

Thermon Snap Trace

Thermon

Chemwatch: 7047-35

Version No: 4.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 1

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L.GHS.AUS.EN

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

Product Identifier

Product name	Thermon Snap Trace
Synonyms	semi rigid black extruded compound
Other means of identification	Not Available

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

Relevant identified uses	<p>Fatty acid amides are nonionic substances which have a strong tendency to reduce friction on various surfaces by forming a layer on surfaces. This coating action may be attributed to their hydrophobic character and strong hydrogen bonding. Primary, secondary, and bisamides are widely used as lubricating or slip agents and alkanolamides. Their ethoxylated counterparts are commonly used as surfactants in personal care and detergent applications.</p> <p>The dehydration of amides that produces nitriles is of great commercial value. The most widely used synthetic route for primary amides is the reaction of a fatty acid with anhydrous ammonia.</p> <p>Typical uses of fatty acid amides include lubricants for synthetic resins (polyethylene, polypropylene, etc.), anti-blocking agents, mold release agents, printing ink additives, and pigment/dye dispersants</p> <p>Heat transfer compound.</p>
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Details of the supplier of the safety data sheet

Registered company name	Thermon
Address	30 London Drive Bayswater Victoria 3153 Australia
Telephone	+61 3 9762 6900
Fax	+61 3 9762 9519
Website	Not Available
Email	Not Available

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	Not Available
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification ^[1]	Acute 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

Hazard pictogram(s)	Not Applicable
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SIGNAL WORD NOT APPLICABLE

Hazard statement(s)

H402 Harmful to aquatic life

Precautionary statement(s) Prevention

P273 Avoid release to the environment.

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

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
7782-42-5	30-60	<u>graphite</u>
24937-78-8	10-30	<u>ethylene/ vinyl acetate copolymer</u>
13983-17-0	1-10	<u>wollastonite</u>
110-30-5	1-10	<u>N,N'-ethylenebisstearamide</u>
64742-57-0	1-10	<u>residual oils, petroleum, hydrotreated</u>
26813-14-9	1-10	<u>1,3-pentadiene/ 2-methyl-2-butene copolymer</u>
27676-62-6	<1	<u>tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> Wash out immediately with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation. <p>In case of burns:</p> <ul style="list-style-type: none"> Immediately apply cold water to burn either by immersion or wrapping with saturated clean cloth. DO NOT remove or cut away clothing over burnt areas. DO NOT pull away clothing which has adhered to the skin as this can cause further injury. DO NOT break blister or remove solidified material. Quickly cover wound with dressing or clean cloth to help prevent infection and to ease pain. For large burns, sheets, towels or pillow slips are ideal; leave holes for eyes, nose and mouth. DO NOT apply ointments, oils, butter, etc. to a burn under any circumstances. Water may be given in small quantities if the person is conscious. Alcohol is not to be given under any circumstances. Reassure. Treat for shock by keeping the person warm and in a lying position. Seek medical aid and advise medical personnel in advance of the cause and extent of the injury and the estimated time of arrival of the patient.
Inhalation	<ul style="list-style-type: none"> If fumes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.

Ingestion

- ▶ Immediately give a glass of water.
- ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES**Extinguishing media**

- ▶ There is no restriction on the type of extinguisher which may be used.
- ▶ Use extinguishing media suitable for surrounding area.

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

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

Advice for firefighters**Fire Fighting**

- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves in the event of a fire.
- ▶ Prevent, by any means available, spillage from entering drains or water courses.
- ▶ Use fire fighting procedures suitable for surrounding area.
- ▶ **DO NOT** approach containers suspected to be hot.
- ▶ Cool fire exposed containers with water spray from a protected location.
- ▶ If safe to do so, remove containers from path of fire.
- ▶ Equipment should be thoroughly decontaminated after use.

Fire/Explosion Hazard

- ▶ The material is not readily combustible under normal conditions.
- ▶ However, it will break down under fire conditions and the organic component may burn.
- ▶ Not considered to be a significant fire risk.
- ▶ Heat may cause expansion or decomposition with violent rupture of containers.
- ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).
- ▶ May emit acrid smoke.

Other decomposition products include:

, carbon dioxide (CO₂)

, aldehydes

, acrolein

, nitrogen oxides (NO_x)

, silicon dioxide (SiO₂)

,

other pyrolysis products typical of burning organic material.

May emit poisonous fumes.

May emit corrosive fumes.

CARE: Contamination of heated / molten liquid with water may cause violent steam explosion, with scattering of hot contents.

A fire in bulk finely divided carbon may not be obviously visible unless the material is disturbed and sparks appear. A straw broom may be useful to produce the disturbance.

Explosion and Ignition Behaviour of Carbon Black with Air

Lower Limit for Explosion:	50 g/m ³ (carbon black in air)
Maximum Explosion Pressure:	10 bar
Maximum Rate of Pressure Rise:	30-100 bar/sec
Minimum Ignition Temperature:	315 deg. C.
Ignition Energy:	>1 kJ
Glow Temperature:	500 deg. C. (approx.)

Notes on Test Methods:

Tests 1, 2 and 3 were conducted by Bergwerkeschaftliche Versuchstrecke, Dortmunde-Derne, using a 1 m³ vessel with two chemical igniters having an intensity of 5000 W.S.

