

Meguiar's A1214, Cleaner Wax-Paste

Motor Active

Chemwatch: 4910-73

Version No: 10.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: 25/01/2019

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

Product Identifier

Product name	Meguiar's A1214, Cleaner Wax-Paste
Synonyms	Product Code: 21-131A, A1214
Other means of identification	Not Available

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

Relevant identified uses	Automotive paste wax.
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Details of the supplier of the safety data sheet

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

Emergency telephone number

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

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

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

Poisons Schedule	S5
Classification ^[1]	Flammable Liquid Category 4, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
SIGNAL WORD	WARNING

Hazard statement(s)

H227	Combustible liquid.
H336	May cause drowsiness or dizziness.
H412	Harmful to aquatic life with long lasting effects.
AUH066	Repeated exposure may cause skin dryness and cracking.

Supplementary statement(s)

Continued...

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Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64742-48-9.	30-60	<u>petroleum distillates HFP</u>
63148-62-9	7-13	<u>polydimethylsiloxane</u>
93821-35-3	5-10	<u>calcined flint clay</u>
64742-47-8.	5-10	<u>isoparaffins petroleum hydrotreated HFP</u>
73138-45-1	5-10	<u>montan wax acids, ethylene esters</u>
Not Available	<5	conditioners, trade secret
8015-86-9	1-5	<u>carnauba wax</u>
92704-41-1.	1-5	<u>calcined china clay, low quartz</u>
9082-00-2	0.5-1.5	<u>polyethylene/ polypropylene glycol glyceryl ether</u>
Not Available	10-30	ingredients determined not to be hazardous [Mfr]

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.
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):

Continued...

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	▶ 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 or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
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]

Treat symptomatically.

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

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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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 (CO₂) silicon dioxide (SiO₂) other pyrolysis products typical of burning organic material. May emit poisonous fumes.
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Continued...

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See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▶ 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.

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. ▶ 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	▶ 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	petroleum distillates HFP	Oil mist, refined mineral	5 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	isoparaffins petroleum hydrotreated HFP	White spirits	790 mg/m ³	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
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petroleum distillates HFP	Naphtha, hydrotreated heavy; (Isopar L-rev 2)	350 mg/m3	1,800 mg/m3	40,000 mg/m3
polydimethylsiloxane	Dimethyl siloxane; (Dimethylpolysiloxane; Syltherm XLT; Syltherm 800; Silicone 360)	65 mg/m3	720 mg/m3	4,300 mg/m3
isoparaffins petroleum hydrotreated HFP	Stoddard solvent; (Mineral spirits, 85% nonane and 15% trimethyl benzene)	300 mg/m3	1,800 mg/m3	29500 mg/m3
polyethylene/ polypropylene glycol glyceryl ether	Polyglycol 15-200; (Oxirane, 2-methyl-, polymer with oxirane, ether with 1,2,3-propanetriol (3:1); Calthane NF and ND "B")	30 mg/m3	330 mg/m3	2,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
petroleum distillates HFP	2,500 mg/m3	Not Available
polydimethylsiloxane	Not Available	Not Available
calcined flint clay	Not Available	Not Available
isoparaffins petroleum hydrotreated HFP	20,000 mg/m3	Not Available
montan wax acids, ethylene esters	Not Available	Not Available
carauaba wax	Not Available	Not Available
calcined china clay, low quartz	Not Available	Not Available
polyethylene/ polypropylene glycol glyceryl ether	Not Available	Not Available

MATERIAL DATA

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)

Because the margin of safety of the quartz TLV is not known with certainty and given the associated link between silicosis and lung cancer it is recommended that quartz concentrations be maintained as far below the TLV as prudent practices will allow.

Exposure to respirable crystalline silicas (RCS) represents a significant hazard to workers, particularly those employed in the construction industry where respirable dusts of cement and concrete are common. Cutting, grinding and other high speed processes, involving their finished products, may further result in dusty atmospheres. Bricks are also a potential source of RCSs under such circumstances.

It is estimated that half of the occupations, involved in construction work, are exposed to levels of RCSs, higher than the current allowable limits. Baudry et al: Journal of Occupational and Environmental Hygiene 10: 71-77; 2013

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.

