

# Meguiars G123, Plastx (22-121A): G12306, G12310

## Motor Active

Chemwatch: 4940-18  
Version No: 12.1.1.1  
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

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L.GHS.AUS.EN

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

### Product Identifier

Product name	Meguiars G123, Plastx (22-121A): G12306, G12310
Synonyms	Product Identification Numbers; 14-1000-0501-7, 14-1000-0502-5; Product Code: G123
Other means of identification	Not Available

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

Relevant identified uses	Automotive plastic cleaner polish.
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### Details of the supplier of the safety data sheet

Registered company name	Motor Active
Address	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia
Telephone	+61 2 9737 9422 1800 350 622
Fax	+61 2 9737 9414
Website	www.motoractive.com.au
Email	andrew.spira@motoractive.com.au

### Emergency telephone number

Association / Organisation	MotorActive
Emergency telephone numbers	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
Other emergency telephone numbers	13 11 26 (In Case of Emergency contact: Poison Information Hotline)

## SECTION 2 HAZARDS IDENTIFICATION

### Classification of the substance or mixture

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

#### CHEMWATCH HAZARD RATINGS

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

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

Poisons Schedule	Not Applicable
Classification <sup>[1]</sup>	Skin Corrosion/Irritation Category 2, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

### Label elements

Hazard pictogram(s)	
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SIGNAL WORD **WARNING**

### Hazard statement(s)

H315	Causes skin irritation.
H412	Harmful to aquatic life with long lasting effects.
AUH066	Repeated exposure may cause skin dryness and cracking.

### Supplementary statement(s)

Not Applicable

Continued...

**CLP classification (additional)**

Not Applicable

**Precautionary statement(s) Prevention**

P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

**Precautionary statement(s) Response**

P362	Take off contaminated clothing and wash before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P332+P313	If skin irritation occurs: Get medical advice/attention.

**Precautionary statement(s) Storage**

Not Applicable

**Precautionary statement(s) Disposal**

P501	Dispose of contents/container in accordance with local regulations.
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**SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS****Substances**

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
64742-88-7.	7-13	<u>naphtha, petroleum, hydrodesulfurised heavy</u>
8042-47-5	5-10	<u>white mineral oil (petroleum)</u>
1344-28-1.	5-10	<u>aluminium oxide</u>
63148-62-9	1-5	<u>polydimethylsiloxane</u>
	50-70	Ingredients determined not to be hazardous

**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>Wash out immediately with fresh running water.</li> <li>Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.</li> <li>Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </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>Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>Transport to hospital, or doctor.</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> </ul>

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

- Heavy and persistent skin contamination over many years may lead to dysplastic changes. Pre-existing skin disorders may be aggravated by exposure to this product.
- In general, emesis induction is unnecessary with high viscosity, low volatility products, i.e. most oils and greases.
- High pressure accidental injection through the skin should be assessed for possible incision, irrigation and/or debridement.

**NOTE:** Injuries may not seem serious at first, but within a few hours tissue may become swollen, discoloured and extremely painful with extensive subcutaneous necrosis. Product may be forced through considerable distances along tissue planes.

**SECTION 5 FIREFIGHTING MEASURES****Extinguishing media**

- Water spray or fog.
- Alcohol stable foam.
- Dry chemical powder.
- Carbon dioxide.

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

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

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear full body protective clothing with breathing apparatus.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</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> </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> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) other pyrolysis products typical of burning organic material.</p> <p>When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.</p> <p>May emit poisonous fumes. May emit corrosive fumes.</p> <p><b>CARE:</b> Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.</p>
<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>	<p>Slippery when spilt.</p> <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>Slippery when spilt. 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>▶ Containers, even those that have been emptied, may contain explosive vapours.</li> <li>▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.</li> <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> </ul>
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	<ul style="list-style-type: none"> <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>	<p><b>CARE:</b> Water in contact with heated material may cause foaming or a steam explosion with possible severe burns from wide scattering of hot material. Resultant overflow of containers may result in fire.</p> <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	naphtha, petroleum, hydrodesulfurised heavy	Petrol (gasoline)	900 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	naphtha, petroleum, hydrodesulfurised heavy	White spirits	790 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	white mineral oil (petroleum)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium oxide	Aluminium oxide	10 mg/m3	Not Available	Not Available	Not Available