Tests 1 and 2 results are confirmed by information in the Handbook of Powder Technology, Vol. 4 (P. Field)

In Test 4, a modified Godbert-Greenwald furnace was used. See U.S. Bureau of Mines, Report 5624, 1960, p.5, "Lab Equipment and Test Procedures".

Test 5 used a 1 m³ vessel with chemical igniters of variable intensity.

	Test 6 was conducted in a laboratory oven. Active glowing appeared after 3 minutes exposure. (European Committee for Biological Effects of Carbon Black) (2/84)
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up waste regularly and abnormal spills immediately. ▶ Avoid breathing dust and contact with skin and eyes. ▶ Wear protective clothing, gloves, safety glasses and dust respirator. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up or sweep up. NOTE: Vacuum cleaner must be fitted with an exhaust micro filter (HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). ▶ Dampen with water to prevent dusting before sweeping. ▶ Place in suitable containers for disposal. <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid contact with skin and eyes. ▶ Wear impervious gloves and safety goggles. ▶ Trowel up/scrape up. ▶ Place spilled material in clean, dry, sealed container. ▶ Flush spill area with water.
Major Spills	<p>Minor hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Control personal contact with the substance, by using protective equipment as required. ▶ Prevent spillage from entering drains or water ways. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▶ Wash area and prevent runoff into drains or waterways. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ Wet, activated carbon removes oxygen from the air thus producing a severe hazard to workers inside carbon vessels and in enclosed or confined spaces where activated carbons might accumulate. ▶ Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
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Other information	<p>Carbon and charcoal may be stabilised for storage and transport, without moistening, by treatment with hot air at 50 deg. C.. Use of oxygen-impermeable bags to limit oxygen and moisture uptake has been proposed. Surface contamination with oxygenated volatiles may generate a heat of reaction (spontaneous heating). Should stored product reach 110 deg. C., stacked bags should be pulled apart with each bag separated by an air space to permit cooling away from other combustible materials.</p> <ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
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Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid overheating in processing as this causes decomposition and degradation of polymer. This may start at temperatures above 90 deg.C, and becomes more rapid at higher temperatures with generation of highly irritating acetic acid vapour. <p>For carbon powders:</p> <ul style="list-style-type: none"> ▶ Avoid oxidising agents, reducing agents. ▶ Reaction with finely divided metals, bromates, chlorates, chloramine monoxide, dichlorine oxide, iodates, metal nitrates, oxygen difluoride, peroxyformic acid, peroxyfuroic acid and trioxxygen difluoride may result in an exotherm with ignition or explosion. Less active forms of carbon will ignite or explode on suitably intimate contact with oxygen, oxides, peroxides, oxosalts, halogens, interhalogens and other oxidising species. ▶ Explosive reaction with ammonium nitrate, ammonium perchlorate, calcium hypochlorite and iodine pentoxide may occur following heating. Carbon may react violently with nitric acid and may be explosively reactive with nitrogen trifluoride at reduced temperatures. In the presence of nitrogen oxide, incandescence and ignition may occur. Finely divided or highly porous forms of carbon, exhibiting a high surface area to mass (up to 2000 m²/g) may function as unusually active fuels possessing both adsorptive and catalytic properties which accelerate the release of energy in the presence of oxidising substances. Dry metal-impregnated charcoal catalysts may generate sufficient static, during handling, to cause ignition. ▶ Graphite in contact with liquid potassium, rubidium or caesium at 300 deg. C. produces intercalation compounds (C8M) which ignite in air and may react explosively with water. The fusion of powdered diamond and potassium hydroxide may produce explosive decomposition. ▶ Activated carbon, when exposed to air, represents a potential fire hazard due to a high surface area and adsorptive capacity. Freshly prepared material may ignite spontaneously in the presence of air especially at high humidity. Spontaneous combustion in air may occur at 90-100 deg. C. The presence of moisture in air facilitates the ignition. Drying oils and oxidising oils promote spontaneous heating and ignition; contamination with these must be avoided. Unsaturated drying oils (linseed oil etc.) may ignite following adsorption owing to an enormous increase in the surface area of oil exposed to air; the rate of oxidation may also be catalysed by metallic impurities in the carbon. A similar, but slower effect occurs on fibrous materials such as cotton waste. Spontaneous heating of activated carbon is related to the composition and method of preparation of the activated carbon. Free radicals, present in charcoal, are responsible for autoignition. Self-heating and autoignition may also result from adsorption of various vapours and gases (especially oxygen). For example, activated carbon auto- ignites in flowing air at 452-518 deg. C.; when the base, triethylenediamine, is adsorbed on the carbon (5%) the autoignition temperature is reduced to 230-260 deg. C.. An exotherm is produced at 230-260 deg. C., at high flow rates of air, although ignition did not occur until 500 deg. C.. Mixtures of sodium borohydride with activated carbons, in air, promote the oxidation of sodium borohydride, producing a self-heating reaction that may result in the ignition of charcoal and in the production of hydrogen through thermal decomposition of the borohydride.

