preservatives ensures that the level of free formaldehyde in the products is always low but sufficient to inhibit microbial growth - it disrupts metabolism to cause death of the organism. However there is a concern that formaldehyde generators can produce amines capable of causing cancers (nitrosamines) when used in formulations containing amines.</p> <p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>		
<b>CHH Antisapstain Hylite NCF Treated Pine Timber &amp; BENZALKONIUM CHLORIDE &amp; FENPROPIMORPH &amp; 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE</b>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p>		
<b>CHH Antisapstain Hylite NCF Treated Pine Timber &amp; 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE</b>	No significant acute toxicological data identified in literature search.		
<b>BENZALKONIUM CHLORIDE &amp; PROPIONAZOLE &amp; 3-iodo-2-propynyl butyl carbamate &amp; 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE</b>	<p>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.</p>		
<b>ALCOHOLS C12-14 ETHOXYLATED &amp; 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE</b>	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.		
<b>FENPROPIMORPH &amp; PROPIONAZOLE</b>	[ * <i>The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council</i> ]		
<b>Acute Toxicity</b>	✗	<b>Carcinogenicity</b>	✓
<b>Skin Irritation/Corrosion</b>	✓	<b>Reproductivity</b>	✗
<b>Serious Eye Damage/Irritation</b>	✓	<b>STOT - Single Exposure</b>	✓
<b>Respiratory or Skin sensitisation</b>	✗	<b>STOT - Repeated Exposure</b>	✗
<b>Mutagenicity</b>	✗	<b>Aspiration Hazard</b>	✗

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

## SECTION 12 Ecological information

## Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
<b>CHH Antisapstain Hylite NCF Treated Pine Timber</b>	Not Available	Not Available	Not Available	Not Available	Not Available

## CHH Antisapstain Hylite NCF Treated Pine Timber

	Endpoint	Test Duration (hr)	Species	Value	Source
benzalkonium chloride	EC50	72h	Algae or other aquatic plants	0.056mg/L	4
	EC50	48h	Crustacea	0.02mg/l	Not Available
	EC50	96h	Algae or other aquatic plants	0.056mg/L	4
	LC50	96h	Fish	0.31mg/l	Not Available
	EC50(ECx)	48h	Crustacea	0.02mg/l	Not Available
alcohols C12-14 ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.044mg/l	2
	EC50	48h	Crustacea	0.125mg/l	2
	NOEC(ECx)	504h	Crustacea	>=0.001<=0.107mg/L	2
LC50	96h	Fish	0.423mg/l	2	
fenpropimorph	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.002-0.012mg/L	4
	EC50	48h	Crustacea	1.605-3.676mg/L	4
	NOEC(ECx)	2256h	Fish	<0.001mg/L	2
LC50	96h	Fish	>1<2.2mg/l	2	
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	
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	
5-chloro-2-methyl-4-isothiazolin-3-one	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	0.172mg/l	1
	EC50	72h	Algae or other aquatic plants	0.018-0.026mg/L	4
	EC50	48h	Crustacea	4.71mg/l	1
	EC50	96h	Algae or other aquatic plants	0.03-0.13mg/L	4
LC50	96h	Fish	0.13-0.31mg/L	4	

**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

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

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
5-chloro-2-methyl-4-isothiazolin-3-one	HIGH	HIGH

**Bioaccumulative potential**

Ingredient	Bioaccumulation
benzalkonium chloride	LOW (LogKOW = 2.93)
alcohols C12-14 ethoxylated	HIGH (LogKOW = 5.96)
fenpropimorph	HIGH (LogKOW = 4.93)
3-iodo-2-propynyl butyl carbamate	LOW (LogKOW = 2.4542)
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (LogKOW = 0.0444)

**Mobility in soil**

Ingredient	Mobility
fenpropimorph	LOW (Log KOC = 26870)
3-iodo-2-propynyl butyl carbamate	LOW (Log KOC = 365.3)
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (Log KOC = 45.15)

**SECTION 13 Disposal considerations****Waste treatment methods**

Product / Packaging disposal	
	<ul style="list-style-type: none"> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Management Authority for disposal.</li> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Authority for disposal.</li> <li>▶ Bury or incinerate residue at an approved site.</li> <li>▶ Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>

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**

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
benzalkonium chloride	Not Applicable
alcohols C12-14 ethoxylated	Not Applicable
fenpropimorph	Not Applicable
propiconazole	Not Applicable
3-iodo-2-propynyl butyl carbamate	Not Applicable
5-chloro-2-methyl-4-isothiazolin-3-one	Not Applicable

**14.7.3. Transport in bulk in accordance with the IGC Code**

Product name	Ship Type
benzalkonium chloride	Not Applicable
alcohols C12-14 ethoxylated	Not Applicable
fenpropimorph	Not Applicable
propiconazole	Not Applicable
3-iodo-2-propynyl butyl carbamate	Not Applicable
5-chloro-2-methyl-4-isothiazolin-3-one	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.

**benzalkonium chloride 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)

**alcohols C12-14 ethoxylated 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

**fenpropimorph 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

**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)

#### 5-chloro-2-methyl-4-isothiazolin-3-one 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)

### 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 - AIIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (fenpropimorph; propiconazole)
Canada - NDSL	No (benzalkonium chloride; alcohols C12-14 ethoxylated; fenpropimorph; propiconazole; 3-iodo-2-propynyl butyl carbamate; 5-chloro-2-methyl-4-isothiazolin-3-one)
China - IECSC	No (fenpropimorph)
Europe - EINEC / ELINCS / NLP	No (benzalkonium chloride)
Japan - ENCS	No (fenpropimorph)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (fenpropimorph)
USA - TSCA	TSCA Inventory 'Active' substance(s) (alcohols C12-14 ethoxylated; 3-iodo-2-propynyl butyl carbamate; 5-chloro-2-methyl-4-isothiazolin-3-one); No (benzalkonium chloride; fenpropimorph; propiconazole)
Taiwan - TCSI	Yes
Mexico - INSQ	No (alcohols C12-14 ethoxylated; fenpropimorph)
Vietnam - NCI	Yes
Russia - FBEPH	No (fenpropimorph; propiconazole)
UAE - Control List (Banned/Restricted Substances)	No (benzalkonium chloride; alcohols C12-14 ethoxylated; fenpropimorph; 3-iodo-2-propynyl butyl carbamate; 5-chloro-2-methyl-4-isothiazolin-3-one)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory

Continued...

National Inventory	Status
	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	21/01/2026
Initial Date	22/12/2025

## SDS Version Summary

Version	Date of Update	Sections Updated
3.1	23/12/2025	Physical and chemical properties - Appearance, Hazards identification - Classification, Identification of the substance / mixture and of the company / undertaking - Use
4.1	21/01/2026	Physical and chemical properties - Appearance, Identification of the substance / mixture and of the company / undertaking - Supplier Information

## Other information

### Ingredients with multiple cas numbers

Name	CAS No
alcohols C12-14 ethoxylated	68439-50-9, 103819-01-8
fenpropimorph	67306-03-0, 67564-91-4
propiconazole	60207-90-1, 75881-82-2
5-chloro-2-methyl-4-isothiazolin-3-one	26172-55-4, 61840-41-3, 55965-84-9, 137086-87-4, 137662-59-0

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

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