

## Duroproof PU 600

Durotech Industries

Chemwatch Hazard Alert Code: 3

Chemwatch: 5245-60

Issue Date: 07/03/2017

Version No: 2.1.1.1

Print Date: 30/03/2017

Safety Data Sheet according to WHS and ADG requirements

L.GHS.AUS.EN

### SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

#### Product Identifier

Product name	Duroproof PU 600
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions. Chemical product for construction and industry.
--------------------------	--

#### Details of the supplier of the safety data sheet

Registered company name	Durotech Industries
Address	14 Essex Street Minto NSW 2566 Australia
Telephone	02 9603 1177
Fax	02 9475 5059
Website	www.durotechindustries.com.au
Email	accounts@durotechindustries.com.au

#### Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	0421 670 636
Other emergency telephone numbers	Not Available

### SECTION 2 HAZARDS IDENTIFICATION


#### Classification of the substance or mixture

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

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	Not Applicable
Classification [1]	Flammable Liquid Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Acute Aquatic Hazard Category 3, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

#### Label elements

GHS label elements	
--------------------	---

SIGNAL WORD **DANGER**

#### Hazard statement(s)

H227	Combustible liquid
H315	Causes skin irritation.
H318	Causes serious eye damage.
H317	May cause an allergic skin reaction.
H335	May cause respiratory irritation.
H412	Harmful to aquatic life with long lasting effects.

**Precautionary statement(s) Prevention**

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

**Precautionary statement(s) Response**

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

**Precautionary statement(s) Storage**

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.

**Precautionary statement(s) Disposal**

P501	Dispose of contents/container in accordance with local regulations.
------	---

**SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS****Substances**

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
471-34-1	10-30	<u>calcium carbonate</u>
9002-86-2	1-10	<u>polyvinyl chloride</u>
13463-67-7	1-10	<u>titanium dioxide</u>
1330-20-7	1-10	<u>xylylene</u>
8002-09-3	1-10	<u>pine oil</u>

**SECTION 4 FIRST AID MEASURES****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>▶ Immediately hold eyelids apart and flush the eye continuously with 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>▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</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> </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>▶ Prosthesis 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> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> <li>▶ Avoid giving milk or oils.</li> <li>▶ Avoid giving alcohol.</li> <li>▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.</li> </ul>

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

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

Treat symptomatically.

For acute or short term repeated exposures to xylene:

- ▶ Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- ▶ Pulmonary absorption is rapid with about 60-65% retained at rest.
- ▶ Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases ( $pO_2 < 50$  mm Hg or  $pCO_2 > 50$  mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

#### BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Methylhippu-ric acids in urine	1.5 gm/gm creatinine	End of shift	
	2 mg/min	Last 4 hrs of shift	

## 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>	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
-----------------------------	--

### 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>
<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> Combustion products include: <ul style="list-style-type: none"> <li>, carbon dioxide (CO<sub>2</sub>)</li> <li>, metal oxides</li> <li>, other pyrolysis products typical of burning organic material.</li> </ul> May emit poisonous fumes. May emit corrosive fumes.
<b>HAZCHEM</b>	Not Applicable

## SECTION 6 ACCIDENTAL RELEASE MEASURES

### Personal precautions, protective equipment and emergency procedures

See section 8

### Environmental precautions

See section 12

### Methods and material for containment and cleaning up

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▶ Remove all ignition sources.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>▶ Wipe up.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> </ul>
---------------------	--

<b>Major Spills</b>	<p>Moderate hazard.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <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 course.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</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.</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</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>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> <li>▶ Avoid all personal 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>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ When handling, <b>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.</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.</li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Store in a cool, dry, well-ventilated area.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>

### Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Metal can or drum</li> <li>▶ Packaging as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.</li> <li>▶ Avoid reaction with oxidising agents</li> </ul>

## SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

### Control parameters

#### OCCUPATIONAL EXPOSURE LIMITS (OEL)

#### INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p- isomers)	350 mg/m3 / 80 ppm	655 mg/m3 / 150 ppm	Not Available	Not Available