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	graphite	Graphite (all forms except fibres) (respirable dust) (natural & synthetic)	3 mg/m ³	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
graphite	Graphite; (Mineral carbon)	6 mg/m ³	16 mg/m ³	95 mg/m ³
ethylene/ vinyl acetate copolymer	Ethylene/vinyl acetate copolmer	30 mg/m ³	330 mg/m ³	2,000 mg/m ³

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Ingredient	Original IDLH	Revised IDLH
graphite	1,250 mg/m3	Not Available
ethylene/ vinyl acetate copolymer	Not Available	Not Available
wollastonite	Not Available	Not Available
N,N'-ethylenebisstearamide	Not Available	Not Available
residual oils, petroleum, hydrotreated	2,500 mg/m3	Not Available
1,3-pentadiene/ 2-methyl-2-butene copolymer	Not Available	Not Available
tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate	Not Available	Not Available

MATERIAL DATA

for calcium silicate:

containing no asbestos and <1% crystalline silica

ES TWA: 10 mg/m3 inspirable dust

TLV TWA: 10 mg/m3 total dust (synthetic nonfibrous) A4

Although in vitro studies indicate that calcium silicate is more toxic than substances described as "nuisance dusts" is thought that adverse health effects which might occur following exposure to 10-20 mg/m3 are likely to be minimal. The TLV-TWA is thought to be protective against the physical risk of eye and upper respiratory tract irritation in workers and to prevent interference with vision and deposition of particulate in the eyes, ears, nose and mouth.

For graphite:

Graphite pneumoconiosis resembles coal workers' pneumoconiosis. Data indicate that the higher the crystalline silica content of graphite the more likely the disease will increase in severity. The presence of anthracite coal in the production of some synthetic grades of graphite appears to make arbitrary the use of the term, "synthetic", "artificial" or "natural".

NOTE: This substance has been classified by the ACGIH as A4 **NOT** classifiable as causing Cancer in humans

The TLV-TWA for carbon black is recommended to minimise complaints of excessive dirtiness and applies only to commercially produced carbon blacks or to soots derived from combustion sources containing absorbed polycyclic aromatic hydrocarbons (PAHs). When PAHs are present in carbon black (measured as the cyclohexane-extractable fraction) NIOSH has established a REL-TWA of 0.1 mg/m3 and considers the material to be an occupational carcinogen.

The NIOSH REL-TWA was "selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs".

This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.

NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP


NOTE L: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 3% DMSO extract as measured by IP 346.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

<p>Appropriate engineering controls</p>	<p>Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air.</p> <p>Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde]</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.</p> <p>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>
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	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
Within each range the appropriate value depends on:		
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
	3: Intermittent, low production.	3: High production, heavy use
	4: Large hood or large air mass in motion	4: Small hood-local control only
<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>		
Personal protection		
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ 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] 	
Skin protection	See Hand protection below	
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber ▶ When handling hot materials wear heat resistant, elbow length gloves. ▶ Rubber gloves are not recommended when handling hot objects, materials ▶ Protective gloves eg. Leather gloves or gloves with Leather facing 	
Body protection	See Other protection below	
Other protection	<ul style="list-style-type: none"> ▶ When handling hot or molten liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. ▶ Usually handled as molten liquid which requires worker thermal protection and increases hazard of vapour exposure. ▶ CAUTION: Vapours may be irritating. ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. 	
Thermal hazards	Not Available	

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
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up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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

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

Appearance	<p>Fatty acid amides (FAAs) comprise a family of neutral lipids that is related to other classes of N-acyl amines, such as N-acyl amino acids, N-acylethanolamines, and more complicated species like sphingomyelins and ceramides.</p> <p>Commercial FAAs generally consist of a fatty acid, usually derived from coconut oil, which is linked to an amide group by a C-N bond. The amide may either be monoethanolamide (MEA), diethanolamide (DEA), or monoisopropanolamide (MIPA). Representative structures of FAA are indicated below. The alkyl chain usually contains 12 to 18 carbon atoms. FAAs can be represented as R-C(=O)-N(R)'R'' .</p> <p>FAAs which contain a saturated or unsaturated alkyl chain derived from a fatty acid, can be divided into three categories (based on the following notation).</p> <ul style="list-style-type: none"> ▶ The first is <i>primary monoamides</i> in which R is a fatty alkyl or alkenyl chain of C5-C23 and R' =R''= H. ▶ The second is <i>substituted monoamides</i>, including secondary, tertiary, and alkanolamides in which R is a fatty alkyl or alkenyl chain of C5-C23; R' and R'' may be a hydrogen, fatty alkyl, aryl, or alkylene oxide condensation groups with at least one alkyl, aryl, or alkylene oxide group. ▶ The third category is <i>bisamides</i> where R groups are fatty alkyl or alkenyl chains. R' and R'' may be hydrogen, fatty alkyl, aryl, or alkylene oxide condensation groups. <p>Primary and secondary amides show strong hydrogen bonding that account for their high melting points and low solubilities in most solvents. With tertiary amides (disubstituted amides), hydrogen bonding is not possible, as exhibited by their increased solubility and lower melting points. Many fatty acid amides are essentially insoluble in water. Amides have a strong tendency to reduce friction on various surfaces by forming a layer on surfaces. This coating action may be attributed to their hydrophobic character and strong hydrogen bonding. Fatty acid amides in general are stable to elevated processing temperatures, air oxidation, and dilute acids and bases. Alkanolamides are made from triglycerides or fatty acid methyl esters reacted with monoethanolamine or diethanolamine that then can be ethoxylated with ethylene oxide under basic catalyses. Common products are stearamide, cocamide, ethylene bis(stearamide), cocamide, DEA or MEA, cocamidopropyl dimethyl amine, and cocamide monoethanolamine ethoxylate</p> <p>Regardless of the carbon number, the melting point of saturated fatty acid amides falls in a range of 100 to 110 C. In contrast, the melting point of unsaturated fatty acid amides is significantly affected by the carbon number of such fatty acid amides; their melting points fall in a range of 70 to 85 C relative to the carbon number in a range of 18 to 22.</p> <p> Semi-rigid black extruded compound; does not mix with water.</p>
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Physical state	Non Slump Paste	Relative density (Water = 1)	1.22
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Applicable	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available