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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
Body protection	See Other protection below
Other protection	▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulphur 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.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Light cream-coloured paste with a sweet hydrocarbon odour; not miscible with water.		
Physical state	Non Slump Paste	Relative density (Water = 1)	0.85
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)	Not Available
Initial boiling point and boiling range (°C)	185	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 = 44.97% (by wt)
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	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
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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. Product is considered stable and hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	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, 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. The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics. Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced. Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics. Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed 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. 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 Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. 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.
Skin Contact	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. The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but mild, 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 (non allergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Open cuts, abraded or irritated skin should not be exposed to this material The material may accentuate any pre-existing dermatitis condition 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 or practical experience suggests, that the material may cause moderate eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged exposure may cause moderate inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. 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.

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Chronic

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Chronic symptoms produced by crystalline silicas included decreased vital lung capacity and chest infections. Lengthy exposure may cause silicosis a disabling form of pneumoconiosis which may lead to fibrosis, a scarring of the lining of the air sacs in the lung.

The form and severity in which silicosis manifests itself depends in part on the type and extent of exposure to silica dusts: chronic, accelerated and acute forms are all recognized. In later stages the critical condition may become disabling and potentially fatal. Restrictive and/or obstructive lung function changes may result from chronic exposure. A risk associated with silicosis is development of pulmonary tuberculosis (silico-tuberculosis). Respiratory insufficiencies due to massive fibrosis and reduced pulmonary function, possibly with accompanying heart failure, are other potential causes of death due to silicosis.

Not all individuals with silicosis will exhibit symptoms (signs) of the disease. However, silicosis can be progressive, and symptoms may potentially appear years after exposures have ceased. Symptoms of silicosis may include (but are not limited to): Shortness of breath; difficulty breathing with or without exertion; coughing; diminished work capacity; diminished chest expansion; reduction of lung volume; heart enlargement and/or failure.

Respirable dust containing newly broken particles has been shown to be more hazardous to animals in laboratory tests than

respirable dust containing older silica particles of similar size. Respirable silica particles which had aged for sixty days or more showed less lung injury in animals than equal exposures of respirable dust containing newly broken pieces of silica. There are reports in the literature indicating that crystalline silica exposure may be associated with adverse health effects involving the kidney, scleroderma (thickening of the skin caused by swelling and thickening of fibrous tissue) and other autoimmune and immunity-related disorders. Several studies of persons with silicosis or silica exposure also indicate or suggest increased risk of developing lung cancer, a risk that may increase with the duration of exposure. Many of these studies of silicosis do not account for lung cancer confounders, especially smoking.

Symptoms may appear 8 to 18 months after initial exposure. Smoking increases this risk. Classic silicosis is a chronic disease characterised by the formation of scattered, rounded or stellate silica-containing nodules of scar tissue in the lungs ranging from microscopic to 1.0 cm or more. The nodules isolate the inhaled silica particles and protect the surrounding normal and functioning tissue from continuing injury. Simple silicosis (in which the nodules are less than 1.0 cm in diameter) is generally asymptomatic but may be slowly progressive even in the absence of continuing exposure. Simple silicosis can develop in complicated silicoses (in which nodules are greater than 1.0 cm in diameter) and can produce disabilities including an associated tuberculous infection (which 50 years ago accounted for 75% of the deaths among silicotic workers). Crystalline silica deposited in the lungs causes epithelial and macrophage injury and activation. Crystalline silica translocates to the interstitium and the regional lymph nodes and cause the recruitment of inflammatory cells in a dose dependent manner. In humans, a large fraction of crystalline silica persists in the lungs. The question of potential carcinogenicity associated with chronic inhalation of crystalline silica remains equivocal with some studies supporting the proposition and others finding no significant association. The results of recent epidemiological studies suggest that lung cancer risk is elevated only in those patients with overt silicosis. A relatively large number of epidemiological studies have been undertaken and in some, increased risk gradients have been observed in relation to dose surrogates - cumulative exposure, duration of exposure, the presence of radiographically defined silicosis, and peak intensity exposure. Chronic inhalation in rats by single or repeated intratracheal instillation produced a significant increase in the incidences of adenocarcinomas and squamous cell carcinomas of the lung. Lifetime inhalation of crystalline silica (87% alpha-quartz) at 1 mg/m³ (74% respirable) by rats, produced an increase in animals with keratinising cystic squamous cell tumours, adenomas, adenocarcinomas, adenosquamous cell carcinomas, squamous cell carcinoma and nodular bronchiolar alveolar hyperplasia accompanied by extensive subpleural and peribronchiolar fibrosis, increased pulmonary collagen content, focal lipoproteinosis and macrophage infiltration. Thoracic and abdominal malignant lymphomas developed in rats after single intrapleural and intraperitoneal injection of suspensions of several types of quartz.