**EMERGENCY LIMITS**

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
naphtha, petroleum, hydrodesulfurised heavy	Naphtha, hydrotreated heavy; (Isopar L-rev 2)	350 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	Petroleum distillates; petroleum ether; includes clay-treated light naphthenic [64742-45-6]; low boiling [68477-31-6]; petroleum extracts [64742-06-9]; petroleum base oil [64742-46-7]; petroleum 50 thinner, petroleum spirits [64475-85-0], Soltrol, VM&P naphtha [8032-32-4]; Ligroine, and paint solvent; petroleum paraffins C5-C20 [64771-72-8]; hydrotreated light naphthenic [64742-53-6]; solvent refined light naphthenic [64741-97-5]; and machine coolant 1	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	Naphtha (coal tar); includes solvent naphtha, petroleum (64742-88-7), naphtha (petroleum) light aliphatic, rubber solvent (64742-89-8), heavy catalytic cracked (64741-54-4), light straight run (64741-46-4), heavy aliphatic solvent (64742-96-7), high flash aromatic and aromatic solvent naphtha (64742-95-6)	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	Stoddard solvent; (Mineral spirits, 85% nonane and 15% trimethyl benzene)	300 mg/m3	1,800 mg/m3	29500 mg/m3
aluminium oxide	Aluminum oxide; (Alumina)	5.7 mg/m3	15 mg/m3	25 mg/m3
polydimethylsiloxane	Dimethyl siloxane; (Dimethylpolysiloxane; Syltherm XLT; Syltherm 800; Silicone 360)	65 mg/m3	720 mg/m3	4,300 mg/m3

Ingredient	Original IDLH	Revised IDLH
naphtha, petroleum, hydrodesulfurised heavy	20,000 mg/m3 / 1,100 ppm / 1,000 ppm	Not Available
white mineral oil (petroleum)	2,500 mg/m3	Not Available
aluminium oxide	Not Available	Not Available
polydimethylsiloxane	Not Available	Not Available

**MATERIAL DATA****Exposure controls**

<b>Appropriate engineering controls</b>	<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>
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## Meguiars G123, Plastx (22-121A): G12306, G12310

An approved self contained breathing apparatus (SCBA) may be required in some situations. 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.

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

Within each range the appropriate value depends on:

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

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

## Personal protection



## Eye and face protection

- ▶ 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

- ▶ Wear chemical protective gloves, e.g. PVC.
  - ▶ Wear safety footwear or safety gumboots, e.g. Rubber
- The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
- The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
- Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
- Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
- frequency and duration of contact,
  - chemical resistance of glove material,
  - glove thickness and
  - dexterity
- Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
  - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
  - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
  - Contaminated gloves should be replaced.
- As defined in ASTM F-739-96 in any application, gloves are rated as:
- Excellent when breakthrough time > 480 min
  - Good when breakthrough time > 20 min
  - Fair when breakthrough time < 20 min
  - Poor when glove material degrades
- For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
- It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
- Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
- Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:
- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
  - Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential
- Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

<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>

### Respiratory protection

Type A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- 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.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### Information on basic physical and chemical properties

<b>Appearance</b>	Light blue liquid with pleasant odour; mixes with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	0.96
<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>	8-8.9	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	>96
<b>Initial boiling point and boiling range (°C)</b>	176.67	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	>93	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Applicable	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not 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>	Partly miscible	<b>pH as a solution (1%)</b>	Not Available
<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>	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. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals,
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## Meguiars G123, Plastx (22-121A): G12306, G12310

	<p>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>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. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p>
<b>Ingestion</b>	Accidental ingestion of the material may be damaging to the health of the individual.
<b>Skin Contact</b>	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</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>	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.
<b>Chronic</b>	<p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</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.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, on the basis that similar materials tested in appropriate animal studies provide some suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production. Very fine Al<sub>2</sub>O<sub>3</sub> powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.</p> <p>When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.</p> <p>Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.</p> <p>There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.</p> <p>Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.</p> <p>Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.</p> <p>After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.</p> <p>At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.</p> <p>Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27,</p>

## Meguiars G123, Plastx (22-121A): G12306, G12310

100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively. Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramidal-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia*, August 1995]