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Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	0
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant 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> <ul style="list-style-type: none"> ▶ Usually handled as molten liquid which requires worker thermal protection and increases hazard of vapour exposure. ▶ CAUTION: Vapours may be irritating.
Ingestion	<p>The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p>
Skin Contact	<p>Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. 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>
Eye	<p>Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<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.</p> <p>Red blood cells and rabbit alveolar macrophages exposed to calcium silicate insulation materials in vitro showed haemolysis in one study but not in another. Both studies showed the substance to be more cytotoxic than titanium dioxide but less toxic than asbestos.</p> <p>In a small cohort mortality study of workers in a wollastonite quarry, the observed number of deaths from all cancers combined and lung cancer were lower than expected. Wollastonite is a calcium inosilicate mineral (CaSiO₃). In some cases, small amounts of iron (Fe), and manganese (Mn), and lesser amounts of magnesium (Mg) substitute for calcium (Ca) in the mineral formulae (e.g., rhodonite)</p> <p>In an inhalation study in rats no increase in tumour incidence was observed but the number of fibres with lengths</p>

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exceeding 5 µm and a diameter of less than 3 µm was relatively low. Four grades of wollastonite of different fibre size were tested for carcinogenicity in one experiment in rats by intrapleural implantation. There was no information on the purity of the four samples used. A slight increase in the incidence of pleural sarcomas was observed with three grades, all of which contained fibres greater than 4 µm in length and less than 0.5 µm in diameter.

In two studies by intraperitoneal injection in rats using wollastonite with median fibre lengths of 8.1 µm and 5.6 µm respectively, no intra-abdominal tumours were found.

Evidence from wollastonite miners suggests that occupational exposure can cause impaired respiratory function and pneumoconiosis. However animal studies have demonstrated that wollastonite fibres have low biopersistence and induce a transient inflammatory response compared to various forms of asbestos. A two-year inhalation study in rats at one dose showed no significant inflammation or fibrosis.

Prolonged or repeated inhalation of dust may result in pneumoconiosis (lung disease caused by inhalation dust).

Graphite workers have reported symptoms of headaches, coughing, depression, low appetite, dyspnoea (difficult breathing) and black sputum.

A number of studies indicate that graphitosis is a progressive and disabling disease and that the presence of crystalline silica and some silicates as graphite impurities have a pronounced synergistic effect.

Workers suffering from graphite pneumoconiosis have generally worked in the industry for long periods, i.e. 10 years or more, although some cases have been reported after as little as four years.

Data indicate the higher the crystalline silica content of graphite the greater is the severity of the pneumoconiosis.

Pre-employment and periodic examinations should be directed towards detecting significant respiratory disease through chest X-rays and pulmonary function tests.

In a small cohort mortality study of workers in a wollastonite quarry, the observed number of deaths from all cancers combined and lung cancer were lower than expected.

In an inhalation study in rats no increase in tumour incidence was observed but the number of fibres with lengths exceeding 5 µm and a diameter of less than 3 µm was relatively low. Four grades of wollastonite of different fibre size were tested for carcinogenicity in one experiment in rats by intrapleural implantation. There was no information on the purity of the four samples used. A slight increase in the incidence of pleural sarcomas was observed with three grades, all of which contained fibres greater than 4 µm in length and less than 0.5 µm in diameter.

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Chronic inhalation exposure of production workers has caused decreased pulmonary function and myocardial dystrophy.

There is suggestive but inconclusive evidence that carbon black containing polyaromatic hydrocarbons (PAHs) has been responsible for induction of skin cancers in exposed workers.

Long term inhalation of carbon black can cause cough, phlegm, tiredness, chest pain and headache. Dermal, mucosal, or inhalation exposure can cause irritation.

Inhalation of carbon black by mice, rats and monkeys caused thickened alveolar walls, increased pulmonary collagen, right atrial and ventricular strain, hypertrophy of the right atrial and ventricular septum and increased heart weights. Although carbon black itself did not cause cancer in treated animals, carbon black containing polyaromatic hydrocarbons (PAHs) did cause cancer following chronic administration by all routes tested.

Epidemiological studies of workers in the carbon black producing industries of North America and Western Europe show no significant health effect due to occupational exposure to carbon black. Several other studies provide conflicting evidence. Early studies in the former USSR and Eastern Europe report respiratory diseases amongst workers exposed to carbon black, including bronchitis, pneumonia, emphysema and rhinitis. These studies are of questionable validity due to inadequate study design and methodology, lack of appropriate controls for cigarette smoking and other confounding factors such as concurrent exposure to carbon dioxide, coal oil and petroleum vapours. Moreover, review of these studies indicates that the concentrations of carbon black were greater than current occupational standards.

Carbon black may cause adverse pulmonary changes following prolonged or repeated inhalation of the dust; these include oral mucosal lesions, bronchitis and pneumoconiosis which may lead to lung tumours.