#### EMERGENCY LIMITS


Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
calcium carbonate	Limestone; (Calcium carbonate; Dolomite)	45 mg/m3	500 mg/m3	3,000 mg/m3
calcium carbonate	Carbonic acid, calcium salt	45 mg/m3	210 mg/m3	1,300 mg/m3
polyvinyl chloride	Polyvinyl chloride	3 mg/m3	33 mg/m3	200 mg/m3
titanium dioxide	Titanium oxide; (Titanium dioxide)	30 mg/m3	330 mg/m3	2,000 mg/m3
xylene	Xylenes	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
calcium carbonate	Not Available	Not Available
polyvinyl chloride	Not Available	Not Available
titanium dioxide	N.E. mg/m3 / N.E. ppm	5,000 mg/m3
xylene	1,000 ppm	900 ppm

pine oil	Not Available	Not Available
----------	---------------	---------------

**MATERIAL DATA**

**Exposure controls**

<p><b>Appropriate engineering controls</b></p>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. 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> <table border="1" data-bbox="363 645 1485 902"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="363 958 1485 1126"> <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> <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 extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)	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
Type of Contaminant:	Air Speed:																				
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)																				
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)																				
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)																				
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)																				
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																				
<p><b>Personal protection</b></p>																					
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ 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], [AS/NZS 1336 or national equivalent]</li> </ul>																				
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>																				
<p><b>Hands/feet protection</b></p>	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>• frequency and duration of contact,</li> <li>• chemical resistance of glove material,</li> <li>• glove thickness and</li> <li>• dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>• When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>• When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> </ul>																				

	<ul style="list-style-type: none"> <li>Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>Contaminated gloves should be replaced.</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.</li> <li>Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential</li> </ul> <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<ul style="list-style-type: none"> <li>Overalls.</li> <li>P.V.C. apron.</li> <li>Barrier cream.</li> <li>Skin cleansing cream.</li> <li>Eye wash unit.</li> </ul>
<b>Thermal hazards</b>	Not Available

## Recommended material(s)

### GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-**

**generated** selection:

Duroproof PU 600

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
TEFLON	C
VITON	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

**NOTE:** As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

\* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

## 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 / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	A-3 P2	-
100+ x ES	-	Air-line**	-

\* - Continuous-flow; \*\* - Continuous-flow or positive pressure demand

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)

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### Information on basic physical and chemical properties

<b>Appearance</b>	Concrete grey viscous liquid with a characteristic odour; does not mix with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	1.30
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available

## Duroproof PU 600

<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	>61 CC	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Combustible.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water (g/L)</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

## SECTION 10 STABILITY AND REACTIVITY

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>▶ Unstable in the presence of incompatible materials.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
<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

## SECTION 11 TOXICOLOGICAL INFORMATION

## Information on toxicological effects

<b>Inhaled</b>	<p>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.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p>
<b>Ingestion</b>	<p>Considered an unlikely route of entry in commercial/industrial environments</p> <p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p>
<b>Skin Contact</b>	<p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> <li>▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▶ produces significant, but moderate, 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.</li> </ul> <p>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.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
<b>Eye</b>	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
<b>Chronic</b>	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body 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.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</p> <p>An epidemiological study conducted with workers engaged in the production of chlorinated derivatives of polyvinyl chloride, showed an increased frequency of ischaemic heart disease, intermittent lameness and strokes when compared to the unexposed cohort.</p> <p>Chronic inhalation exposure to polyvinyl chloride dusts produce dyspnea, decreased pulmonary function, occupational asthma with cough and breathlessness, interstitial pneumonitis, pneumoconiosis with diffuse fibrosis, granulomatous tissue and secondary polyglobulia.</p> <p>Animal studies show exposure produces bronchiolitis, alveolitis, random lung lesions, and pathomorphological degenerative and hyperplastic inflammatory changes in the liver. Foetal mortality was also increased although malformations were not evident.</p> <p>In the presence of air, a number of common flavour and fragrance chemicals can form peroxides surprisingly fast. Antioxidants can in most cases minimise the oxidation.</p> <p>Fragrance terpenes are generally easily oxidised in air. Non-oxidised limonene, linalool and caryophyllene turned out to be very weak sensitizers, however after oxidation limonene hydroperoxide and linalool hydroperoxide are strong sensitizers. Of the patients tested 2.6% showed positive reaction to oxidised limonene, 1.3% to oxidised linalool, 1.1% to linalool hydroperoxide, 0.5% to oxidised caryophyllene, while testing with caryophyllene oxide and oxidised myrcene resulted in</p>