Principal route of exposure is by skin contact; lesser exposures include inhalation of fumes from hot oils, oil mists or droplets. Prolonged contact with mineral oils carries with it the risk of skin conditions such as oil folliculitis, eczematous dermatitis, pigmentation of the face (melanosis) and warts on the sole of the foot (plantar warts). With highly refined mineral oils no appreciable systemic effects appear to result through skin absorption. Exposure to oil mists frequently elicits respiratory conditions, such as asthma; the provoking agent is probably an additive. High oil mist concentrations may produce lipid pneumonia although clinical evidence is equivocal. In animals exposed to concentrations of 100 mg/m<sup>3</sup> oil mist, for periods of 12 to 26 months, the activity of lung and serum alkaline phosphatase enzyme was raised; 5 mg/m<sup>3</sup> oil mist did not produce this response. These enzyme changes are sensitive early indicators of lung damage. Workers exposed to vapours of mineral oil and kerosene for 5 to 35 years showed an increased prevalence of slight basal lung fibrosis.

Many studies have linked cancers of the skin and scrotum with mineral oil exposure. Contaminants in the form of additives and the polycyclic aromatic hydrocarbons (PAHs - as in the crude base stock) are probably responsible. PAH levels are higher in aromatic process oils/used/reclaimed motor oils. Subchronic 90-day feeding studies conducted on male and female rats on highly refined white mineral oils and waxes found that higher molecular-weight hydrocarbons (microcrystalline waxes and the higher viscosity oils) were without biological effects. Paraffin waxes and low- to mid viscosity oils produced biological effects that were inversely proportional to molecular weight, viscosity and melting point: oil-type and processing did not appear to be determinants. Biological effects were more pronounced in females than in males. Effects occurred mainly in the liver and mesenteric lymph nodes and included increased organ weights, microscopic inflammatory changes, and evidence for the presence of saturated mineral hydrocarbons in affected tissues. Inflammation of the cardiac mitral valve was also observed at high doses in rats treated with paraffin waxes.

Smith J.H., et al: *Toxicologic Pathology*: 24, 2, 214-230, 1996  
Repeated application of mildly hydrotreated oils (principally paraffinic), to mouse skin, induced skin tumours; no tumours were induced with severely hydrotreated oils.  
Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]  
Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Animal studies:  
No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human

Meguiars G123, Plastx (22-121A): G12306, G12310	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
naphtha, petroleum, hydrodesulfurised heavy	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1-900 mg/kg <sup>[1]</sup> Oral (rat) LD50: >4-800 mg/kg <sup>[1]</sup>	Not Available
white mineral oil (petroleum)	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2-000 mg/kg <sup>[1]</sup> Inhalation (rat) LC50: 0.007-0.64 mg/l4 h <sup>[1]</sup> Oral (rat) LD50: >5-000 mg/kg <sup>[1]</sup>	Not Available
aluminium oxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (rat) LD50: >2-000 mg/kg <sup>[1]</sup>	Not Available
polydimethylsiloxane	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >3000 mg/kg <sup>[2]</sup> Oral (rat) LD50: >35000 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg/1h - mild

## Meguiars G123, Plastx (22-121A): G12306, G12310

**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

<p><b>NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY</b></p>	<p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.</p>
<p><b>WHITE MINERAL OIL (PETROLEUM)</b></p>	<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"> <li>• The adverse effects of these materials are associated with undesirable components, and</li> <li>• The levels of the undesirable components are inversely related to the degree of processing;</li> <li>• Distillate base oils receiving the same degree or extent of processing will have similar toxicities;</li> <li>• The potential toxicity of residual base oils is independent of the degree of processing the oil receives.</li> <li>• The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing.</li> </ul> <p>The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential. Unrefined and 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</p> <p>Skin irritation is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).</p> <p>Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class (Other Lubricant Base Oils).</p> <p>Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class (Other Lubricant Base Oils))</p> <p>Germ cell mutagenicity: The tests performed within the "in vivo" studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class (Other Lubricant Base Oils)).</p> <p>Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction.</p> <p>STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects &gt; 280 mg/m3 and for systemic effects NOAEL &gt; 980 mg/m3.</p> <p>Sub-chronic toxicity</p> <p>90-day study Dermal: NOAEL &gt; 2000 mg/kg (CONCAWE studies).</p> <p>Repeat dose toxicity:</p> <p>Oral</p> <p>NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.</p> <p>Inhalation</p> <p>The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m3. As no systemic toxicity was observed, the overall NOAEL for systemic effects was &gt; 980 mg/m3.</p> <p>Dermal</p> <p>In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.</p> <p>Toxicity to reproduction:</p> <p>Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.</p> <p>Developmental toxicity, teratogenicity:</p> <p>Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.</p> <p>The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic</p> <p>Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.</p> <p>Highly and Severely Refined Distillate Base Oils</p> <p><b>Acute toxicity:</b> Multiple studies of the acute toxicity of highly &amp; severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be &gt;5 g/kg (bw) and the dermal LD50s have ranged from &gt;2 to &gt;5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to &gt; 4 mg/l.</p>