The body of evidence of carcinogenicity in animal studies comes from two chronic inhalation studies and two intratracheal instillation studies in rats, which showed significantly elevated rates of lung cancer in exposed animals. An inhalation study was tested on mice, but did not show significantly elevated rates of lung cancer in exposed animals. Epidemiologic data comes from three different cohort studies of carbon black production workers. Two studies, from the United Kingdom and Germany, with over 1,000 workers in each study group, showed elevated mortality from lung cancer in the carbon black workers. Another study of over 5,000 workers in the United States did not show elevated mortality from lung cancer in the carbon black workers. Newer findings of increased lung cancer mortality in an update from the UK study may suggest that carbon black could be a late-stage carcinogen. However, a more recent and larger study from Germany did not confirm this hypothesis that carbon black acts as a late-stage carcinogen.

In studies employing channel and furnace black, hamsters, mice, guinea pigs, rabbits and monkeys exposed to dusts for 7 hours/day, 5 days/week, at concentrations of 87.4 mg/m³ for channel black and 56.5 mg/m³ for furnace black, no malignancies were observed in any of the animals. Channel black had little if any absorbed polyaromatic hydrocarbons (PAHs) (as benzene extractables) whilst furnace black had 0.28%.

Several findings have strengthened the association between inflammation and cancer and between the particle surface area dose of carbon black and other poorly soluble low toxicity (PSLT) particles and the pulmonary inflammation response in mice and the proinflammatory effects in lung cells in vitro. Other evidence suggests that in addition to a cancer mechanism involving indirect genotoxicity through inflammation and oxidative stress, nanoparticles may act as direct carcinogens.

Carbon black appears to act like PSLT particles, which can elicit lung tumours in rats following prolonged exposure to sufficiently high concentrations of particles. Particle surface area dose was found to be most predictive of pulmonary inflammation and tumour response in rats when comparing the dose-response relationships for various types and sizes of

PSLT including carbon black. Compared to fine PSLT, much lower concentrations of ultrafine PSLT (e.g. 2.5, 6.5 or 11.5 mg/m³ carbon black and ~10 mg/m³ ultrafine titanium dioxide) were associated with impaired clearance, persistent inflammation, and malignant lung tumours in chronic inhalation studies in rats. Most evidence suggests that carbon black and other PSLT-elicited lung tumours occurs through a secondary genotoxic mechanism, involving chronic inflammation and oxidative stress. Experimental studies have shown that when the particle lung dose reaches a sufficiently high concentration (e.g., mass dose of ~0.5 mg fine-sized PSLT/g lung in rats), the alveolar macrophage-mediated clearance process begins to be impaired (complete impairment occurs at ~10 mg/g lung. Overloading of lung clearance is accompanied by pulmonary inflammation, leading to increased production of reactive oxygen and nitrogen species, depletion of antioxidants and/or impairment of other defense mechanisms, cell injury, cell proliferation, fibrosis, and as seen in rats, induction of mutations and eventually cancer. Rats appear to be more sensitive to carbon black and other PSLT than other rodent species. Although studies in humans have not shown a direct link between inhaled PSLT and lung cancer, many of the steps in the mechanism observed in rats have also been observed in humans who work in dusty jobs, including increased particle lung retention and pulmonary inflammation in workers exposed to coal dust or crystalline silica and elevated lung cancer has been observed in some studies of workers exposed to carbon black, crystalline silica, and diesel exhaust particles

Monkeys exposed to channel black for 1000-1500 hours showed evidence of electrocardiac changes indicative of right atrial and right ventricular strain. These changes increased progressively until after 10,000 hours of exposure, when the changes were marked. The authors of this study concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of non-toxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours' exposure and marked atrial and right ventricular strain after 10,000 hours' exposure. The authors concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of nontoxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours exposure and marked atrial and right ventricular strain after 10,000 hours exposure.

Chromatographic fractions of oily material extracted from carbon black have been shown to be carcinogenic whilst the unfractionated extracts are not. The activity of some carcinogens appear to be inhibited by carbon black itself.

Overexposure to respirable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity, chest infections

Repeated exposures, in an occupational setting, to high levels of fine- divided dusts may produce a condition known as pneumoconiosis which is the lodgement of any inhaled dusts in the lung irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50,000 inch), are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion (exertional dyspnea), increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Other signs or symptoms include altered breath sounds, diminished lung capacity, diminished oxygen uptake during exercise, emphysema and pneumothorax (air in lung cavity) as a rare complication.

Removing workers from possibility of further exposure to dust generally leads to halting the progress of the lung abnormalities. Where worker-exposure potential is high, periodic examinations with emphasis on lung dysfunctions should be undertaken

Dust inhalation over an extended number of years may produce pneumoconiosis.. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. It is further classified as being of noncollagenous or collagenous types. Noncollagenous pneumoconiosis, the benign form, is identified by minimal stromal reaction, consists mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible.