Some studies show excess numbers of cases of scleroderma, connective tissue disorders, lupus, rheumatoid arthritis chronic kidney diseases, and end-stage kidney disease in workers

NOTE: Some jurisdictions require health surveillance be conducted on workers occupationally exposed to silica, crystalline. Such surveillance should emphasise

- demography, occupational and medical history and health advice
- standardised respiratory function tests such as FEV1, FVC and FEV1/FVC
- standardised respiratory function tests such as FV1, FVC and FEV1/FVC
- chest X-ray, full size PA view
- records of personal exposure

Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals.

Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human

Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

Meguiar's A1214, Cleaner Wax-Paste	TOXICITY	IRRITATION
	Not Available	Not Available
petroleum distillates HFP	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Not Available
	Inhalation (rat) LC50: 8.5 mg/l/4h ^[2]	
	Oral (rat) LD50: >4500 mg/kg ^[1]	
polydimethylsiloxane	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye (rabbit): 100 mg/1h - mild
	Oral (rat) LD50: >17000 mg/kg ^[2]	

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calcined flint clay	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[1]	
isoparaffins petroleum hydrotreated HFP	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >5000 mg/kg ^[2]	
montan wax acids, ethylene esters	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): non-irritating *
	Oral (rat) LD50: >2000 mg/kg ^[2]	Skin (rabbit): non-irritating *
carnauba wax	TOXICITY	IRRITATION
	Not Available	Not Available
calcined china clay, low quartz	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[1]	
polyethylene/ polypropylene glycol glyceryl ether	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Not Available
	Oral (rat) LD50: >10000 mg/kg ^[2]	

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 A1214, Cleaner Wax-Paste	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
PETROLEUM DISTILLATES HFP	data for CAS 64742-88-7 i.e. CCINFO record 1441735
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 <i>in vitro</i> or <i>in vivo</i>. Preliminary results indicate that D5 has a potential carcinogenic effect. D4 is considered to impair fertility in rats by inhalation and is classified as a substance toxic to reproduction in category 3 with the risk phrase R62 ('Possible risk of impaired fertility'). The results of a study to screen for oestrogen activity indicate that D4 has very weak oestrogenic and antioestrogenic activity and is a partial agonist (enhances the effect of the estrogen). It is not uncommon for compounds that are weakly oestrogenic to also have antioestrogenic properties. Comparison of the oestrogenic potency of D4 relative to ethinyloestradiol (steroid hormone) indicates that D4 is 585,000 times less potent than ethinyloestradiol in the rat 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. Studies are available for linear siloxanes from an analogue group comprising di- to hexa- siloxanes, as well as key physicochemical properties. The results of the acute toxicity studies for this analogue group are in agreement: there is no evidence from any of the available studies that the substances in this group have any potential for acute toxicity (in terms of either lethality or adverse clinical effects) by any route up to and exceeding the maximum dose levels tested according to current OECD guidelines. It is therefore valid to read-across the lack of acute toxicity between the members of the group where there are data gaps The metabolism of silanes and siloxanes is influenced by the chemistry of silicon, and it is fundamentally different from that of carbon compounds. These differences are due to the fact that silicon is more electropositive than carbon; Si-Si bonds are less stable than C-C bonds and Si-O bonds form very readily, the latter due to their high bond energy. Functional groups such as -OH, -CO₂H, and -CH₂OH are commonly seen in organic drug metabolites. If such functionalities are formed from siloxane metabolism, they will undergo rearrangement with migration of the Si atom from carbon to oxygen. Consequently, alpha hydroxysilanes may isomerise to silanols and this provides a mechanism by which very polar metabolites may be formed from highly