few positive patch tests. 2/3 of the patients reacting positive to oxidised terpenes had fragrance related contact allergy and/or positive history for adverse reactions to fragrances.

As well as the hydroperoxides produced by linalol, limonene and delta-3-carene other oxidation and resinification effects progressively causes other fairly major changes in essential oil quality over time. Autoxidation of fragrance terpenes contributes greatly to fragrance allergy, which emphasizes the need of testing with compounds that patients are actually exposed to and not only with the ingredients originally applied in commercial formulations.

Hydroperoxides of d-limonene are potent contact allergens when studied in guinea pigs. They may result when d-limonene is unstabilised against oxidation, or upon prolonged standing at room temperature and/ or upon exposure to light, or when stabiliser levels diminish. The two major hydroperoxides in auto-oxidised d-limonene, are cis- and trans- limonene-2-hydroperoxide (2-hydroperoxy-p-mentha-6,8-diene). In photo-oxidised d-limonene, they represent a minor fraction. Hydroperoxides may bind to proteins of the skin to make antigens either via a radical mechanism or after reactions to give epoxides. The cross-reactivity between the epoxide limonene-1,2-oxide, a potent contact allergen, and the hydroperoxides is NOT significant, indicating different mechanisms of sensitisation.

d-Limonene was considered to be weakly carcinogenic for the mouse fore-stomach epithelium, but not tumour producing. In 13-week and 2-year gavage-studies, male rats showed a range of compound-related kidney lesions including exacerbation of age-related nephropathy, mineralisation in the renal medulla, hyperplasia of the transitional epithelium overlying the renal papilla and proliferation of the renal tubular epithelium. Neoplasms were believed to be caused by progression to tubular cell hyperplasia to tubular cell adenomas and, with increasing size, to adenocarcinomas or carcinomas. The similarity of the nephrotoxicity caused by trichloroethylene and N-(4'-fluoro-4-biphenyl)acetamide, tris(2,3-dibromopropyl)phosphate in rats and the species specific nature of the response suggests that degeneration and necrosis of convoluted tubules may be associated with the accumulation of alpha-2u-globin (a2u-G). Since a2u-G is a species and gender-specific protein that is causal for both the cytotoxic and carcinogenic response in male rats, extrapolation of d-limonene carcinogenicity data from rat studies to other species (including humans) is probably not warranted. Humans do not synthesise a2u-G; they do however produce other related low molecular weight proteins capable of binding chemicals that cause a2u-G nephropathy in rats but this does not necessarily connote human risk. The Risk Assessment Forum of the USA EPA concluded;

- ▶ Male renal rat tumours arising as a result of a process involving a2u-G accumulation do not contribute to the qualitative weight-of-evidence that the chemical poses a human carcinogenic hazard. Such tumours are included in dose-response extrapolations for the estimation of human carcinogenic risk.
- ▶ If the chemical induces a2u-G accumulation in male rats, the associated nephropathy is not to be used as an end-point for determining non-carcinogenic hazard.

Peroxidisable terpenes and terpenoids should only be used when the level of peroxides is kept to the lowest practicable level, for instance by adding antioxidants at the time of production. Such products should have a peroxide value of less than 10 millimoles peroxide per liter. This requirement is based on the published literature mentioning sensitising properties when containing peroxides.

Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.

Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.

Xylene has been classed as a developmental toxin in some jurisdictions.

Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

Duroproof PU 600	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
calcium carbonate	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 0.75 mg/24h - SEVERE Skin (rabbit): 500 mg/24h-moderate
polyvinyl chloride	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
titanium dioxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation (rat) LC50: >2.28 mg/l/4hr <sup>[1]</sup>	Skin (human): 0.3 mg /3D (int)-mild *
	Inhalation (rat) LC50: >3.56 mg/l/4hr <sup>[1]</sup>	
	Inhalation (rat) LC50: >6.82 mg/l/4hr <sup>[1]</sup>	
	Inhalation (rat) LC50: 3.43 mg/l/4hr <sup>[1]</sup>	
	Inhalation (rat) LC50: 5.09 mg/l/4hr <sup>[1]</sup> Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	
xylene	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1700 mg/kg <sup>[2]</sup> Inhalation (rat) LC50: 5000 ppm/4hr <sup>[2]</sup>	Eye (human): 200 ppm irritant Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kg <sup>[2]</sup>	Eye (rabbit): 87 mg mild Skin (rabbit):500 mg/24h moderate
pine oil	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 5000 mg/kg <sup>[2]</sup> Oral (rat) LD50: 3200 mg/kg <sup>[2]</sup>	Skin (rabbit): 500 mg/24h-SEVERE

**Legend:** 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. \* Value obtained from manufacturer's SDS. Unless otherwise specified data

Continued...



extracted from RTECS - Register of Toxic Effect of chemical Substances

<b>CALCIUM CARBONATE</b>	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
<b>TITANIUM DIOXIDE</b>	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>For titanium dioxide:</p> <p>Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.</p> <p>Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.</p> <p>Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.</p> <p>Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.</p> <p><b>Animal carcinogenicity data</b></p> <p>Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.</p> <p>In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.</p> <p>Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.</p> <p>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.</p> <p><b>WARNING:</b> This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. * IUCLID</p>
<b>XYLENE</b>	Reproductive effector in rats
<b>PINE OIL</b>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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> <p>d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine.</p> <p>Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs.</p> <p>Renal tumours induced by limonene in male rats is thought to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaptation as no toxic effects on the liver have been reported. From available data it is not possible to identify a NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.</p> <p>Camphor appears to have moderate acute oral toxicity, with an LD50 of 1310 mg/kg in mice. It demonstrated moderate to high toxicity in acute inhalation studies (450 mg/m<sup>3</sup> (72 ppm) in mice and 500 mg/m<sup>3</sup> (80 ppm) in rats). In subchronic studies, inhaled camphor resulted in emphysema in mice at 210 mg/m<sup>3</sup> (33 ppm) and rabbits at 33 mg/m<sup>3</sup> (5 ppm). In 13-week subchronic dermal studies, camphor had NOAELs of 1000 mg/kg bw/day in mice and 250 mg/kg bw/day in rats. IPCS reported negative results in carcinogenicity tests for camphor. In addition, camphor was negative for genotoxicity in a microsome mutagenesis test, and a peripheral blood micronucleus assay. Reproductive toxicity studies were not available for camphor, however, in developmental toxicity studies, camphor demonstrated no foetal toxicity (with NOAELs 800 mg/kg bw/day in rats) at dose levels that resulted in maternal toxicity</p> <p>For monoterpenes:</p> <p>The chemical category designated terpenoid hydrocarbons includes three simple C10 isomeric monocyclic terpene hydrocarbons (d-limonene, dl-limonene, and terpinolene) two simple C10 acyclic terpene hydrocarbons (beta-myrcene and dihydromyrcene) and mixtures composed primarily of d-limonene, dl-limonene (dipentene), terpinolene, myrcene, and alpha and beta-pinene</p> <p>Monoterpene hydrocarbons are mainly released by coniferous woodland such as pine trees, cedars, redwood and firs. To a lesser extent, they are also produced and released by deciduous plants. They are common components of traditional foods occurring in essentially all fruits and vegetables.</p> <p>Members of this chemical category are of very low acute toxicity</p> <p>Studies of terpene hydrocarbons indicate that they are rapidly absorbed, distributed, metabolised and excreted. The principal metabolic pathway involves side chain oxidation to yield monocyclic terpene alcohols and carboxylic acids. These metabolites are mainly conjugated with glucuronic acid and excreted in the</p>

urine, or to a lesser extent in the feces. A secondary pathway involves epoxidation of either the exocyclic or endocyclic double bond yielding an epoxide that is subsequently detoxicated via formation of the corresponding diol or conjugation with glutathione. Although some species- and sex-specific differences exist, studies for *d*-limonene and *beta*-myrcene indicate that the monoterpene hydrocarbons in this chemical category will participate in common pathways of absorption, distribution, metabolism and excretion.