Thermon Snap Trace	TOXICITY	IRRITATION
	Not Available	Not Available
graphite	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2 mg/l4 h ^[1] Oral (rat) LD50: >2000 mg/kg ^[2]	Not Available
ethylene/ vinyl acetate copolymer	TOXICITY	IRRITATION
	Not Available	Not Available
wollastonite	TOXICITY	IRRITATION
	Not Available	Not Available
N,N'-ethylenebisstearamide	TOXICITY	IRRITATION
	Oral (mouse) LD50: >20000 mg/kg ^[2]	Non-irritant
		Skin (rabbit) patch in PEG400 Slight irritant
residual oils, petroleum, hydrotreated	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Not Available

Thermon Snap Trace

	Inhalation (rat) LC50: >3.9 mg/l4 h ^[1]	
	Oral (rat) LD50: >2000 mg/kg ^[1]	
1,3-pentadiene/ 2-methyl-2-butene copolymer	TOXICITY	IRRITATION
	Not Available	Not Available
tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >10000 mg/kg ^[2]	Eye : Not irritating *
	Oral (rabbit) LD50: >6800 mg/kg ^[2]	Skin : Not irritating *
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

WOLLASTONITE	<p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
RESIDUAL OILS, PETROLEUM, HYDROTREATED	<p>The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives; The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:</p> <ul style="list-style-type: none"> ▶ The adverse effects of these materials are associated with undesirable components, and ▶ The levels of the undesirable components are inversely related to the degree of processing; ▶ Distillate base oils receiving the same degree or extent of processing will have similar toxicities; ▶ The potential toxicity of <i>residual base oils</i> is independent of the degree of processing the oil receives. ▶ The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing. <p>Unrefined & mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.</p> <p>Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing for Unrefined and Mildly Refined Distillate Base Oils</p> <p>Acute toxicity: LD50s of >5000 mg/kg (bw) and >2g/kg (bw) for the oral and dermal routes of exposure, respectively, have been observed in rats dosed with an unrefined light paraffinic distillate. The same material was also reported to be "moderately irritating" to the skin of rabbits. When tested for eye irritation in rabbits, the material produced Draize scores of 3.0 and 4.0 (unwashed/washed eyes) at 24 hours, with the scores returning to zero by 48 hours. The material was reported to be "not sensitising" when tested in guinea pigs</p> <p>Repeat dose toxicity: 200, 1000 and 2000 mg/kg (bw)/day of an unrefined base oil has been applied undiluted to the skin of male and female rabbit.. The test material was applied to the rabbits' skins 3 times/week for 4 weeks. To ensure maximum exposure, the applied material was covered with an occlusive dressing for 6 hours. In the high dose group, body weight gains were affected by treatment. These effects were largely due to effects on growth rate during the first week of the study. There were no significant differences between treated and control groups for any of the recorded haematological and clinical chemistry values. Gross and microscopic pathology findings relating to the treated skin were seen in all rabbits in the highest dose group. The findings consisted of "slight" to "moderate" proliferative changes in the treated skin.</p> <p>Reproductive/ developmental toxicity No reproductive or developmental toxicity studies have been reported for unrefined & mildly refined distillate base oils. However, a developmental toxicity screening study has been reported for heavy vacuum gas oil, a material with a process history similar to the unrefined distillate base oils.. As an unrefined vacuum distillate material, heavy vacuum gas oil contains the broadest spectrum of chemical components and highest concentration of bioavailable and/or biologically active components. Because of their lack of or low level of processing, in comparison to other refined base oils, the unrefined lubricating base oils will also have higher concentrations of bioavailable and/or biologically active components.</p> <p>Heavy vacuum gas oil was applied daily to the skin of pregnant rats on days 0-19 of gestation. Dose levels administered included: 30, 125, 500 and 1000 mg/kg (bw)/day. All animals were euthanised on day 20. In the dams, the only dose-related finding at gross necropsy was pale colored lungs in four animals in the highest dose group and in one animal in the 500 mg/kg (bw)/day group. Mean thymus weights of the dams in the highest dose group were approximately half those of the control groups. Although absolute liver weights were unaffected by exposure to the gas oil, mean relative liver weights were increased (approximately 15%) in groups exposed to doses greater than 125 mg/kg (bw)/day. Maternal and foetal body weights were reduced at 500 and 1000 mg/kg (bw)/day. Significant increases in resorptions were also seen in these two dose groups. Soft tissue variations and malformations, and skeletal malformations were also increased at 500 and 1000 mg/kg</p>

Genotoxicity: Modified Ames assays have been carried out on a number of base oils that were either unrefined or poorly refined. The oils were found to be mutagenic, with a strong correlation between mutagenicity and 3-7 ring PAC content.

Carcinogenicity: The general conclusions that can be drawn from the animal carcinogenicity studies are potential skin carcinogens. When applied repeatedly to the skin, carcinogenic base oils are associated only with skin tumours and not with an increase in systemic tumours

Residual Base Oils

Residual oils have substantial polycyclic aromatic compound (PAC) levels when assayed by traditional methods. On this basis, they would be expected to have mutagenic and/or carcinogenic activity. However, no adverse effects have been seen in either *in vitro* mutagenicity or dermal carcinogenicity testing of residual base oils, irrespective of the degree of processing they have undergone. Ultraviolet, HPLC/UV, GC/MS, and infrared analyses of these oils indicate that the aromatics they contain are predominantly 1-3 rings that are highly alkylated (paraffinic and naphthenic). Because they are found in such a high boiling material (> 550 C), it is estimated that the alkyl side-chains of these 1-3 ring aromatics would be approximately 13 to 25 carbons in length. These highly alkylated aromatic ring materials are either devoid of the biological activity necessary to cause mutagenesis and carcinogenesis, or are largely non-bioavailable to the organisms

Acute toxicity: There are no acute toxicity data available for the residual base oils. It is thought that the high molecular weight of these materials and associated low bioavailability preclude the systemic doses necessary to produce acute toxicity. Furthermore, tests of a variety of distillate base oils, including unrefined materials that contain high levels of biologically active materials, have consistently shown low acute toxicity.

