Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A)

Motor Active

Chemwatch Hazard Alert Code: **2**

Chemwatch: **4804-97**
Version No: **11.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **03/07/2014**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A)
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains beta-pinene and alpha-pinene)
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions. Release agent.

Details of the supplier of the safety data sheet

Registered company name	Motor Active	Meguiars
Address	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia	17991 Mitchell South Irvine CA 92714 United States
Telephone	+61 2 9737 9422|1800 350 622	+1 949 752 8000|+1 800 347 5700
Fax	+61 2 9737 9414	+1 949 752 5784
Website	www.motoractive.com.au	https://www.meguiars.com/
Email	andrew.spira@motoractive.com.au	Not Available

Emergency telephone number

Association / Organisation	MotorActive	Not Available
Emergency telephone numbers	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)	Not Available
Other emergency telephone numbers	13 11 26 (In Case of Emergency contact: Poison Information Hotline)	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

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

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

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

Label elements

GHS label elements

SIGNAL WORD	**DANGER**

Hazard statement(s)

Continued...

H227	Combustible liquid
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H336	May cause drowsiness or dizziness.
H304	May be fatal if swallowed and enters airways.
H411	Toxic to aquatic life with long lasting effects.
AUH066	Repeated exposure may cause skin dryness and cracking

Precautionary statement(s) Prevention

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

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P331	Do NOT induce vomiting.
P362	Take off contaminated clothing and wash before reuse.
P363	Wash contaminated clothing before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P391	Collect spillage.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

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
64742-47-8	10-30	distillates, petroleum, light, hydrotreated
64742-48-9.	10-30	petroleum distillates HFP
Not Available	<20	conditioners, trade secret
68649-48-9	7-13	paraffin and hydrocarbon waxes, oxidised, lithium salts
19902-08-0	5-10	beta-pinene
80-56-8	5-10	alpha-pinene
63148-62-9	5-10	polydimethylsiloxane
8002-74-2	5-10	paraffin wax
Not Available	1-5	other terpenes

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ 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.

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Skin Contact	If skin contact occurs: ▸ 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. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket. ▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

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

- Lithium produces a generalised slowing of the electroencephalogram; the anion gap may increase in severe cases.
- Emesis (or lavage if the patient is obtunded or convulsing) is indicated for ingestions exceeding 40 mg (Li)/Kg.
- Overdose may delay absorption; decontamination measures may be more effective several hours after cathartics.
- Charcoal is not useful. No clinical data are available to guide the administration of catharsis.
- Haemodialysis significantly increases lithium clearance; indications for haemodialysis include patients with serum levels above 4 meq/L.
- There are no antidotes.

[Ellenhorn and Barceloux: Medical Toxicology]

In acute poisonings by essential oils the stomach should be emptied by aspiration and lavage. Give a saline purgative such as sodium sulfate (30 g in 250 ml water) unless catharsis is already present. Demulcent drinks may also be given. Large volumes of fluid should be given provided renal function is adequate. [MARTINDALE: The Extra Pharmacopoeia, 28th Ed.]

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

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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. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use water delivered as a fine spray to control fire and cool adjacent 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	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include; carbon monoxide (CO) carbon dioxide (CO_2) silicon dioxide (SiO_2) other pyrolysis products typical of burning organic material **CARE**: 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.

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	Environmental hazard - contain spillage. ▸ 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	▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services. Environmental hazard - contain spillage. **CARE**: Absorbent materials wetted with occluded oil must be moistened with water as they may auto-oxidize, become self heating and ignite. Some oils slowly oxidise when spread in a film and oil on cloths, mops, absorbents may autoxidise and generate heat, smoulder, ignite and burn. In the workplace oily rags should be collected and immersed in water.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. ▸ Electrostatic discharge may be generated during pumping - this may result in fire. ▸ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▸ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). ▸ Avoid splash filling. ▸ Do NOT use compressed air for filling discharging or handling operations. ▸ 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.

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	▸ 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.
Other information	▸ 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.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Traces of benzene, a carcinogen, may form when silicones are heated in air above 230 degrees C. Concentrated acids and bases cause degradation of polymer. Boiling water may soften and weaken material. **HAZARD**: ▸ Although anti-oxidants may be present, in the original formulation, these may deplete over time as they come into contact with air. ▸ Rags wet / soaked with unsaturated hydrocarbons / drying oils may auto-oxidise; generate heat and, in-time, smoulder and ignite. This is especially the case where oil-soaked materials are folded, bunched, compressed, or piled together - this allows the heat to accumulate or even accelerate the reaction ▸ Oily cleaning rags should be collected regularly and immersed in water, or spread to dry in safe-place away from direct sunlight.or stored, immersed, in solvents in suitably closed containers. ▸ Avoid reaction with oxidising agents

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	distillates, petroleum, light, hydrotreated	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	petroleum distillates HFP	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	paraffin wax	Paraffin wax (fume)	2 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
petroleum distillates HFP	Naphtha, hydrotreated heavy; (Isopar L-rev 2)	171 ppm	171 ppm	570 ppm
petroleum distillates HFP	Solvent naphtha, petroleum, medium aliphatic; (Mineral spirits, naphtha)	0.32 mg/m3	3.5 mg/m3	21 mg/m3
alpha-pinene	Trimethylbicyclo(3.1.1)-2-hept-2-ene, 2,6,6-; (alpha-Pinene)	22 ppm	22 ppm	130 ppm
polydimethylsiloxane	Dimethyl siloxane; (Dimethylpolysiloxane; Syltherm XLT; Syltherm 800; Silicone 360)	1.5 mg/m3	16 mg/m3	990 mg/m3
paraffin wax	Paraffin, n-	4.9 mg/m3	4.9 mg/m3	29 mg/m3

Ingredient	Original IDLH	Revised IDLH
distillates, petroleum, light, hydrotreated	Not Available	Not Available
petroleum distillates HFP	Not Available	Not Available
conditioners, trade secret	Not Available	Not Available
paraffin and hydrocarbon waxes, oxidised, lithium salts	Not Available	Not Available
beta-pinene	Not Available	Not Available
alpha-pinene	Not Available	Not Available
polydimethylsiloxane	Not Available	Not Available
paraffin wax	Not Available	Not Available
other terpenes	Not Available	Not Available

MATERIAL DATA

For kaolin:
Kaolin dust appears to have fibrogenic potential even in the absence of crystalline silica. Kaolinosis can exist as simple and complicated forms with the latter often associated with respiratory symptoms. Crystalline silica enhances the severity of the pneumoconiosis.
For paraffin waxes and hydrocarbon waxes a complex combination of hydrocarbons obtained from petroleum fractions by solvent crystallisation:
TLV TWA: 2 mg/m3
Animals exposed by inhalation to 10 mg/m3 titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

Odour threshold: 0.25 ppm.
The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m3 is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.
Odour Safety Factor (OSF)
OSF=0.042 (gasoline)
for kerosene CAS 8008-20-6
TLV TWA: 100 mg/m3 as total hydrocarbon vapour Skin A3
OEL TWA: 14 ppm, 100 mg/m3 [NIOSH, 1985]
REL TWA: 150 ppm [Shell]

Continued...

CEL TWA: 300 ppm, 900 mg/m3
(CEL = Chemwatch Exposure Limit)

for petroleum distillates:
CEL TWA: 500 ppm, 2000 mg/m3 (compare OSHA TWA)
(CEL = Chemwatch Exposure Limit)
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 P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.
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

Appropriate engineering controls

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

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection.
Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
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.

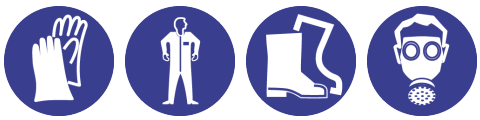
Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at 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

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

Body protection

See Other protection below

Other protection

- Overalls.
- P.V.C. apron.
- Barrier cream.
- Skin cleansing cream.

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	▸ 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
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	Gold paste with a pleasant odour; not miscible with water.