	<p>When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating" Testing in guinea pigs for sensitization has been negative</p> <p><b>Repeat dose toxicity:</b> . Several studies have been conducted with these oils. The weight of evidence from all available data on highly &amp; severely refined base oils support the presumption that a distillate base oil's toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.</p> <ul style="list-style-type: none"> <li>▶ The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,</li> <li>▶ The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and</li> <li>▶ The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.</li> </ul> <p><b>Reproductive and developmental toxicity:</b> A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study's authors.</p> <p>A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed fetuses were found among three litters The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.</p> <p><b>Genotoxicity:</b> <i>In vitro</i> (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices. <i>In vivo</i> (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.</p> <p><b>Carcinogenicity:</b> Highly &amp; severely refined base oils are not carcinogens, when given either orally or dermally. 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. Oral (rat) TCLo: 92000 mg/kg/92D-Cont. Generally the toxicity and irritation is of low order. White oils and highly/solvent refined oils have not shown the long term risk of skin cancer that follows persistent skin contamination with some other mineral oils, due in all probability to refining that produces low content of both polyaromatics (PAH) and benz-alpha-pyrenes (BaP)</p>
<p><b>POLYDIMETHYLSILOXANE</b></p>	<p>For siloxanes: Effects which based on the reviewed literature do not seem to be problematic are acute toxicity, irritant effects, sensitization and genotoxicity. Some studies indicate that some of the siloxanes may have endocrine disrupting properties, and reproductive effects have caused concern about the possible effects of the siloxanes on humans and the environment. Only few siloxanes are described in the literature with regard to health effects, and it is therefore not possible to make broad conclusions and comparisons of the toxicity related to short-chained linear and cyclic siloxanes based on the present evaluation. Data are primarily found on the cyclic siloxanes D4 (octamethylcyclotetrasiloxane) and D5 (decamethylcyclopentasiloxane) and the short-linear HMDS (hexamethyldisiloxane). These three siloxanes have a relatively low order of acute toxicity by oral, dermal and inhalatory routes and do not require classification for this effect. They are not found to be irritating to skin or eyes and are also not found sensitizing by skin contact. Data on respiratory sensitization have not been identified. Subacute and subchronic toxicity studies show that the liver is the main target organ for D4 which also induces liver cell enzymes. This enzyme induction contributes to the elimination of the substance from the tissues. Primary target organ for D5 exposure by inhalation is the lung. D5 has an enzyme induction profile similar to that of D4. Subacute and subchronic inhalation of HMDS affect in particular the lungs and kidneys in rats. None of the investigated siloxanes show any signs of genotoxic effects <i>in vitro</i> or <i>in vivo</i>. Preliminary results indicate that D5 has a potential carcinogenic effect. D4 is considered to impair fertility in rats by inhalation and is classified as a substance toxic to reproduction in category 3 with the risk phrase R62 ('Possible risk of impaired fertility'). The results of a study to screen for oestrogen activity indicate that D4 has very weak oestrogenic and antioestrogenic activity and is a partial agonist (enhances the effect of the estrogen). It is not uncommon for compounds that are weakly oestrogenic to also have antioestrogenic properties. Comparison of the oestrogenic potency of D4 relative to ethinyloestradiol (steroid hormone) indicates that D4 is 585,000 times less potent than ethinyloestradiol in the rat strain Sprague- Dawley and 3.7 million times less potent than ethinyloestradiol in the Fisher-344 rat strain. Because of the lack of effects on other endpoints designated to assess oestrogenicity, the oestrogenicity as mode of action for the D4 reproductive effects has been questioned. An indirect mode of action causing a delay of the LH (luteinising hormone) surge necessary for optimal timing of ovulation has been suggested as the mechanism. Based on the reviewed information, the critical effects of the siloxanes are impaired fertility (D4) and potential carcinogenic effects (uterine tumours in females). Furthermore there seem to be some effects on various organs following repeated exposures, the liver (D4), kidney (HMDS) and lung (D5 and HMDS) being the target organs. A possible oestrogenic effect contributing to the reproductive toxicity of D4 is debated. There seems however to be some indication that this toxicity may be caused by another mechanism than oestrogen activity. Studies are available for linear siloxanes from an analogue group comprising di- to hexa- siloxanes, as well as key physicochemical properties. The results of the acute toxicity studies for this analogue group are in agreement: there is no evidence from any of the available studies that the substances in this group have any potential for acute toxicity (in terms of either lethality or adverse clinical effects) by any route up to and exceeding the maximum dose levels tested according to current OECD guidelines. It is therefore valid to read-across the lack of acute toxicity between the members of the group where there are data gaps The metabolism of silanes and siloxanes is influenced by the chemistry of silicon, and it is fundamentally different from that of carbon compounds. These differences are due to the fact that silicon is more electropositive than carbon; Si-Si bonds are less stable than C-C bonds and Si-O bonds form very readily, the latter due to their high bond energy. Functional groups such as -OH, -CO<sub>2</sub>H, and -CH<sub>2</sub>OH are commonly seen in organic drug metabolites. If such functionalities are formed from siloxane metabolism, they will undergo rearrangement with migration of the Si atom from carbon to oxygen. Consequently, alpha hydroxysilanes may isomerise to silanols and this provides a mechanism by which very polar metabolites may be formed from highly hydrophobic alkylsiloxanes in relatively few metabolic steps The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. No toxic response noted during 90 day subchronic inhalation toxicity studies The no observable effect level is 450 mg/m3. Non-irritating and non-sensitising in human patch test. [Xerox]*</p>
<p><b>NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY &amp; ALUMINIUM OXIDE</b></p>	<p>No significant acute toxicological data identified in literature search.</p>