**Genotoxicity:** Based on the results of this *in vivo* genotoxicity assay and the numerous *in vitro* genotoxicity assays, it is unlikely that any of these materials would exhibit a significant genotoxic potential *in vivo*.

**Carcinogenicity:** Under the conditions of 2-year gavage studies, conducted by NTP, there was clear evidence of carcinogenic activity of *d*-limonene for male F344/N rats as shown by increased incidences in tubular cell hyperplasia, adenomas, and adenocarcinomas of the kidney. There was no evidence of carcinogenic activity of *d*-limonene for female rats receiving 300 or 600 mg/kg bw/d. It has been demonstrated that renal lesions, which were observed in the NTP study, resulted from the accumulation of aggregates of *alpha*-2 microglobulin (a low molecular-weight protein synthesised in the liver) and limonene-1,2-epoxide in the P2 segment of the renal proximal tubule. While humans produce low molecular weight serum proteins, which are reabsorbed by the kidney, there is no evidence that a similar *alpha*-2 microglobulin is produced.

The kidney changes seen in male rats administered limonene have been well characterized, and are known to be specific to the male rat and of no significance in human risk assessment.

**Reproductive toxicity:** Substances within this chemical category exhibit low reproductive toxicity potential. This is based on the results of three reproductive toxicity assays, using sweet orange peel oil predominantly composed of *d*-limonene and *beta*-myrcene.

**Developmental toxicity:** Given the results of six developmental toxicity assays using limonene, sweet orange oil and *beta*-myrcene, it may be concluded that the substances within this chemical category exhibit low developmental toxicity potential

For terpenoid tertiary alcohols and their related esters:

Substances assigned to this category, as part of the HPV Challenge Program, possess close structural relationships, similar physicochemical properties and participate in the same pathways of metabolic detoxification and have similar toxicologic potential.

**Acute Toxicity:** Oral and dermal LD50 values for members of this chemical category indicate a low order of both oral and dermal toxicity. All rabbit dermal, and mouse and rat oral LD50 values exceed 2000 mg/kg with the majority of values greater than 5000 mg/kg

**Repeat dose toxicity:** In a safety evaluation study, a 50/50 mixture of linalool and citronellol was fed to male and female rats (number and strain not specified) in the diet. The daily intake was calculated to be 50 mg/kg bw of each. Measurements of haematology, clinical chemistry, and urinalysis at weeks 6 and 12 showed no statistically significant differences between test and control groups. Histopathology revealed no dose-related lesions. A slight retardation of growth was observed in males only, but was concluded by the authors to be biologically insignificant

**Reproductive toxicity:** Four groups of 10 virgin CrI CD rats were administered 0, 250, 500, or 1000 mg/kg bw of an essential oil (coriander oil) known to contain 73% linalool by mass. The test material was given by gavage once daily, 7 days prior to cohabitation, through cohabitation (maximum of 7 days), gestation, delivery, and a 4-day post-parturition period. The duration of the study was 39 days. Maternal effects reported included increased body weight and increased food consumption at 250 mg/kg/d, a non-statistically significant decrease in body weight and food consumption and decreased gestation index and decreased length of gestation at 500 mg/kg/d, and a statistically significant decrease in body weight and food consumption, statistically significant decrease in gestation index, length of gestation, and litter size at 1000 mg/kg/d. The only effect on pups was a decrease in viability of pups at the highest dose level. The authors concluded that there were no effects observed in the dams at the low dose of 250 mg/kg bw/d or in the offspring at the 250 and 500 mg/kg bw/d levels. The authors concluded that the maternal NOAEL was 250 mg/kg/d and the developmental NOAEL was 500 mg/kg/d.