Physical state	Non Slump Paste	**Relative density (Water = 1)**	0.86
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	100 cps
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	66 (PMCC)	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Combustible.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	VOC = 65.34%
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	▸ Silicone fluids are stable under normal storage conditions. ▸ Hazardous polymerisation will not occur. ▸ At temperatures > 150 C, silicones can slowly react with the oxygen in air. ▸ When heated > 300 C, silicones can slowly depolymerise to volatile siloxanes whether or not air is present. ▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ 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	Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. 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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	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. Inhalation hazard is increased at higher temperatures. High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. 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. Inhalation of essential oil volatiles may produce dizziness, rapid, shallow breathing, tachycardia, bronchial irritation and unconsciousness or convulsions. Complications include anuria, pulmonary oedema and bronchial pneumonia. 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
Ingestion	Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Accidental ingestion of the material may be damaging to the health of the individual. Large doses of lithium ion have caused dizziness and prostration and can cause kidney damage if sodium intake is limited. Dehydration, weight-loss, dermatological effects and thyroid disturbances have been reported. Central nervous system effects that include slurred speech, blurred vision, sensory loss, impaired concentration, irritability, lethargy, confusion, disorientation, drowsiness, anxiety, spasticity, delirium, stupor, ataxia (loss of muscle coordination), sedation, fine and gross tremor, giddiness, twitching and convulsions may occur. Diarrhoea, vomiting and neuromuscular effects such as tremor, clonus (rapid contraction and relaxation of muscles) and hyperactive reflexes may occur as a result of repeated exposure to lithium. Acute severe overexposure may affect the kidneys, resulting in renal dysfunction, albuminuria, oliguria and degenerative changes. Cardiovascular effects may also result in cardiac arrhythmias and hypotension. The primary target organ for lithium toxicity is the central nervous system. Lithium is therefore used therapeutically on membrane transport proteins in the central nervous system when treating manic-depression. Lithium is moderately toxic with lethal dose of LiCl in rats of 526-840 mg/kg body weight. After chronic exposure to 1 meq/L decreased brain weight was observed in male offspring . Chemically, lithium resembles sodium, but is more toxic: in humans 5 g LiCl can result in fatal poisoning. In therapeutic doses, damages on the central nervous system and the kidneys have been reported. Terpenes and their oxygen-containing counterparts, the terpenoids, produce a variety of physiological effects. Pine oil monoterpenes, for example, produce a haemorrhagic gastritis characterised by stomach pain and bleeding and vomiting. Systemic effects of pine oils include weakness and central nervous depression, excitement, loss of balance, headache, with hypothermia and respiratory failure. Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage. Taken internally the essential oils exert a mild irritant effect on the mucous membranes of the mouth and digestive tract which induces a feeling of warmth and increases salivation. Taken by mouth, many essential oils can be dangerous in high concentrations. Typical effects begin with a burning feeling, followed by salivation. In the stomach, the effect is carminative (relieve flatulence), relaxing the gastric sphincter and encouraging eructation (belching). Further down the gut, the effect typically is antispasmodic, Excessive oral doses irritate the gastro-intestinal tract and may cause nausea, vomiting and diarrhoea. Occasional irritation of the urinary tract and aggravation of pre-existing inflammatory conditions have been reported. Other effects include dysuria, haematuria, unconsciousness and shallow respiration. Complications arising from ingestion of volatile oils include anuria, pulmonary oedema, and bronchial pneumonia. Central nervous system depression may lead to stupor and possible respiratory failure whilst central system stimulation may lead to excitement and convulsions. Pathologic findings include renal degeneration and intense congestion and oedema in the lungs, brain and gastric mucosa. Excretion takes place through the lungs, skin and kidneys. Most essential oils are reported to be ecbolic (inducing contractions of the uterus leading to expulsion of a fetus). but abortions cannot be induced at safe doses. Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Open cuts, abraded or irritated skin should not be exposed to this material It is likely that older pine oils become irritants from the build up of peroxides of delta- 3-carene and limonene etc. 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. 5510essoil Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.
Eye	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.

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	Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.
Chronic	Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Neuromuscular effects result from chronic over-exposure to lithium compounds. These may include tremor, ataxia, clonus and hyperactive reflexes. Some animal studies have shown that exposure during pregnancy may produce birth defects. Other studies with rats, rabbits and monkeys have not shown teratogenic effects. Human data are ambiguous; it is well established that lithium can cross the human placenta. Of 225 registered pregnancies in which the mothers had received lithium (as a tranquiliser) there were 25 instances of congenital malformation. Although pharmacological doses of lithium cannot be unequivocally designated as a human teratogen, lithium therapy is contraindicated in women of childbearing potential. Prolonged exposure may produce anorexia, weight loss and emaciation. The kidneys, behavioural/ central nervous system and peripheral nervous system may also show adverse effects. Various types of dermatitis (psoriasis, alopecia, cutaneous ulcers, acne, follicular papules, xerosis cutis, exfoliative) may also result from chronic skin exposure. Lithium ion can be an effective treatment for manic depression. It is thought to bind the enzyme IMPase (inositol monophosphatase) and thereby mediates its influence in producing a response to calcium-induced production of neurotransmitters and hormones thought to be responsible for the clinical picture. In subchronic studies, rats were exposed to 3 milliequivalents Li/kg/day (equivalent to 1450 mg for a 70 kg person) but did not accumulate Li whilst on a high sodium diet. However when sodium was restricted, fatal kidney toxicity developed. Dogs survived daily dose of 50 mg LiCl/kg for 150 days to the termination of the experiment on a normal sodium intake, whereas the same dose was lethal in 12 to 18 days on a low sodium diet: 20 mg LiCl/kg/day resulted in death in 18 to 30 days. 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. Essential oils and isolates derived from the Pinacea family, including Pinus and Abies genera, should only be used when the level of peroxides is kept to the lowest practicable level, for instance by adding antioxidants at the time of production. Such products should have a peroxide value of less than 10 millimoles peroxide per liter. Based on the published literature mentioning sensitising properties when containing peroxides (Food and Chemical Toxicology 11,1053(1973); 16,843(1978); 16,853(1978). In the presence of air, a number of common flavour and fragrance chemicals can form peroxides surprisingly fast. Antioxidants can in most cases minimise the oxidation. Fragrance terpenes are generally easily oxidised in air. Non-oxidised limonene, linalool and caryophyllene turned out to be very weak sensitizers, however after oxidation limonene hydroperoxide and linalool hydroperoxide are strong sensitizers. Of the patients tested 2.6% showed positive reaction to oxidised limonene, 1.3% to oxidised linalool, 1.1% to linalool hydroperoxide, 0.5% to oxidised caryophyllene, while testing with caryophyllene oxide and oxidised myrcene resulted in few positive patch tests. 2/3 of the patients reacting positive to oxidised terpenes had fragrance related contact allergy and/or positive history for adverse reactions to fragrances. As well as the hydroperoxides produced by linalol, limonene and delta-3-carene other oxidation and resinification effects progressively causes other fairly major changes in essential oil quality over time. Autoxidation of fragrance terpenes contributes greatly to fragrance allergy, which emphasizes the need of testing with compounds that patients are actually exposed to and not only with the ingredients originally applied in commercial formulations. Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

	TOXICITY	IRRITATION
Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A)	Not Available	Not Available
distillates, petroleum, light, hydrotreated	Dermal (rabbit) LD50: >2000 mg/kg[1]	Not Available
	Oral (rat) LD50: >5000 mg/kg[1]	
petroleum distillates HFP	Dermal (rabbit) LD50: >1900 mg/kg[1]	* [Shell - Canada]
	dermal (rat) LD50: 28000 mg/kg[2]	
	Oral (rat) LD50: >19650 mg/kg[2]	
	Oral (rat) LD50: >4500 mg/kg[1]	
paraffin and hydrocarbon waxes, oxidised, lithium salts	Not Available	Not Available
beta-pinene	Oral (rabbit) LD50: 4700 mg/kg[2]	Skin (rabbit):500 mg/24h-moderate
alpha-pinene	Dermal (rabbit) LD50: >2000 mg/kg[1]	Skin (man): 100% - SEVERE
	Oral (rat) LD50: 3700 mg/kg[2]	Skin (rabbit): 500 mg/24h - mod
polydimethylsiloxane	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (rabbit): 100 mg/1h - mild

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	Oral (rat) LD50: >17000 mg/kg[2]	
paraffin wax	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 100 mg/24 hr-mild
	dermal (rat) LD50: >2000 mg/kg[1]	Skin (rabbit): 500 mg/24 hr-mild
	Oral (rat) LD50: >4500 mg/kg[1]	
	Oral (rat) LD50: >4500 mg/kg[1]	