Repeat dose toxicity: No subchronic repeat-dose studies have been reported on residual base oils. However, two dermal carcinogenicity studies have been performed

Reproductive and developmental toxicity: There are no reproductive or developmental toxicity data available for the residual base oils

Carcinogenicity: A dermal carcinogenicity study of a residual base oil in mice has been reported. The test substance was described as "a non-solvent refined, deasphalted, dewaxed residual paraffinic lubricant base oil". For eighteen months, three times/week, undiluted test material was applied to the skin of female CF1 mice. Two other groups of mice underwent similar treatments, but for only 22 or 52 weeks. The base oil produced minimal clinical evidence of skin irritation. No tumours of epidermal origin were observed in animals dosed with the base oil. Furthermore, no treatment-related effects were observed with regard to clinical condition, body weight gain, mortality or post mortem findings.

A second dermal carcinogenicity study of a residual base oil has been conducted in male C3H/HeJ mice. The test substance was described as "deasphalted, dewaxed, residual oil". The test material was applied undiluted to the animals' backs, three times/week for 24 months. None of the animals treated with the test material developed skin tumours, or any other tumours considered treatment-related.

The absence of systemic toxicity in these two dermal carcinogenicity studies supports the belief that the high molecular weight of the residual base oils and the resulting low bio-availability preclude the internal doses necessary to elicit systemic toxicity.

Genotoxicity:

In vitro (mutagenicity): Samples of a vacuum residuum and four residual base oils tested negative for the induction of frame shift mutations in modified Ames assays

In vivo (chromosomal aberrations): There is no *in vivo* genotoxicity data available for the residual base oils. However, *in vitro* mutagenicity tests have been conducted on residual base oils and have produced negative results. Dermal carcinogenicity studies on these materials have also been negative. Given these consistent results, and the low bioavailability of these materials, it is expected that *in vivo* mutagenicity tests would also be negative.

For hindered phenols:

Available data shows that acute toxicity of these substances is low.

Mutagenicity. Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.

In Vitro Chromosome Aberration Studies. *In vitro* chromosome aberration studies are available for several members. All except 2,6-di-tert-butyl-p-cresol were negative

In Vivo Chromosome Aberration Studies. *In vivo* studies evaluating chromosome damage are available for six of the hindered phenols. All *in vivo* evaluations were negative.

Repeated Dose Toxicity. Repeated dose toxicity data of approximately three months (90-day, 12- and 13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day)

Carcinogenicity: Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats

For tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate Available mammalian acute toxicity data indicate very low toxicity by oral and dermal exposure. The LD50 values are >5000 mg/kg bw (oral) and >2000 mg/kg bw (dermal). The material does not show mutagenic or clastogenic properties. In sub-chronic toxicity studies in the rat and dog only minor effects have been observed. The substance was found to be neither carcinogenic nor a reproductive toxicant. Skin Sensitization: (Guinea pig) Maximization test: Not a sensitiser. RIPT (Humans): Not a primary irritant, fatiguing agent, or sensitiser in any of 200 individuals when tested as a slurry (concentration not specified). Subchronic Toxicity: In a three month study, rats were administered dietary concentrations of 0, 150, 800, 3,000 and

TRIS(3,5-DI-TERT-BUTYL-4-HYDROXYBENZYL) ISOCYANURATE

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	<p>15,000 ppm. There were no treatment-related changes in blood chemistry, organ weights or urinalysis. Macroscopic and microscopic examination did not reveal treatment-related effects. The females in the 3,000 and 15,000 dose groups were found to have a statistically significant, but slight, increase in mean circulating blood platelets. In the absence of other effects on the blood, it is not thought to be biologically meaningful. The males were found to have a slight increase in food consumption at the 15,000 ppm dose level. The no observable effect level (NOEL) was determined as 3,000 ppm (201 mg/kg/day) in males and 800 ppm (50 mg/kg/day) in females. Treatment of rats with 0, 100, 1,000, or 10,000 ppm in the diet for 28 days was found to cause increased food and water consumption in both sexes at the 10,000 ppm level. The no observable adverse effect level (NOAEL) was 1,000 ppm, equivalent to 95 mg/kg/day. Genetic toxicity: Chromosomal Aberration Assay: Negative</p>
<p>Thermon Snap Trace & GRAPHITE & ETHYLENE/ VINYL ACETATE COPOLYMER & WOLLASTONITE & RESIDUAL OILS, PETROLEUM, HYDROTREATED & 1,3-PENTADIENE/ 2-METHYL-2-BUTENE COPOLYMER</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>Thermon Snap Trace & N,N'-ETHYLENEBISSTEARAMIDE</p>	<p>For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides) The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented.</p> <p>The Fatty nitrogen-derived amides (FND amides) comprise four categories:</p> <p>Subcategory I: Substituted Amides</p> <p>Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components)</p> <p>Subcategory III: Imidazole Derivatives</p> <p>Subcategory IV: FND Amphoteric</p> <p>Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies.</p> <p>Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II.</p> <p>Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories.</p> <p>Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonella reverse mutation assay exist for all of the subcategories.</p> <p>Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II.</p> <p>In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.</p> <p>Some typical applications of FND Amides are:</p> <p>masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers.</p> <p>The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax.</p> <p>The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health. The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals.</p>

**Thermon Snap Trace &
N,N'-ETHYLENEBISSTEARAMIDE**

Fatty acid amides (FAA) are ubiquitous in household and commercial environments. The most common of these are based on coconut oil fatty acids alkanolamides. These are the most widely studied in terms of human exposure.