Four groups of 10 virgin CrI CD rats were administered 0, 375, 750, or 1500 mg/kg bw of an essential oil (cardamom oil) known to contain greater than 65 % tertiary terpenoid alcohols with 5 % alpha-terpineol acetate by mass. Maternal observations included a non-statistically significant decrease in body weight gain and food consumption at 375 mg/kg/d.

Mortality, clinical signs, a statistically significant decrease in body weight gain and food consumption, and gross lesions at necropsy were seen at 750 and 1500 mg/kg/d. The only effects on pups were a reduced body weight gain in pups at 750 and 1500 mg/kg/d and increased mortality at 1500 mg/kg/d. The authors concluded that there were no significant adverse effects in the dams or offspring at the 375 mg/kg/d dose. A maternal NOEL was reported to be less than 375 mg/kg/d based on reduced body weight gain and food consumption at 375 mg/kg/d and a developmental NOAEL was reported to be 375 mg/kg/d

**Developmental toxicity:** A range finding study and follow-up teratology study was performed with pine oil. Pregnant CrI:CD(SD) BR rats were given 0, 50, 100, 500, 750, or 1000 mg/kg/d by gavage in corn oil on days 6 to 20 of gestation. Laparotomies were performed, corpora lutea were counted, and the uterus of each rat was removed, weighed and then examined for number, placement and viability of implantations. Live foetuses were weighed, sexed and gross external alterations were identified. There were no deaths or abortions during the course of this study. Necropsy revealed no gross lesions. Maternal effects included local alopecia, decreased body weight gain and food consumption for the 3 highest dose levels. At 750 and 1000 mg/kg, average gravid uterine weight was reduced. In foetuses, decreased body weight was observed at dose levels of 100 mg/kg and above, and at dose levels of 500 and above there was a slight increase in average number of resorptions/litter.

In the follow-up teratology study, pregnant CrI:CD(SD) BR rats were given 0, 50, 600, or 1200 mg/kg/d by gavage in corn oil on days 6 to 20 of gestation. Six of the 25 rats in 1200 mg/kg dose group died and necropsies revealed that adrenal weights were significantly increased in these rats. At 1200 mg/kg/d, foetuses exhibited increased incidences of delayed ossification, delayed brain development, decreased weights, increased embryo-foetal mortality, and sunken eye bulge with associated soft and hard tissue findings, a dose that also resulted in maternal death and a low incidence of embryo-foetal death (resorption). The maternal and developmental NOEL for pine oil was greater than 50 mg/kg/d but less than 600 mg/kg/d

**Genotoxicity:** Mutagenicity/genotoxicity testing has been performed on six members of this chemical category, including a complete battery of *in vitro* genotoxicity tests using linalool. In nineteen separate *in vitro* tests on the mutagenicity and genotoxicity of terpenoid tertiary alcohols and related esters, all but two were negative. One of the positive results for linalool was observed in a rec assay using differences in growth rates in two strains of *Bacillus subtilis* as a measure of DNA changes. In contrast, no evidence of mutagenicity was observed in the same test at a higher concentrations nor was DNA damage observed in a rat hepatocyte UDS assay. The authors of the mouse lymphoma assay which gave a weak positive result for linalool, emphasized that positive results in this assay are commonly observed for polar substances in the absence of S-9 and may be associated with changes in physiologic culture conditions (pH and osmolality).

Based on a weight of evidence evaluation of the available *in vitro* and *in vivo* mutagenicity and genotoxicity assays on terpenoid tertiary alcohols and related esters, this group of flavouring substances would not be expected to exhibit a low genotoxic potential *in vivo*

**Metabolic fate:** Based on the results of hydrolysis, the reactivity of linalool in aqueous media, and data on metabolism it is concluded that members of this chemical category exhibit similar chemical and biochemical fate. The esters are readily hydrolyzed to the corresponding alcohols, linalool and alpha-terpineol. Linalool is then partially converted to alpha-terpineol mainly under acidic conditions. Alicyclic and aliphatic tertiary alcohols are efficiently detoxicated by two principal pathways: conjugation primarily with glucuronic acid and excretion primarily in urine, and omega-oxidation to eventually yield diacids and their reduced or hydrated analogs. These polar metabolites will be efficiently excreted primarily in the urine either unchanged or as the glucuronic acid conjugates. The physicochemical and toxicological properties of these substances are consistent with their known reactivity and common metabolic fate.