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

Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A)	Goitrogenic:. Goitrogens are substances that suppress the function of the thyroid gland by interfering with iodine uptake, which can, as a result, cause an enlargement of the thyroid, i.e., a goitre Goitrogens include: ▸ Vitexin, a flavanoid, which inhibits thyroid peroxidase thus contributing to goiter. ▸ Ions such as thiocyanate and perchlorate which decrease iodide uptake by competitive inhibition; as a consequence of reduced thyroxine and triiodothyronine secretion by the gland, at low doses, this causes an increased release of thyrotropin (by reduced negative feedback), which then stimulates the gland. ▸ Lithium which inhibits thyroid hormone release. ▸ Certain foods, such as soy and millet (containing vitexins) and vegetables in the genus Brassica (e.g. broccoli, brussels sprouts, cabbage, horseradish). ▸ Caffeine (in coffee, tea, cola, chocolate) which acts on thyroid function as a suppressant. d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine. Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs. Renal tumours induced by limonene in male rats is though to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.
PETROLEUM DISTILLATES HFP	data for CAS 64742-88-7 i.e. CCINFO record 1441735
ALPHA-PINENE	The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
POLYDIMETHYLSILOXANE	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 *in vitro* or *in vivo*. 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 stain 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 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]*
PARAFFIN WAX	"Hydrocarbon wax" describes a group of solid C20 to C36 paraffinic hydrocarbons which are not absorbed in the gastro-intestinal tract and in small quantity will pass through undigested. The widespread use in cosmetic and in cosmetic surgery over many years demonstrates the low toxicity of refined waxes and many guidelines exist for their safe use Notwithstanding this, there are occasional reports of adverse effects with these products. Subcutaneous deposits often referred to as paraffinoma, have been described frequently following injection of these materials under the skin but these are not normally associated with other progressive changes. Paraffin wax and microcrystalline were each administered orally as a solution in arachis oil to groups of 5 male and 5 female rats at dose levels of 1000 and 5000 g/kg bw. produced no clinical signs of toxicity during the seven day observation period and growth rates were normal. There were no mortalities and no macroscopic changes were observed at autopsy. Three samples of 50% paraffin in petrolatum were tested in repeated, open patch applications to 6 rabbits. Two samples produced erythema in four animals that lasted three days, and one produced erythema in one rabbit that lasted two days. A microcrystalline wax was slightly irritating, to rabbit skin, in a 24 hour occluded patch test.

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	Four 50% solutions of paraffin in petrolatum were each instilled into the eyes of six albino rabbits with no rinse. Eyes were observed for irritation for three days. Two of the samples caused mild irritation in one rabbit on day 1; the other samples were not irritating In a long-term feeding study with Sprague-Dawley rats, no wax-related effects were observed. In a series of 180-day feeding studies in rats that were performed over a period of approximately 15 years (beginning in 1955) on chewing-gum bases containing hydrocarbon wax in proportions varying from 2% to 57% of the gum base, no compound-related effects were observed. Long-term toxicity studies indicated that petroleum-derived paraffin and microcrystalline waxes are non-toxic and non-carcinogenic. Eight slack waxes and eight aromatic hydrocarbon extracts derived from the slack waxes were tested for carcinogenicity after applying these to the skin of mice.The slack waxes showed only a low order of carcinogenicity at 250 days. However by 450 days every sample of slack wax had elicited papillomas and for 5 of them cancers as well. The aromatic extracts on the other hand exhibited a greater potency. At 250 days all but one sample had produced papillomas and 5 samples had produced cancers. At 450 days all but one sample had elicited cancers and all had elicited papillomas.The authors concluded that the carcinogenicity of slack wax can be attributed to the aromatic compounds found in the oils from which the waxes were pressed and which are retained on the waxes as impurities, and.is not due to paraffins. Five petrolatum waxes were negative for local and systemic carcinogenicity or toxicity in skin-painting studies in mice and rabbits. However, wax disk implants, but not ground wax implants, were associated with the development of fibrosarcomas at the implantation site in rats. A description of the accumulation of long-chain alkanes (C29, C31, and C33) in a patient who had died of heart disease led the author to conclude that these hydrocarbons were of dietary (plant) origin as judged by the tissue distribution of the alkanes. The EU Scientific Committee for Food (SCF) reviewed the available information on mineral hydrocarbons, which included the petroleum waxes. Their opinion was published in 1995. The SCF reached the following conclusion: There are sufficient data to allow a full Group ADI (Average daily Intake)of 0-20 mg/kg bw for waxes conforming to the following specification: - · Highly refined waxes derived from petroleum based or synthetic hydrocarbon feedstocks, with viscosity not less than 11 m3/s (cSt) at 100 deg C · Carbon number not less than 25 at the 5% boiling point · Average molecular weight not less than 500 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: ▸ 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 *residual base oils* 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. 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. 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 Highly and Severely Refined Distillate Base Oils **Acute toxicity:** Multiple studies of the acute toxicity of highly & 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 >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to> 4 mg/l. 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 **Repeat dose toxicity: .** Several studies have been conducted with these oils**. T**he weight of evidence from all available data on highly & 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. ▸ 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, ▸ 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 ▸ 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. **Reproductive and developmental toxicity:** 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. 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 foetuses were found among three litters The study authors considered these malformations to be minor and within the normal ranges for the strain of rat. **Genotoxicity**: *In vitro* (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. *In vivo* (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. **Carcinogenicity:** Highly & severely refined base oils are not carcinogens, when given either orally or dermally. Tumorigenic in rats
Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A) & BETA-PINENE & ALPHA-PINENE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A) & DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED & PARAFFIN AND HYDROCARBON WAXES, OXIDISED, LITHIUM SALTS	No significant acute toxicological data identified in literature search.

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Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A) & PARAFFIN WAX	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 that iso- or cyclo-paraffins. 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.
Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A) & PETROLEUM DISTILLATES HFP	**for petroleum:** This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic. This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents **Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans. **Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays. **Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed. **Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.
Meguiar's M08 - Mirror Glaze Maximum Mold Release Wax (23-135A) & DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED	For "kerosenes" **Acute toxicity:** Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg The dermal LD50s of the same three kerosenes were all >2.0 g//kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No. 8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same material produced an LC50 of >6.4 mg/l When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced “moderate” to “severe” irritation. Six additional skin irritation studies on a range of kerosenes produced “mild” to “severe” irritation. An eye irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed and washed eyes) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurized kerosene and jet fuel. These materials produced more irritation in the unwashed eyes at 1 hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but by day 7 had resolved. Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurized kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in guinea pigs **Repeat-Dose toxicity:** Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24 respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from “slight” to “moderate” in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss. Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related: • increased relative heart weights for the mid- and high- dose males and females, • increased absolute and relative spleen weights in treated females, and • differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stress-related and therefore, indirectly related to treatment). Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group. These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes. In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Opthalomological examination of all animals also found no treatment-related effects. There were no treatment-related effects on growth rates, hematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin. A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study . In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m3 kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues. **Carcinogenicity:** In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. .Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation . If the equivalent dose of kerosene was applied to the skin in manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred . Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration . The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice . Animal survivals were not effected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity. ***In-Vitro* (Genotoxicity):** The potential *in vitro* genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation . Modified Ames assays on four kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation . The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results . Hydrodesulfurized kerosene tested in a sister chromatid exchange assay produced negative results

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	(with/without activation) ***In-Vivo* Genotoxicity:** Multiple *in vivo* genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in *in vivo* bone marrow cytogenetic tests in Sprague-Dawley rats . One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay . Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation. **Reproductive/Developmental Toxicity** Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days premating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day. Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported . There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds.
BETA-PINENE & ALPHA-PINENE	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.
BETA-PINENE & ALPHA-PINENE	Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits. Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water. Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis. Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure. **Hands:** Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear. **Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy. **Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic. **Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen. **Pigmentary anomalies:** The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil. **Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare. **General/respiratory:** Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association