<p><b>Acute Toxicity</b></p>	<p>☹</p>	<p><b>Carcinogenicity</b></p>	<p>☹</p>
<p><b>Skin Irritation/Corrosion</b></p>	<p>✔</p>	<p><b>Reproductivity</b></p>	<p>☹</p>
<p><b>Serious Eye Damage/Irritation</b></p>	<p>☹</p>	<p><b>STOT - Single Exposure</b></p>	<p>☹</p>
<p><b>Respiratory or Skin sensitisation</b></p>	<p>☹</p>	<p><b>STOT - Repeated Exposure</b></p>	<p>☹</p>

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

Meguiars G123, Plastx (22-121A): G12306, G12310	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

  

naphtha, petroleum, hydrodesulfurised heavy	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	72	Algae or other aquatic plants	=13mg/L	1
	NOEC	72	Algae or other aquatic plants	=0.1mg/L	1
	EC50	48	Crustacea	>100mg/L	1
	EC50	96	Algae or other aquatic plants	=450mg/L	1
	EC50	72	Algae or other aquatic plants	=6.5mg/L	1
	NOEC	72	Algae or other aquatic plants	<0.1mg/L	1
	LC50	96	Fish	0.00746mg/L	4
	EC50	48	Crustacea	0.058mg/L	4
	BCF	96	Fish	0.2mg/L	4
	NOEC	168	Crustacea	<=0.05mg/L	4
	LC50	96	Fish	8.8mg/L	4
	EC50	48	Crustacea	3.7mg/L	4
	EC50	72	Algae or other aquatic plants	=6.5mg/L	1
	NOEC	72	Algae or other aquatic plants	<0.1mg/L	1
	EC50	72	Algae or other aquatic plants	=6.5mg/L	1
	NOEC	72	Algae or other aquatic plants	<0.1mg/L	1
LC50	96	Fish	2.5mg/L	2	
EC50	96	Algae or other aquatic plants	0.58mg/L	2	

  

white mineral oil (petroleum)	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

  

aluminium oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>0.218-0.6441mg/L	2
	EC50	48	Crustacea	0.7364mg/L	2
	EC50	72	Algae or other aquatic plants	0.001-0.799mg/L	2
NOEC	240	Crustacea	0.001-0.1002mg/L	2	

  

polydimethylsiloxane	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	3.16mg/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 discharge into sewer or waterways.**

## Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

## Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

## Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

Continued...