Fatty acid diethanolamides (C8-C18) are classified by Comité Européen des Agents de Surface et de leurs Intermediaires Organiques (CESIO) as Irritating (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes). Fatty acid monoethanolamides are classified as Irritant (Xi) with the risk phrases R41

Several studies of the sensitization potential of cocoamide diethanolamide (DEA) indicate that this FAA induces occupational allergic contact dermatitis and a number of reports on skin allergy patch testing of cocoamide DEA have been published. These tests indicate that allergy to cocoamide DEA is becoming more common.

Alkanolamides are manufactured by condensation of diethanolamine and the methylester of long chain fatty acids. Several alkanolamides (especially secondary alkanolamides) are susceptible to nitrosamine formation which constitutes a potential health problem. Nitrosamine contamination is possible either from pre-existing contamination of the diethanolamine used to manufacture cocoamide DEA, or from nitrosamine formation by nitrosating agents in formulations containing cocoamide DEA. According to the Cosmetic Directive (2000) cocoamide DEA must not be used in products with nitrosating agents because of the risk of formation of N-nitrosamines. The maximum content allowed in cosmetics is 5% fatty acid dialkanolamides, and the maximum content of N-nitrosodialkanolamines is 50 mg/kg. The preservative 2-bromo-2-nitropropane-1,3-diol is a known nitrosating agent for secondary and tertiary amines or amides. Model assays have indicated that 2-bromo-2-nitropropane-1,3-diol may lead to the N-nitrosation of diethanolamine forming the carcinogenic compound, N-nitrosodiethanolamine which is a potent liver carcinogen in rats (IARC 1978).

Several FAAs have been tested in short-term genotoxicity assays. No indication of any potential to cause genetic damage was seen. Lauramide DEA was tested in mutagenicity assays and did not show mutagenic activity in *Salmonella typhimurium* strains or in hamster embryo cells. Cocoamide DEA was not mutagenic in strains of *Salmonella typhimurium* when tested with or without metabolic activation.

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Miljøministeriet (Danish Environmental Protection Agency)

**Thermon Snap Trace &
GRAPHITE &
N,N'-ETHYLENEBISSTEARAMIDE**

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic 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.

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

Legend: ✘ – Data 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

Thermon Snap Trace	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

Thermon Snap Trace

	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
graphite	Not Available	Not Available	Not Available	Not Available	Not Available
	Not Available	Not Available	Not Available	Not Available	Not Available
ethylene/ vinyl acetate copolymer	Not Available	Not Available	Not Available	Not Available	Not Available
	Not Available	Not Available	Not Available	Not Available	Not Available
wollastonite	Not Available	Not Available	Not Available	Not Available	Not Available
	Not Available	Not Available	Not Available	Not Available	Not Available
N,N'-ethylenebisstearamide	Not Available	Not Available	Not Available	Not Available	Not Available
	Not Available	Not Available	Not Available	Not Available	Not Available
residual oils, petroleum, hydrotreated	Not Available	Not Available	Not Available	Not Available	Not Available
	Not Available	Not Available	Not Available	Not Available	Not Available
1,3-pentadiene/ 2-methyl-2-butene copolymer	Not Available	Not Available	Not Available	Not Available	Not Available
	Not Available	Not Available	Not Available	Not Available	Not Available
tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate	LC50	96	Fish	>1000mg/L	2
	NOEC	504	Crustacea	100mg/L	2

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.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
N,N'-ethylenebisstearamide	HIGH	HIGH
tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
N,N'-ethylenebisstearamide	LOW (BCF = 6.2)
tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate	LOW (BCF = 5.8)

Mobility in soil

Ingredient	Mobility
N,N'-ethylenebisstearamide	LOW (KOC = 5754000000)
tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate	LOW (KOC = 10000000000)

SECTION 13 DISPOSAL CONSIDERATIONS

Thermon Snap Trace

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

GRAPHITE(7782-42-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

ETHYLENE/ VINYL ACETATE COPOLYMER(24937-78-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

WOLLASTONITE(13983-17-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
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N,N'-ETHYLENEBISSTEARAMIDE(110-30-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

RESIDUAL OILS, PETROLEUM, HYDROTREATED(64742-57-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

1,3-PENTADIENE/ 2-METHYL-2-BUTENE COPOLYMER(26813-14-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

TRIS(3,5-DI-TERT-BUTYL-4-HYDROXYBENZYL) ISOCYANURATE(27676-62-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	N (wollastonite)
Canada - NDSL	N (ethylene/ vinyl acetate copolymer; 1,3-pentadiene/ 2-methyl-2-butene copolymer; graphite; tris(3,5-di-tert-butyl-4-hydroxybenzyl) isocyanurate; residual oils, petroleum, hydrotreated; wollastonite; N,N'-ethylenebisstearamide)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (ethylene/ vinyl acetate copolymer; 1,3-pentadiene/ 2-methyl-2-butene copolymer)
Japan - ENCS	N (1,3-pentadiene/ 2-methyl-2-butene copolymer; graphite; residual oils, petroleum, hydrotreated)

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Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	N (wollastonite)
Legend:	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
wollastonite	13983-17-0, 9056-30-8, 57657-07-5

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

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TEL (+61 3) 9572 4700.