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	was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.
BETA-PINENE & ALPHA-PINENE	Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. In the case of prehaptens, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers. **Prehaptens** Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxyl radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context. It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clincal studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil. **Prohaptens** Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens. In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal. The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity. **QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.
BETA-PINENE & ALPHA-PINENE	For bicyclic terpenes: **Acute toxicity**: The literature abounds with clinical reports of accidental and intentional acute poisoning with pinene-based turpentine. Rat oral LD50 values are available for *alpha*-pinene, *beta*-pinene, camphene and turpentine oil and indicate these materials to be very low in oral acute toxicity with LD50 values in the range from 3388 mg/kg to greater than 5000 mg/kg. Rabbit dermal LD50 values similarly indicate very low toxicities with values greater than the limit doses of 2000 or 5000 mg/kg. Acute inhalation toxicity has been measure in different animal species. The acute LC50 was reported to be 13,500 mg/m3 in rats, 13,500 mg/m3 in guinea pigs, and 9000 mg/m3 in mice . The acute inhalation LC50 of commercial grade turpentine in Wistar rats is reported to be in the range of 12,000-20,000 mg/m3 for 1 to 6 hour exposures and the LC50 for a 2-hour exposure in Swiss-Webster mice is 29,000 mg/m3 . Based on these results the acute oral, dermal, and inhalation toxicities of bicyclic terpene hydrocarbons is concluded to be low. **Repeat dose toxicity**: A 28-day repeat dose study has been performed with camphene according to an OECD Guideline 407 in both sexes of Wistar rats. Animals of both sexes at the 1000 mg/kg bw/day dose exhibited vacuolization of hepatocytes and increase liver weights. Male rats also exhibited *alpha*-2-microglobulin-type nephrotoxicity at all dose levels. Subsequent investigations have shown that the *alpha*-2-microglobulin nephropathy found in the F344/N male rat does not develop in mammals that do not express the hepatic form of *alpha*-2-microglobulin (e.g. other strains of rats, mice, dogs, humans). Therefore, the nephrotoxicity observed in the camphene study in male F344 rats is not relevant to the human health risk assessment. Based on liver toxicity, the NOAEL for this study is concluded to be 250 mg/kg bw/day **Reproductive toxicity:** In the a-animal species study, no reproductive effects were observed when dose levels of up to 260 to 600 mg/kg bw of an essential oil predominantly composed of bicyclic terpene hydrocarbons (*alpha*-pinene, *beta*-pinene, and sabinene) was administered daily to mice, rats, or hamsters during gestation. When this data is combined with the fact that no adverse effects were observed to the reproductive organs in a 28-day study with camphene at dose levels up to 250 mg/kg bw/day, it is concluded that bicyclic terpene hydrocarbons including *alpha*-pinene and *beta*-pinene are not reproductive toxicants Two ninety day inhalation studies have been performed for alpha-pinene in which a full complement of male and female sex organs and tissues were subjected to histopathological examination. Both studies reported no microscopic changes that could be associated with exposure to the test substance. Taking into account the lack of any effects to females in a earlier teratology study, the absence of any maternal or developmental effects in a reproductive/developmental study of a pinene-based oil and for a structurally related monoterpene hydrocarbon, myrcene, it can be concluded that the members of this category show no significant reproductive or developmental toxicity **Developmental toxicity:.** Based on the NOAELs for maternal and developmental toxicity in studies with camphene (250 and 1000 mg/kg bw/day) and a terpene hydrocarbon mixture containing *alpha*- and *beta*-pinene and camphene (688 mg/kg bw/day) , and the lack of any signs of maternal or developmental toxicity in a mice, rats, or hamsters given 260 to 600 mg/kg bw/day of a mixture composed primarily (>80%) of *alpha*- and *beta*-pinene and sabinene, it is concluded that bicyclic terpene hydrocarbons are not maternal or developmental toxicants. **Genotoxicity:** ***In vitro:*** *In vitro* genotoxicity assays available for *alpha*-pinene, *beta*-pinene and camphene demonstrate that these substances have a little, if any, genotoxic potential. In standard Ames assays of *alpha*-pinene, *beta*-pinene and camphene, *Salmonella typhimurium* strains TA97, TA98, TA100, TA1535, TA1537, and TA1538 provided no evidence of mutagenicity at any dose tested. ***In vivo:*** Based on the lack of any evidence of genotoxicity in numerous *in vitro* assays with and without metabolic activation, it is unlikely that any of these

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	bicyclic terpenes would exhibit a significant genotoxic potential *in vivo*.
BETA-PINENE & ALPHA-PINENE	A member or analogue of a group of of aliphatic and aromatic terpene hydrocarbons generally considered as safe (GRAS) based, in part, on their self-limiting properties as flavouring substances in food; their rapid absorption, metabolic detoxication, and excretion in humans and other animals; their low level of flavour use; the wide margins of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from subchronic and chronic studies and the lack of significant genotoxic potential. Consumers are exposed to aliphatic and terpene hydrocarbons from a variety of ingested and environmental source. Quantitative natural occurrence data for 17 aliphatic terpene hydrocarbons in the group demonstrate that their consumption occurs predominantly as natural components of traditional food . Oral LD50 values have been reported for 16 of the 17 substances in this group. LD50 values range from 1590 to greater than 8000 mg/kg bw in rats, and 2000 to greater than 13,360 mg/kg bw in mice. These values indicate that aliphatic and aromatic hydrocarbons exhibit low acute oral toxicity. Although members of this group have been shown to exhibit renal carcinogenic potential in the male F344N/rat, the mechanism leading to these findings is known and strongly indicates that the nephropathy associated with monoterpene hydrocarbons have no significance for human risk. Flavor and Extracts Manufacturers Manufacturers Association (FEMA)

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 required to make classification available*
⊘ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
distillates, petroleum, light, hydrotreated	LC50	96	Fish	2.2mg/L	4
distillates, petroleum, light, hydrotreated	NOEC	3072	Fish	=1mg/L	1
petroleum distillates HFP	EC50	96	Algae or other aquatic plants	64mg/L	2
petroleum distillates HFP	EC50	48	Crustacea	>100mg/L	1
petroleum distillates HFP	EC50	96	Algae or other aquatic plants	=450mg/L	1
beta-pinene	EC50	384	Crustacea	0.113mg/L	3
beta-pinene	EC50	96	Algae or other aquatic plants	0.563mg/L	3
beta-pinene	LC50	96	Fish	0.445mg/L	3
alpha-pinene	NOEC	96	Crustacea	=0.18mg/L	1
alpha-pinene	EC50	384	Crustacea	0.129mg/L	3
alpha-pinene	EC50	96	Algae or other aquatic plants	0.663mg/L	3
alpha-pinene	LC50	96	Fish	0.28mg/L	2
polydimethylsiloxane	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 - 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*				

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the
oxygen transfer between the air and the water
Oils of any kind can cause:

- drowning of water-fowl due to lack of buoyancy, loss of insulating capacity of feathers, starvation and vulnerability to predators due to lack of mobility
- lethal effects on fish by coating gill surfaces, preventing respiration
- asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom and
- adverse aesthetic effects of fouled shoreline and beaches

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.
For kerosene:
For kerosene-range refinery streams ("kerosene"):
Kerosene is the name for the lighter end of a group of petroleum streams known as the middle distillates.
Kerosene may be obtained either from the distillation of crude oil under atmospheric pressure (straight-run kerosene) or from catalytic, thermal or steam cracking of heavier petroleum streams (cracked kerosene). The kerosenes, are further treated by a variety of processes (including hydrogenation) to remove or reduce the level of sulfur, nitrogen or olefinic materials. The precise composition of any particular kerosene will depend on the crude oil from which it was derived and on the refinery processes used for its production.
The streams are complex mixtures of paraffinic, isoparaffinic, naphthenic (cycloparaffinic) and aromatic (mainly alkylbenzene) hydrocarbons ranging in carbon number from C5-25 (mainly C9-16) and boil in the range 145 to 300 C. Olefins constitute less than 5% of the mixtures, by volume, and polycyclic aromatic hydrocarbons (PAHs) (3-7 fused rings) content is typically very low. Jet fuels (e.g., Jet A, JP-8, etc.) are included because they are composed almost entirely of two of these streams straight run kerosene (CAS No. 8008-20-6) or hydrodesulfurised kerosene (CAS No. 64742-81-0)
Environmental Fate
Terrestrial fate:: If released to soil, kerosene is expected to biodegrade under both aerobic and anaerobic conditions. Kerosene is a mixture of petroleum hydrocarbons, chiefly C10-C16 alkanes, and a typical analysis includes the identification of n-dodecane, alkyl benzene derivatives, naphthalene, and tetrahydronaphthalenes. Soil adsorption coefficients for these representative classes of compounds ranging from 1500 to 17,000 obtained from estimated log octanol/water partition coefficients of 3.3 to 5.25 indicate that some components of kerosene may display low mobility and some will be essentially immobile in soil. The vapour pressure of kerosene, 0.48 mm Hg indicates that it may rapidly volatilise from dry soil to the atmosphere although its expected strong adsorption to