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	hydrophobic alkylsiloxanes in relatively few metabolic steps The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. No toxic response noted during 90 day subchronic inhalation toxicity studies The no observable effect level is 450 mg/m3. Non-irritating and non-sensitising in human patch test. [Xerox]*
MONTAN WAX ACIDS, ETHYLENE ESTERS	Group A aliphatic monoesters (fatty acid esters) According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group A substances are simple monoesters derived from a monofunctional alcohol, such as 2-ethylhexyl alcohol (C8-alcohol) or tridecyl alcohol (C13 alcohol) and fatty acids such as palmitic, stearic, oleic or linoleic acid. Metabolism of the parent esters is expected to yield the corresponding fatty acids and alcohols. The fatty acids are naturally occurring and have a low order of toxicity. Group A substances are rather lipophilic (log Kow 10-15) in character due to the large number of carbon numbers in the ester molecule (e.g., 24,26, 31 carbons) and have relatively high boiling points. Owing to the non-volatile nature of these esters, their vapour pressures are very low and difficult to determine experimentally. Water solubility is also very low. Mammalian Toxicity: Acute Toxicity: Many higher fatty acid esters, such as the stearates, oleates and palmitates, have been cleared for use in the food industry ; thus, their general physiological response and toxicity are very low. Many of the higher fatty acid esters are considered safe for use in cosmetics. Available acute toxicity data indicate that the fatty acid esters in Group A, in general, have a low order of toxicity [e.g., palmitic acid, 2-ethylhexyl ester (LD50 > 5 g/kg) and tall oil fatty acid 2-ethylhexyl ester (LD50 > 64 g/kg)]. Consistent with that, available data spanning the carbon range of C22 to C34 indicate that the alkyl fatty acid esters are not toxic by oral administration [rat LD50 (oral) > 5g/kg, with range from 5 g/kg to 64 kg/kg]. Butyl stearate is tolerated by rats without lethal effects at oral doses of 32 g/kg while octyl oleate has a reported LD50 of >40 ml/kg. In addition, many alkyl fatty acid esters, such as the stearates, oleates and palmitates, have been demonstrated to be not toxic by dermal administration Because of the low volatility of these substances, inhalation exposure at toxicological significant levels is not expected. Repeated Dose Toxicity: 28-Day oral gavage studies in rats with decyl oleate (CAS 3687-46-5) at doses of 100,500 and 1000 mg/kg showed no toxicity as noted with respect to clinical symptoms, biochemistry, hematology, gross lesions or tissue/organ histopathology. The NOAEL was estimated to be 1000 mg/kg. Similarly, octyl or (2-ethylhexyl) stearate showed a NOAEL of 1000 mg/kg in 28-day oral gavage studies in rats. In chronic two-year feeding studies with butyl stearate at concentrations of 1.25% or 6.25% in the diet, exposed rats showed no significant difference from control animals with respect to growth, survival, blood counts or other haematological parameters. Besides the two substances above, various other long-chain fatty acid esters have also been studied for their repeated dose toxicity and the findings support a low order of toxicity. Genotoxicity: Genetic Toxicity (Salmonella). Fatty acid, C 16- 18 saturated and C 18 unsaturated, 2-ethylhexanoate (CAS 85049-37-2); octyl stearate (CAS 109-36-4); and decyl oleate (CAS 3687-46-5)] were shown to be negative in the Ames assay. Since the monoesters are similar in chemical structure and carbon-number range, it is unlikely that esters in Group A will induce point mutation. In addition, the chemistry of the long-chain fatty acids does not suggest the likelihood that these substances or their constituent substructures (i.e., fatty acids, alcohols) are reactive or electrophilic in nature. Genetic Toxicity (Chromosomal Aberrations). The chemistry of the long-chain fatty acid esters does not suggest the likelihood that these substances or their constituent substructures (i.e., fatty acids, alcohols) are reactive or electrophilic in nature. Therefore, the likelihood that the fatty acid monoesters may cause chromosomal mutation is very low. Reproductive toxicity: Assessment of reproductive effects of alkyl fatty acid esters in Group A is based primarily on studies with butyl stearate. Fertility, litter size and survival of offspring were normal in rats fed diets containing 6.25% butyl stearate for 10 weeks. However, growth was reduced in offspring during the pre-weaning and post-weaning periods. No gross lesions were noted among the offspring killed at the end of the 21-day post-weaning periods. These results indicate that long-chain fatty acid esters do not cause reproductive toxicity in rats. Given the relative low order of toxicity for long-chain fatty acid esters and their relative non-electrophilic and non-reactive nature, it seems unlikely that the long-chain fatty acid esters would present serious reproductive concerns. Developmental Toxicity/ Teratogenicity. Assessment of developmental effects for the long-chain fatty acid esters in this group was based primarily on data reported for fatty acid, C16-18, 2-ethylhexyl ester (CAS 91031-48-0). In oral gavage studies in rats administered doses of 100,300 and 1000 mg/kg during gestation, the maternal NOAEL was > 1000 mg/kg and the NOAEL for teratogenicity was >1000 mg/kg. Based on these findings and the fact Group A substances, are very chemically similar to the structure of the tested material, read-across assessment is thought to be appropriate.
Meguiar's A1214, Cleaner Wax-Paste & CALCINED FLINT CLAY & ISOPARAFFINS PETROLEUM HYDROTREATED HFP & CARNAUBA WAX & CALCINED CHINA CLAY, LOW QUARTZ & POLYETHYLENE/ POLYPROPYLENE GLYCOL GLYCERYL ETHER	No significant acute toxicological data identified in literature search.
Meguiar's A1214, Cleaner Wax-Paste & PETROLEUM DISTILLATES HFP & ISOPARAFFINS PETROLEUM HYDROTREATED HFP	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins. 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 A1214, Cleaner Wax-Paste & PETROLEUM DISTILLATES HFP	for petroleum: Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline This product may contain 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.