Esters belonging to this category can be hydrolysed to their corresponding terpenoid alcohol and organic acid. Hydrolysis can also be catalysed by a class of esters known as carboxylesterases or B-type esterases that predominated in hepatocytes.

Esters of tertiary terpenoid alcohols are readily hydrolyzed in animals, including fish. Once hydrolysed, the resulting alcohols undergo excretion unchanged or as the glucuronic acid conjugate. To a minor extent, CYP-450 mediated oxidation at the omega or omega-1 position yields polar oxidized metabolites capable of excretion primarily in the urine. Terpenoid alcohols formed in the gastrointestinal tract are readily absorbed. During hydrolysis under acidic condition cyclisation may occur.

In humans and animals, terpenoid tertiary alcohols primarily conjugate with glucuronic acid and are excreted in the urine and feces. Terpenoid alcohols with unsaturation may also undergo allylic oxidation to form polar diol metabolites that may be excreted either free or conjugated. If the diol contains a primary alcohol function, it may undergo further oxidation to the corresponding carboxylic acid. In a minor pathway, the endocyclic alkene of alpha-terpineol is epoxidised and then hydrolyzed to yield a triol metabolite 1,2,8-trihydroxy-p-menthane which also has been reported in humans following inadvertent oral ingestion of a pine oil disinfectant containing alpha-terpineol.

Bicyclic tertiary alcohols are conjugated with glucuronic acid and excreted primarily in the urine. In rabbits the structurally related bicyclic tertiary alcohols thujyl alcohol (4-methyl-1-(1-methylethyl)bicyclo[3.1.0]-hexan-3-ol) and beta-santenol (2,3,7-

trimethylbicyclo[2.2.1]-heptan-2-ol) are conjugated with glucuronic acid. In a metabolism study using the terpenoid tertiary alcohol trans-sobrerol, in humans, dogs, and rats, ten metabolites were isolated in urine, eight of which were characterised in humans. Two principle modes of metabolism were observed, allylic oxidation of the ring positions and alkyl substituents, and conjugation of the tertiary alcohol fractions with glucuronic acid. These metabolic patterns are common modes of converting tertiary and secondary terpenoid alcohols to polar metabolites, which are easily excreted in the urine and faeces. Menthol forms

similar conjugation products in rats

Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and conjugal contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.

Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.

Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.

Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease. Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

**Hands:** Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

**Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

**Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.

**Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

**Pigmentary anomalies:** The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

**Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

**General/respiratory:** Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems.

In the case of prehapten, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers.

#### Prehapten

Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen

Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxy radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context.

It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clinical studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure

synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.

#### Prohaptens

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.

In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

**QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.

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.

55rad

<b>CALCIUM CARBONATE &amp; POLYVINYL CHLORIDE</b>	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
<b>CALCIUM CARBONATE &amp; XYLENE</b>	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
<b>CALCIUM CARBONATE &amp; XYLENE</b>	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
<b>POLYVINYL CHLORIDE &amp; PINE OIL</b>	No significant acute toxicological data identified in literature search.
<b>POLYVINYL CHLORIDE &amp; XYLENE</b>	The substance is classified by IARC as Group 3: <b>NOT</b> classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.

<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 available but does not fill the criteria for classification  
✔ – Data available to make classification  
☹ – Data Not Available to make classification

## SECTION 12 ECOLOGICAL INFORMATION

### Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
calcium carbonate	LC50	96	Fish	>56000mg/L	4
calcium carbonate	EC50	72	Algae or other aquatic plants	>14mg/L	2
calcium carbonate	NOEC	72	Algae or other aquatic plants	14mg/L	2
polyvinyl chloride	LC50	96	Fish	2.315mg/L	3
polyvinyl chloride	EC50	96	Algae or other aquatic plants	25.141mg/L	3
titanium dioxide	LC50	96	Fish	9.214mg/L	3
titanium dioxide	EC50	48	Crustacea	>10mg/L	2
titanium dioxide	EC50	72	Algae or other aquatic plants	5.83mg/L	4
titanium dioxide	EC20	72	Algae or other aquatic plants	1.81mg/L	4

Continued...