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soil may significantly attenuate the rate of this process.
Aquatic fate: : If released to water, kerosene is expected to biodegrade under both aerobic and anaerobic conditions. Bioconcentration factors for components of kerosene were estimated to be 190 to 5800 (based on estimated log octanol/water partition coefficients of 3.3 to 5.25) indicating that some components of kerosene may significantly bioconcentrate in fish and aquatic organisms. Soil adsorption coefficients for kerosene ranging from 1500 to 17,000 indicate that it may strongly adsorb to sediment and suspended organic matter. The estimated half-life for volatilisation of kerosene from a model river 1 m deep flowing at 1 m/sec with a wind speed of 3 m/sec which does not take into account adsorptive processes is 3-6 hrs. The estimated half-life for volatilisation of kerosene from a model lake, which accounts for adsorptive processes, is >130 days.
Atmospheric fate If released to the atmosphere, kerosene may undergo oxidation by a gas-phase reaction with photochemically produced hydroxyl radicals. Estimated rate constants for the oxidation of these representative classes of compounds ranging from 1.2-2.2x10-11 cm/molec-sec at 25 deg C translates to an atmospheric half-life for kerosene of 2-3.4 days using an average atmospheric hydroxyl radical concentration of 5x10+5 molec/cu-cm.
The stability of kerosene in soils as affected by volatilization was determined in a laboratory column experiment by following the losses in the total concentration and the change in composition of the residuals in a dune sand, a loamy sand, and a silty loam soil during a 50 day period. Seven major compounds ranging between C9 and C15 were selected from a large variety of hydrocarbons forming kerosene and their presence in the remaining petroleum product was determined. The change in composition of kerosene during the experimental period was determined by gas chromatography and related to the seven major compounds selected. The experimental conditions air-dry soil and no subsequent addition of water excluded both biodegradative and leaching losses. The losses of kerosene in air-dried soil columns during the 50-day experimental period and the changes in the composition of the remaining residues due to volatilization are reported. The volatilization of all the components determined was greater from the dune sand and loamy sand soils than from the silty loam soil. It was assumed that the reason for this behavior was that the dune sand and the loamy sand soils contain a greater proportion of large pores (> 4.5 um) than the silty loam soil, even though the total porosity of the loamy sand and the silty loam is similar. In all the soils in the experiment, the components with a high carbon number formed the main fraction of the kerosene residues after 50 days of incubation.
Voltatilisation in the air phase and saturated mass flow of kerosene in the three sands ((fine, medium and coarse) were studied in the laboratory under controlled conditions. Volatilisation was the major physico-chemical process affecting the fate of kerosene in the inert porous medium. During volatilization the liquid kerosene changed its composition by gradually losing its light components (C9-C13), and the viscosity of the remaining liquid kerosene increased. The increase in viscosity led to a decrease in the infiltration rate, for example, by about 20% when the viscosity increased
Ecotoxicity:
Data for various kerosene streams is available. Kerosenes and jet fuels are moderately to acutely toxicity to aquatic organisms All studies used exposures to water accommodated fractions (WAFs) of the process streams Each of the different streams exhibited similar toxicity to rainbow trout (*Oncorhynchus mykiss*, 96-hour LC 50 values of 18 - 25 mg/L); likewise, toxicity to the alga *Selenastrum capricornutum*, with 96-hour growth rate EC50 values of 5.0 - 6.2 mg/L and biomass inhibition EC50 values of 5.9 - 11 mg/L, did not vary greatly among the streams. There was considerable variation in the measured toxicity of the category member (CAS No. 64742-81-0) to daphnids (*Daphnia magna*) when evaluated in different tests; in the test using daily renewal of freshly-prepared WAF , the 48-hr EC50 was estimated at 1.4 mg/L, while in the test where solution was not renewed it was estimated at between 40 and 89 mg/L. In spite of daily renewal, a sample of sweetened kerosene (CAS No. 91770-15-9) exhibited considerably less toxicity than the hydrodesulfurised and hydrocracked materials tested in the same laboratory , indicating the difference in that measurement is due to the nature of the sample rather than variations in the testing approach.
For siloxanes:
Environmental fate:
It is well accepted that polydimethylsiloxane fluids become permanent residents of sediment but should not exert adverse environmental effects.
Silicone fluids are very surface active because the flexible siloxane linkages permit alignment of the hydrophobic methyl substituents towards the non-polar phase, and of the polysiloxane backbone towards the polar phase. The polar medium is generally water, and a polar media to which polydimethylsiloxanes become attached may be textiles, sewage sludge, hair, algae, sediment etc. In aqueous environments, polydimethylsiloxanes are adsorbed onto sedimenting particles. Also, in the presence of nitrate ions, which exist at various concentrations in the environment, short chain siloxanes are photodegraded to the level of silicate within days
The stability of the siloxanes, desirable from a technical point of view, makes the siloxanes very persistent, and once released to the environment the siloxanes remain for many years.
The main source of releases of siloxanes to the air is volatile siloxanes used in cosmetics, wax, polishes, and to a minor extent in several other applications. the volatile siloxanes may account for a significant part of the siloxanes used for cosmetics.
Non-volatile silicone fluids used in cosmetics, wax, polishes, cleaning products and for textile applications (softeners) will to a large extent end up in wastewater and be directed to wastewater treatment plants.
The cyclic siloxanes and small-chain linear siloxanes are bioconcentrated (bioconcentration factors for long-chained siloxanes have not been assessed). The estimated bioconcentration factors (BCF) of the small siloxanes range from 340 for HMDS to 40,000 for a phenylated trisiloxane (phenyl trimethicone). The small phenylated siloxanes seem to have very high BCF, and model estimates indicate that these substances are the most toxic for aquatic organisms.
PBT profiler screening
In order to make a first comparison between the substances as to persistence, bioaccumulation and toxicity, the substances were screened using the PBT profiler developed by U.S. EPA (U.S. EPA 2003). The profiler uses a procedure to predict persistence, bioaccumulation, and toxicity of organic chemicals on the basis of the chemical structure and physical
parameters of the substances combined with experimental parameters for substance with a similar structure, using a QSAR approach.
The results for six members of the siloxane family predict the highest bioconcentration factors for the two phenyl siloxanes, one order of magnitudes higher than the values for the cyclic siloxanes and two orders of magnitudes higher than the values for the small linear methyl siloxanes. The predicted toxicity is as well significantly higher (lowest ChV values) for the phenyl siloxanes. The predicted half-life is nearly the same for all substances.
Using U.S. EPA's criteria, the screening indicates that all substances are of high concern as to environmental toxicity, and that the phenyl siloxanes are considered very bioaccumulative.
Ecotoxicity:
The environmental fate and effects of volatile methylsiloxanes (mainly cyclosiloxanes) and polydimethylsiloxane (PDMS) have been reported:
For octamethylcyclosiloxane:
Fish acute LC50 (14 day):: rainbow trout 10 ug/l; sheepshead minnow >6.3 ug/l
Daphnia magna acute EC50 (48 h): >15 ug/l; NOEC 15 ug/l
Mysid shrimp acute LC50 (96 h): >9.1 ug/l; NOEC 9.1 ug/l
For PDMS
Daphnia magna NOEC 572 mg/kg
Physical effects such as surface entrapment have been observed when testing aquatic invertebrates in clean laboratory water, but similar effects are not expected in natural environments where a large variety of other surfaces provide opportunities for deposition
for lubricating oil base stocks:
Vapor Pressure Vapor pressures of lubricating base oils are reported to be negligible. In one study, the experimentally measured vapour pressure of a solvent-dewaxed heavy paraffinic distillate base oil was 1.7 x 10exp-4 Pa . Since base oils are mixtures of C15 to C50 paraffinic, naphthenic, and aromatic hydrocarbon isomers, representative components of those structures were selected to calculate a range of vapor pressures. The estimated vapor pressure values for these selected components of base oils ranged from 4.5 x 10exp-1 Pa to 2 x 10exp-13Pa. Based on Dalton's Law the expected total vapour pressure for base oils would fall well below minimum levels (10exp-5 Pa) of recommended experimental procedures.
Partition Coefficient (log Kow): In mixtures such as the base oils, the percent distribution of the hydrocarbon groups (i.e., paraffins, naphthenes, and aromatics) and the carbon chain lengths determines in-part the partitioning characteristics of the mixture. Generally, hydrocarbon chains with fewer carbon atoms tend to have lower partition coefficients than those with higher carbon numbers .However, due to their complex composition, unequivocal determination of the log Kow of these hydrocarbon mixtures cannot be made. Rather, partition coefficients of selected C15 chain-length hydrocarbon structures representing paraffinic, naphthenic, and aromatic constituents in base oil lubricants were modelled . Results showed typical log Kow values from 4.9 to 7.7, which were consistent with values of >4 for lubricating oil basestocks
Water Solubility: When released to water, base oils will float and spread at a rate that is viscosity dependent. While water solubility of base oils is typically very low, individual hydrocarbons exhibit a wide range of solubility depending on molecular weight and degree of unsaturation. Decreasing molecular weight (i.e., carbon number) and increasing levels of unsaturation increases the water solubility of these materials. As noted for partition coefficient, the water solubility of lubricating base oils cannot be determined due to their complex mixture characteristics. Therefore, the water solubility of individual C15 hydrocarbons representing the different groups making up base oils (i.e., linear and branched paraffins, naphthenes, and aromatics) was modelled. Based on water solubility modelling of those groups, aqueous solubilities are typically much less than 1 ppm. (0.003-0.63 mg/l)
Environmental Fate:
Photodegradation: Chemicals having potential to photolyse have UV/visible absorption maxima in the range of 290 to 800 nm. Some chemicals have absorption maxima significantly below 290 nm and consequently cannot undergo direct photolysis in sunlight (e.g. chemicals such as alkanes, alkenes, alkynes, saturated alcohols, and saturated acids). Most hydrocarbon constituents of the materials in this category are not expected to photolyse since they do not show absorbance within the 290-800 nm range. However, photodegradation of polyaromatic hydrocarbons (PAHs) can occur and may be a significant degradation pathway for these constituents of lubricating base oils. The degree and rate at which PAHs may photodegrade depend upon whether conditions allow penetration of light with sufficient energy to effect a change. For example,polycyclic aromatic compounds (PAC) compounds bound to sediments may persist due to a lack of sufficient light penetration
Atmospheric gas-phase reactions can occur between organic chemicals and reactive molecules such as photochemically produced hydroxyl radicals, ozone and nitrogen oxides. Atmospheric oxidation as a result of radical attack is not direct photochemical degradation, but indirect degradation. In general, lubricating base oils have low vapour pressures and volatilisation is not expected to be a significant removal mechanism for the majority of the hydrocarbon components. However, some components (e.g., C15 branched paraffins and naphthenes) appear to have the potential to volatilise Atmospheric half-lives of 0.10 to 0.66 days have been calculated for representative C15 hydrocarbon components of lubricating base oils