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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.

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 either not available or does not fill the criteria for classification
 ✓ – Data available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Meguiar's A1214, Cleaner Wax-Paste	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
petroleum distillates HFP	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	4.1mg/L	2
	EC50	48	Crustacea	4.5mg/L	2
	EC50	72	Algae or other aquatic plants	>1-mg/L	2
	LC50	96	Fish	18mg/L	2
	EC50	48	Crustacea	1.4mg/L	2
	EC50	72	Algae or other aquatic plants	3.7mg/L	2
polydimethylsiloxane	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	3.16mg/L	4
calcined flint clay	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.57mg/L	2
	EC50	48	Crustacea	1.5mg/L	2
	EC50	96	Algae or other aquatic plants	0.024mg/L	2
	NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	2-500mg/L	2
	EC10	72	Algae or other aquatic plants	33mg/L	2
	NOEC	504	Crustacea	1-mg/L	2
isoparaffins petroleum hydrotreated HFP	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>1-mg/L	2
	EC50	48	Crustacea	>1-mg/L	2
	EC50	72	Algae or other aquatic plants	>1-mg/L	2
	NOEC	3072	Fish	=1mg/L	1
	LC50	96	Fish	4.1mg/L	2
	EC50	48	Crustacea	4.5mg/L	2
	EC50	72	Algae or other aquatic plants	>1-mg/L	2
	LC50	96	Fish	0.14mg/L	2
	EC50	96	Algae or other aquatic plants	0.277mg/L	2
	NOEC	720	Crustacea	0.024mg/L	2
montan wax acids, ethylene esters	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	=50mg/L	1
	EC50	48	Crustacea	440mg/L	2
	EC50	72	Algae or other aquatic plants	>100mg/L	2
	EC10	72	Algae or other aquatic plants	>100mg/L	2
	NOEC	72	Algae or other aquatic plants	>=100mg/L	2

Continued...