## SECTION 13 DISPOSAL CONSIDERATIONS

### Waste treatment methods

<b>Product / Packaging disposal</b>	<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>
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## SECTION 14 TRANSPORT INFORMATION

### Labels Required

<b>Marine Pollutant</b>	NO Not Applicable
<b>HAZCHEM</b>	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

#### NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY(64742-88-7.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Inventory of Chemical Substances (AICS)	International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)	

#### WHITE MINERAL OIL (PETROLEUM)(8042-47-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Inventory of Chemical Substances (AICS)	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)	

#### ALUMINIUM OXIDE(1344-28-1.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
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#### POLYDIMETHYLSILOXANE(63148-62-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

### National Inventory Status

National Inventory	Status
Australia - AICS	N (Ingredients determined not to be hazardous) Non-disclosed ingredients
Canada - DSL	N (Ingredients determined not to be hazardous) Non-disclosed ingredients
Canada - NDSL	N (polydimethylsiloxane; aluminium oxide; naphtha, petroleum, hydrodesulfurised heavy; white mineral oil (petroleum); Ingredients determined not to be hazardous) Non-disclosed ingredients
China - IECSC	N (Ingredients determined not to be hazardous) Non-disclosed ingredients
Europe - EINEC / ELINCS / NLP	N (polydimethylsiloxane; Ingredients determined not to be hazardous) Non-disclosed ingredients
Japan - ENCS	N (polydimethylsiloxane; naphtha, petroleum, hydrodesulfurised heavy; white mineral oil (petroleum); Ingredients determined not to be hazardous) Non-disclosed ingredients
Korea - KECI	N (Ingredients determined not to be hazardous) Non-disclosed ingredients
New Zealand - NZIoC	N (Ingredients determined not to be hazardous) Non-disclosed ingredients
Philippines - PICCS	N (Ingredients determined not to be hazardous) Non-disclosed ingredients
USA - TSCA	N (Ingredients determined not to be hazardous) Non-disclosed ingredients

#### 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**

<b>Revision Date</b>	08/05/2018
<b>Initial Date</b>	20/12/2002

**SDS Version Summary**

Version	Issue Date	Sections Updated
11.1.1.1	07/07/2017	Acute Health (eye), Acute Health (inhaled), Acute Health (swallowed), Classification, Physical Properties
12.1.1.1	08/05/2018	Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Appearance, Chronic Health, Classification, Disposal, Engineering Control, Environmental, Exposure Standard, First Aid (swallowed), Handling Procedure, Ingredients, Personal Protection (Respirator), Physical Properties, Spills (major), Spills (minor), Storage (storage incompatibility), Synonyms, Toxicity and Irritation (Other), Use, Name

**Other information****Ingredients with multiple cas numbers**

Name	CAS No
naphtha, petroleum, hydrodesulfurised heavy	64742-82-1., 64741-92-0., 8052-41-3., 1030262-12-4., 8032-32-4., 8030-30-6., 64742-88-7., 64742-89-8., 8002-05-9., 61789-95-5., 64742-48-9., 101795-02-2., 8031-06-9., 8030-31-7., 50813-73-5., 54847-97-1., 121448-83-7., 8031-38-7., 8031-39-8.
aluminium oxide	1344-28-1., 1011245-20-7, 1022097-81-9, 107462-07-7, 107874-14-6, 1097999-44-4, 1197416-35-5, 122784-35-4, 1234495-70-5, 1239586-42-5, 12522-88-2, 127361-04-0, 12737-16-5, 131689-14-0, 1346644-15-2, 135152-65-7, 1355357-83-3, 135667-70-8, 138361-58-7, 148619-39-0, 152743-26-5, 153858-98-1, 157516-29-5, 163581-50-8, 165390-91-0, 170448-81-4, 190401-78-6, 200295-99-4, 205316-36-5, 209552-43-2, 230616-05-4, 252756-35-7, 253606-46-1, 253606-47-2, 253606-45-0, 268724-08-9, 39354-49-9, 457654-46-5, 488831-46-5, 521982-71-8, 53809-96-4, 54352-04-4, 546141-61-1, 663170-52-3, 67853-35-4, 67894-14-8, 67894-42-2, 68189-68-4, 68389-42-4, 68389-43-5, 74871-10-6, 76363-81-0, 84149-21-3, 90669-62-8, 916225-60-0, 960377-08-6, 11092-32-3

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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