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Stability in Water: Chemicals that have a potential to hydrolyze include alkyl halides, amides, carbamates, carboxylic acid esters and lactones, epoxides, phosphate esters, and sulfonic acid esters. Because lubricating base oils do not contain significant levels of these functional groups, materials in the lubricating base oils category are not subject to hydrolysis
Chemical Transport and Distribution in the Environment : Based on the physical-chemical characteristics of component hydrocarbons in lubricating base oils, the lower molecular weight components are expected to have the highest vapour pressures and water solubilities, and the lowest partition coefficients. These factors enhance the potential for widespread distribution in the environment. To gain an understanding of the potential transport and distribution of lubricating base oil components, the EQC (Equilibrium Criterion) model was used to characterize the environmental distribution of different C15 compounds representing different structures found in lube oils (e.g., paraffins, naphthenes, and aromatics). The modelling found partitioning to soil or air is the ultimate fate of these C15 compounds. Aromatic compounds partition principally to soil. Linear paraffins partition mostly to soil, while branching appears to allow greater distribution to air. Naphthenes distribute to both soil and air, with increasing proportions in soil for components with the greater number of ring structures. Because the modelling does not take into account degradation factors, levels modelled in the atmosphere are likely overstated in light of the tendency for indirect photodegradation to occur.
Biodegradation: The extent of biodegradation measured for a particular lubricating oil basestock is dependent not only on the procedure used but also on how the sample is presented in the biodegradation test. Lubricant base oils typically are not readily biodegradable in standard 28-day tests. However, since the oils consist primarily of hydrocarbons that are ultimately assimilated by microorganisms, and therefore inherently biodegradable. Twenty-eight biodegradability studies have been reported for a variety of lubricating base oils. Based on the results of ultimate biodegradability tests using modified Sturm and manometric respirometry testing the base oils are expected to be, for the most part, inherently biodegradable. Biodegradation rates found using the modified Sturm procedure ranged from 1.5 to 29%. Results from the manometric respirometry tests on similar materials showed biodegradation rates from 31 to 50%. Biodegradation rates measured in 21-day CEC tests for similar materials ranged from 13 to 79%.
Ecotoxicity:
Numerous acute studies covering fish, invertebrates, and algae have been conducted to assess the ecotoxicity of various lubricating base oils. None of these studies have shown evidence of acute toxicity to aquatic organisms. Eight, 7-day exposure studies using rainbow trout failed to demonstrate toxicity when tested up to the maximum concentration of 1000 mg/L applied as dispersions. Three, 96-hour tests with rainbow trout also failed to show any toxic effects when tested up to 1000 mg/L applied as dispersions. Similarly, three 96-hour tests with fathead minnows at a maximum test concentration of 100 mg/L water accommodated fractions (WAF) showed no adverse effects. Two species of aquatic invertebrates (Daphnia magna and Gammarus sp.) were exposed to WAF solutions up to 10,000 mg/L for 48 and 96-hours, respectively, with no adverse effects being observed. Four-day exposures of the freshwater green alga (Scenedesmus subspicatus) to 500 mg/L WAF solutions failed to show adverse effects on growth rate and algal cell densities in four studies
Multiple chronic ecotoxicity studies have shown no adverse effects to daphnid survival or reproduction. In 10 of 11 chronic studies, daphnids were exposed for 21 days to WAF preparations of lubricating base oils with no ill effects on survival or reproduction at the maximum concentration of 1000 mg/L. One test detected a reduction in reproduction at 1000 mg/L. Additional data support findings of no chronic toxicity to aquatic invertebrates and fish. No observed effect levels ranged from 550 to 5,000 mg/L when tested as either dispersions or WAFs.
The data described above are supported by studies on a homologous series of alkanes. The author concluded that the water solubility of carbon chains .C10 is too limited to elicit acute toxicity. This also was shown for alkylbenzene compounds having carbon numbers .C15. Since base oils consist of carbon compounds of C15 to C50, component hydrocarbons that are of acute toxicological concern are, for the most part, absent in these materials. Similarly, due to their low solubility, the alkylated two to three ring polyaromatic components in base oils are not expected to cause acute or chronic toxicity. This lack of toxicity is borne out in the results of the reported studies.
The effects of crude and refined oils on organisms found in fresh and sea water ha been extensively reviewed.
sea water. Where spillages occur the non-mobile species suffer the greatest mortality, whereas fish species can often escape from the affected region. The extent of the initial mortality depends on the chemical nature of the oil, the location, and the physical conditions, particularly the temperature and wind velocity. Most affected freshwater and marine communities recover from the effects of an oil spill within a year. The occurrence of biogenic hydrocarbons in the world's oceans is well recorded. They have the characteristic isoprenoid structure, and measurements made in water columns indicate a background concentration of 1.0 to 10 ul/l. The higher molecular weight materials are dispersed as particles, with the highest concentrations of about 20 ul/l occurring in the top 3 mm layer of water.
A wide variation in the response of organisms to oil exposures has been noted. The larvae of fish and crustaceans appear to be most susceptible to the water-soluble fraction of crude oil.
Exposures of plankton and algae have indicated that certain species of diatoms and green algae are inhibited, whereas microflagellates are not.
For the most part, molluscs and most intertidal worm species appear to be tolerant of oil contamination.
For bicyclic monoterpenes:
Photodegradation: The calculated photodegradation half-lives for the structurally defined materials in this group are in the range from 1.4 to 9.4 hours. These calculations are based on measured OH rate constants for *alpha*-pinene, *beta*-pinene, camphene and *trans*-pinane, measured ozone and NO3 rate constants with the exception of *trans*-pinane.
Stability In Water: No hydrolysis is possible for any of the materials in this group. All are expected to be very stable in aqueous solution.
Biodegradation: Studies evaluating biodegradability are available for this group of substances using standard OECD Guideline protocols. Additional studies in soil horizons obtained from coniferous and deciduous forests provide a broader perspective on the biodegradation of bicyclic terpene hydrocarbons in the environment.. Four studies on *alpha*-pinene showed limited biodegradability. The first, evaluated inherent biodegradability, and reported 37% biodegradation at 31 days ; the second, evaluated ready biodegradability, and reported 38% biodegradation at 28 days ; and a third,evaluated ready biodegradability using a mixture mainly of *alpha* and *beta*-pinene in a closed bottle test, reported very limited biodegradability In the fourth experiment, a mixture of 50.9% *alpha*-pinene and 36.8% *beta*-pinene was concluded to be inherently biodegradable based on the results of a closed bottle Sturm test. The mixture was 52% biodegraded within 28 days, but there was no indication that biodegradation had ceased.
Very limited biodegradability was also reported for 3-carene and for camphene (less than 20%). In studies showing limited biodegradability, the authors concluded that the high vapor pressure and low water solubility of these substances led to volatilization of the test substance in the upper parts of the test vessel, thereby, limiting aerobic biodegradation.
Additional studies in extracts and slurries prepared from soils of coniferous and deciduous forest indicate rapid and complete biodegradation of *alpha*-pinene in a closed bottle test. Soil extracts from coniferous and hardwood watersheds were added to sealed flasks containing oxygen-saturated media that were preconditioned with *alpha*-pinene for 24 hours. *alpha*-Pinene underwent 100% biodegradation after approximately 8 days in acclimated medium and after day 15 in non-acclimated medium. The authors concluded the pinene is completely degradable in extracts prepared from watershed soils of coniferous or deciduous forests.
Ecotoxicity:
Fish LC50 (96 h): fathead minnow 0.28 mg/l (alpha-pinene); 0.5 mg/l (beta-pinene); Brachydanio rerio 0.72 mg/l (camphene) (closed system flow through).
The calculated values for camphene, *cis*-pinane, dihydropinene, and *l-alpha*-pinene, are 0.62, 0.63, 0.63 and 0.28 mg/l, respectively. These values indicate that all of these materials and mixtures that are made up primarily of these substances, should have acute fish toxicities on the order of 0.5 mg/l.
Daphnia magna LC50 (48 h): 1.44 mg/l (alpha-pinene); 1.256 mg/l (beta-pinene)
The calculated values for camphene, *cis*-pinane, dihydropinene, and *l-alpha*-pinene, 0.79, 0.8, 0.8 and 0.22 mg/l, respectively, indicates that all of these materials and mixtures that primarily are made up of these substances, should all have acute aquatic invertebrate toxicities on the order of 1.0 mg/l.
The 96-hour calculated values for camphene, *cis*-pinane, dihydropinene, and *l-alpha*-pinene, 0.56, 0.57, 0.57 and 0.22 mg/l, respectively, indicates that all of these materials and mixtures that primarily are made up of these substances, should all have acute aquatic plant toxicity on the order of 0.5 mg/l.
Terpenes such as limonene and isoprene contribute to aerosol and photochemical smog formation. Emissions of biogenic hydrocarbons, such as the terpenes, to the atmosphere may either decrease ozone concentrations when oxides of nitrogen are low or, if emissions take place in polluted air (i.e containing high concentrations of nitrogen oxides), leads to an increase in ozone concentrations. Lower terpenoids can react with unstable reactive gases and may act as precursors of photochemical smog therefore indirectly influencing community and ecosystem properties.
Complex chlorinated terpenes such as toxaphene (a persistent, mobile and toxic insecticide) and its degradation products, were produced by photoinitiated reactions in an aqueous system, initially containing limonene and other monoterpenes, simulating pulp bleach conditions
The reactions of ozone with larger unsaturated compounds, such as the terpenes can give rise to oxygenated species with low vapour pressures that subsequently condense to form secondary organic aerosol.
Substances containing unsaturated carbons are ubiquitous in indoor environments. They result from many sources (see below). Most are reactive with environmental ozone and many produce stable products which are thought to adversely affect human health.The potential for surfaces in an enclosed space to facilitate reactions should be considered.