## Duroproof PU 600

titanium dioxide	NOEC	336	Fish	0.089mg/L	4
xylene	LC50	96	Fish	2.6mg/L	2
xylene	EC50	48	Crustacea	>3.4mg/L	2
xylene	EC50	72	Algae or other aquatic plants	4.6mg/L	2
xylene	EC50	24	Crustacea	0.711mg/L	4
xylene	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
pine oil	LC50	96	Fish	18.35mg/L	4
pine oil	EC50	48	Crustacea	24.5mg/L	4

**Legend:**

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (*Palaemonetes pugio*) and brown shrimp (*Penaeus aztecus*) was dimethylnaphthalenes > methylnaphthalenes > naphthalenes.

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

**DO NOT discharge into sewer or waterways.**

**Persistence and degradability**

Ingredient	Persistence: Water/Soil	Persistence: Air
polyvinyl chloride	LOW	LOW
titanium dioxide	HIGH	HIGH
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)

**Bioaccumulative potential**

Ingredient	Bioaccumulation
polyvinyl chloride	LOW (LogKOW = 1.6233)
titanium dioxide	LOW (BCF = 10)
xylene	MEDIUM (BCF = 740)

**Mobility in soil**

Ingredient	Mobility
polyvinyl chloride	LOW (KOC = 23.74)
titanium dioxide	LOW (KOC = 23.74)

**SECTION 13 DISPOSAL CONSIDERATIONS****Waste treatment methods**

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Containers may still present a chemical hazard/ danger when empty.</li> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> </ul> <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>▶ Reduction</li> <li>▶ Reuse</li> <li>▶ Recycling</li> <li>▶ Disposal (if all else fails)</li> </ul> <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</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>
-------------------------------------	---

## SECTION 14 TRANSPORT INFORMATION

### Labels Required

COMBUSTIBLE LIQUID	COMBUSTIBLE LIQUID, regulated for storage purposes only
Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

## SECTION 15 REGULATORY INFORMATION

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

**CALCIUM CARBONATE(471-34-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

**POLYVINYL CHLORIDE(9002-86-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)

**TITANIUM DIOXIDE(13463-67-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

**XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

Australia Hazardous Substances Information System - Consolidated Lists

**PINE OIL(8002-09-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (xylene; polyvinyl chloride; pine oil)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (polyvinyl chloride; pine oil)
Japan - ENCS	N (pine oil)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
<b>Legend:</b>	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

## SECTION 16 OTHER INFORMATION

### Other information

#### Ingredients with multiple cas numbers

Name	CAS No
calcium carbonate	471-34-1, 13397-26-7, 15634-14-7, 1317-65-3, 72608-12-9, 878759-26-3, 63660-97-9, 459411-10-0, 198352-33-9, 146358-95-4
titanium dioxide	13463-67-7, 1317-70-0, 1317-80-2, 12188-41-9, 1309-63-3, 100292-32-8, 101239-53-6, 116788-85-3, 12000-59-8, 12701-76-7, 12767-65-6, 12789-63-8, 1344-29-2, 185323-71-1, 185828-91-5, 188357-76-8, 188357-79-1, 195740-11-5, 221548-98-7, 224963-00-2, 246178-32-5, 252962-41-7, 37230-92-5, 37230-94-7, 37230-95-8, 37230-96-9, 39320-58-6, 39360-64-0, 39379-02-7, 416845-43-7, 494848-07-6, 494848-23-6, 494851-77-3, 494851-98-8, 55068-84-3, 55068-85-4, 552316-51-5, 62338-64-1, 767341-00-4, 97929-50-5, 98084-96-9

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  
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

This document is copyright.

Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH.

TEL (+61 3) 9572 4700.