Source of unsaturated substances	Unsaturated substances (Reactive Emissions)	Major Stable Products produced following reaction with ozone.
Occupants (exhaled breath, ski oils, personal care products)	Isoprene, nitric oxide, squalene, unsaturated sterols, oleic acid and other unsaturated fatty acids, unsaturated oxidation products	Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.
Soft woods, wood flooring, including cypress, cedar and silver fir boards, houseplants	Isoprene, limonene, alpha-pinene, other terpenes and sesquiterpenes	Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles
Carpets and carpet backing	4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters	Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonenal
Linoleum and paints/polishes containing linseed oil	Linoleic acid, linolenic acid	Propanal, hexanal, nonanal, 2-heptenal, 2-nonenal, 2-decenal, 1-pentene-3-one, propionic acid, n-butyric acid
Latex paint	Residual monomers	Formaldehyde
Certain cleaning products, polishes, waxes, air fresheners	Limonene, alpha-pinene, terpinolene, alpha-terpineol, linalool, linalyl acetate and other terpenoids, longifolene and other sesquiterpenes	Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and organic peroxides, acetone, benzaldehyde, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H)-furanone, 4-AMC, SOAs including ultrafine particles
Natural rubber adhesive	Isoprene, terpenes	Formaldehyde, methacrolein, methyl vinyl ketone

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Photocopier toner, printed paper, styrene polymers	Styrene	Formaldehyde, benzaldehyde
Environmental tobacco smoke	Styrene, acrolein, nicotine	Formaldehyde, benzaldehyde, hexanal, glyoxal, N-methylformamide, nicotinaldehyde, cotinine
Soiled clothing, fabrics, bedding	Squalene, unsaturated sterols, oleic acid and other saturated fatty acids	Acetone, geranyl acetone, 6MHO, 40PA, formaldehyde, nonanal, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid
Soiled particle filters	Unsaturated fatty acids from plant waxes, leaf litter, and other vegetative debris; soot; diesel particles	Formaldehyde, nonanal, and other aldehydes; azelaic acid; nonanoic acid; 9-oxo-nonanoic acid and other oxo-acids; compounds with mixed functional groups (=O, -OH, and -COOH)
Ventilation ducts and duct liners	Unsaturated fatty acids and esters, unsaturated oils, neoprene	C5 to C10 aldehydes
"Urban grime"	Polycyclic aromatic hydrocarbons	Oxidized polycyclic aromatic hydrocarbons
Perfumes, colognes, essential oils (e.g. lavender, eucalyptus, tea tree)	Limonene, alpha-pinene, linalool, linalyl acetate, terpinene-4-ol, gamma-terpinene	Formaldehyde, 4-AMC, acetone, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H) furanone, SOAs including ultrafine particles
Overall home emissions	Limonene, alpha-pinene, styrene	Formaldehyde, 4-AMC, pinonaldehyde, acetone, pinic acid, pinonic acid, formic acid, benzaldehyde, SOAs including ultrafine particles

Abbreviations: 4-AMC, 4-acetyl-1-methylcyclohexene; 6MHQ, 6-methyl-5-heptene-2-one, 4OPA, 4-oxopentanal, SOA, Secondary Organic Aerosols
Reference: Charles J Weschler; Environmental Helath Perspectives, Vol 114, October 2006
For alkenes (olefins)
Environmental fate:
Ecotoxicity studies conducted with a wide range of products have shown little potential for toxicity to aquatic organisms under expected conditions of use or in the event of an accidental release. Not all alpha olefins are readily biodegradable; however, they will ultimately biodegrade. While the octanol/water partition coefficients of alpha olefins suggest a potential for bioaccumulation of these materials in aquatic organisms, the volatility of these materials (especially for the liquid alpha olefins) and the low-water solubility (indicative of limited bioavailability), would indicate that bioaccumulation will not occur. Under most environmental scenarios, extensive evaporation and subsequent degradation in the atmosphere would preclude bioaccumulation. Therefore, alpha olefins are not expected to be toxic to aquatic organisms, will biodegrade, and will not bioaccumulate
The potential for exposure of aquatic organisms to members of the higher olefins will be influenced by their physico-chemical properties. The predicted or measured water solubilities of these olefins range from 50 mg/L at 20 C for hexene to 0.00015 mg/L at 25 C for 1-octadecene, and to 6.33 [E-23] mg/L at 25 C for C54 alpha olefin, which suggests there is a lower potential for the larger olefins to be bioavailable to aquatic organisms due to their low solubilities. Their vapor pressures range from 230.6 hPa at 25 C for hexene to 0.00009 hPa at 25 C for 1-octadecene, and to 1.13 [E-16] hPa at 25 C for C54 alpha olefin, which suggests the shorter chain olefins will tend to partition to the air at a significant rate and not remain in the other environmental compartments for long periods of time; while the longer chain olefins will tend to partition primarily to water, soil or sediment, depending on water solubility and sorption behavior. The predicted soil adsorption coefficients (Koc) range from 149 for C6 to 230,800 for C18 and to 1.0 [E10] for C54, indicating increasing partitioning to soil/sediment with increasing carbon number. Level I fugacity modelling predicts that the C6-13 olefins would partition primarily to air, while the C16 and longer chain olefins would partition primarily to soil. Results of Level III fugacity modelling suggest that the C6 -8 olefins will partition primarily to the water compartment; and, as the chain length increases beyond C10, soil and sediment become the primary compartments. These chemicals have a very low potential to hydrolyse and do not photodegrade directly. However, in the air, all members of the category are subject to atmospheric oxidation from hydroxyl radical attack, with calculated degradation half- lives of 1.8 to 4.8 hours. C6 -30 olefins have been shown to degrade to an extent of approximately 8-92% in standard 28 day biodegradation tests. These results were not clearly correlated with carbon number or any other identifiable parameter; however, the weight of evidence shows that the members of the higher olefins have potential for degradation in the environment. Volatilisation from water is predicted to occur rapidly (hours to days), with Henry's Law Constants (bond method) ranging from 0.423 (C6) to 10.7 (C18), and to 2.89 [E5] (C54) atm- m3/mol. Consideration of these degradation processes supports the assessment that these substances will degrade relatively rapidly in the environment and not persist. Based on calculated bioconcentration factors, the C6, C7, and C16 and longer chain length category members are not expected to bioaccumulate (BCF: C6 = 44-46, C7 = 236, C16 = 71-92 and >= C18 = 3.2-4.6). Although the C8 - 15 olefins have BCFs ranging from 313 to 2030, and Kow values ranging from 4.13 to 7.49, and thus are considered to have the potential for bioaccumulation, their physico-chemical properties and fate indicate that there would be limited environmental exposure because of volatility, biodegradability and limited solubility.
Ecotoxicity:
Data indicate that acute aquatic toxicity can be observed for C6 through the C10 olefins (C6: EC/LC50 range of 1-10 mg/L; C7-C10: EC/LC50 range of 0.1-1.0 mg/L), and that toxicity increases with increasing carbon number within that range, which is consistent with increasing Kow values (3.07 -5.12). Above a chain length of 10, toxicity is not observed within the limits of solubility. However, data indicate that chronic aquatic toxicity can be observed in the C10 olefins (EC10 = 20.0 ug/L, EC50= 28.1 ug/L, NOEC = 19.04 ug/L). Data also suggest that aquatic toxicity does not differ with bond location or presence of branching.
For lithium (anion):
Environmental fate:
Experiments with experimental animals have shown that lithium can have reprotoxic effects, and increasing consumption might therefore result in adverse effects on health and environment. Lithium has significant bioavailability only when administered as a partially soluble salt such as lithium carbonate. Lithium is not a dietary mineral for plants but it does stimulate plant growth.
Ecotoxicity:
Fish LC50 (28, 35 days) rainbow trout 9.28, 1.4 mg/l (salt)
Fish LC50 (96 h): fathead minnow 42 mg/l; NOEC 13 mg/l (salt)
Daphnia magna EC50 (48 h): 24 mg/l; NOEC 11 mg/l
Lithium is not expected to bioaccumulate in mammals and its human and environmental toxicity are low. Lithium does accumulate in several species of fish, molluscs and crustaceans where it stored in the digestive tract and exoskeleton
Methanogenesis of granular anaerobic sludge (initial COD 5750 mg/l O2, pH 7.2) was stimulated at lithium ion concentration 10-20 mg/l, slightly inhibited at lithium ion concentration 350 mg/l and seriously inhibited at lithium ion concentration > 500 mg/l.
Microinjection of lithium chloride into prospective ventral blastomeres of a 32-cell Xenopus larvis embryo gives rise to duplication of dorsoanterior structures such as the notochord, neural tube and eyes.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
beta-pinene	HIGH	HIGH
alpha-pinene	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
distillates, petroleum, light, hydrotreated	LOW (BCF = 159)
beta-pinene	MEDIUM (LogKOW = 4.16)
alpha-pinene	MEDIUM (LogKOW = 4.44)

Mobility in soil

Ingredient	Mobility
beta-pinene	LOW (KOC = 1204)
alpha-pinene	LOW (KOC = 1204)

SECTION 13 DISPOSAL CONSIDERATIONS

Continued...

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ **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.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains beta-pinene and alpha-pinene)
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

UN number	3082
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains beta-pinene and alpha-pinene)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A97 A158 A197 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 Passenger and Cargo Maximum Qty / Pack: 450 L Passenger and Cargo Limited Quantity Packing Instructions: Y964 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3082

Continued...

UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains beta-pinene and alpha-pinene)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-A, S-F Special provisions: 274 335 969 Limited Quantities: 5 L

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

DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED(64742-47-8) 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

PETROLEUM DISTILLATES HFP(64742-48-9.) 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

PARAFFIN AND HYDROCARBON WAXES, OXIDISED, LITHIUM SALTS(68649-48-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

BETA-PINENE(19902-08-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

ALPHA-PINENE(80-56-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

POLYDIMETHYLSILOXANE(63148-62-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

PARAFFIN WAX(8002-74-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (petroleum distillates HFP; polydimethylsiloxane; paraffin and hydrocarbon waxes, oxidised, lithium salts; beta-pinene; distillates, petroleum, light, hydrotreated; paraffin wax)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (polydimethylsiloxane)
Japan - ENCS	N (petroleum distillates HFP; polydimethylsiloxane; paraffin wax)
Korea - KECI	Y
New Zealand - NZIoC	N (paraffin and hydrocarbon waxes, oxidised, lithium salts)
Philippines - PICCS	Y
USA - TSCA	Y
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
petroleum distillates HFP	64742-48-9., 64742-88-7
beta-pinene	19902-08-0, 18172-67-3, 127-91-3
alpha-pinene	80-56-8, 1330-16-1, 2437-95-8, 7785-70-8, 7785-26-4

Continued...

paraffin wax	8002-74-2, 12704-91-5, 105054-93-1, 105845-08-7, 115251-23-5, 115251-24-6, 12704-92-6, 12795-75-4, 160936-34-5, 37220-23-8, 37339-80-3, 39355-22-1, 39373-78-9, 51331-35-2, 54692-42-1, 57572-43-7, 57608-84-1, 58057-11-7, 64742-43-4, 64742-51-4, 68607-08-9, 68649-50-3, 70431-26-4, 72993-88-5, 72993-89-6, 72993-90-9, 8035-62-9, 8044-02-8, 8044-79-9, 9083-41-4, 92045-74-4

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.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net

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