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carnauba wax	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
calcined china clay, low quartz	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	2-500mg/L	2
	EC10	72	Algae or other aquatic plants	33mg/L	2
	NOEC	504	Crustacea	1-mg/L	2
polyethylene/ polypropylene glycol glyceryl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

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

Harmful to aquatic organisms.

May cause long-term adverse effects in the aquatic environment.

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

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

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 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 octamethylcyclotrisiloxane:

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

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

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

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

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

For Group A aliphatic esters (fatty acid esters):

Environmental fate:

Group A substances are rather lipophilic (log Kow 10-15) in character due to the large number of carbons in the ester molecule (e.g., 24,26, 31 carbons) and have relatively high boiling points. Owing to the non-volatile nature of these esters, their vapor pressures are very low and difficult to determine experimentally. Water solubility is also very low.

Hydrolysis half lives and atmospheric photodegradation rates were calculated by EPIWIN. The monoester hydrolysis rates were determined to be quite low and not a significant environmental fate route. Fugacity modeling indicates that the fatty acid esters have similar distribution patterns in the environmental compartments (e.g., air, water, soil, sediment).

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Biodegradation of alkyl fatty acid esters are expected to occur extensively based on the reported 28 day test results (80-85% biodegradation, OECD 301D) for decyl oleate and for the 2-ethylhexyl ester of C16- 18 saturated and C18 unsaturated fatty acids (CAS 85049-37-2). Group A Substances are expected to be extensively biodegraded since the fatty acids in these esters are primarily comprised of palmitic, stearic or oleic acids, which are known to be rapidly biodegraded

Ecotoxicity:

Aquatic toxicity results have been reported for decyl oleate and fatty acid, C16- 18 saturated and C18 unsaturated, 2-ethylhexanoate. They are not acutely toxic to fish (LC50 3200 mg/L). In daphnids, the acute LC50 was reported to be 17 mg/L and in algae, the LC50 was reported to be 40-42 mg/L based on biomass and growth rate endpoints. Because of their limited water solubility, the alkyl fatty acid esters and Group A esters are not likely to cause acute aquatic toxicity.

For hydrocarbons:

Environmental fate:

The lower molecular weight hydrocarbons are expected to form a "slick" on the surface of waters after release in calm sea conditions. This is expected to evaporate and enter the atmosphere where it will be degraded through reaction with hydroxy radicals.

Some hydrocarbon will become associated with benthic sediments, and it is likely to be spread over a fairly wide area of sea floor. Marine sediments may be either aerobic or anaerobic. The material, in probability, is biodegradable, under aerobic conditions (isomerised olefins and alkenes show variable results). Evidence also suggests that the hydrocarbons may be degradable under anaerobic conditions although such degradation in benthic sediments may be a relatively slow process.

Under aerobic conditions hydrocarbons degrade to water and carbon dioxide, while under anaerobic processes they produce water, methane and carbon dioxide.

Alkenes have low log octanol/water partition coefficients (Kow) of about 1 and estimated bioconcentration factors (BCF) of about 10; aromatics have intermediate values (log Kow values of 2-3 and BCF values of 20-200), while C5 and greater alkanes have fairly high values (log Kow values of about 3-4.5 and BCF values of 100-1,500).

The estimated volatilisation half-lives for alkanes and benzene, toluene, ethylbenzene, xylene (BTEX) components were predicted as 7 days in ponds, 1.5 days in rivers, and 6 days in lakes. The volatilisation rate of naphthalene and its substituted derivatives were estimated to be slower.

Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds. Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media.

The final products of microbial degradation are carbon dioxide, water, and microbial biomass. The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics, and the aromatics in the C10-C22 range are the most readily biodegradable; n-alkanes, n-alkyl aromatics, and aromatics in the C5-C9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilisation and thus are unavailable in most environments; n-alkanes in the C1-C4 ranges are biodegradable only by a narrow range of specialised hydrocarbon degraders; and n-alkanes, n-alkyl aromatics, and aromatics above C22 are generally not available to degrading microorganisms. Hydrocarbons with condensed ring structures, such as PAHs with four or more rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is, greater than 7.

All biological transformations are affected by temperature. Generally, as the temperature increases, biological activity tends to increase up to a temperature where enzyme denaturation occurs.

Atmospheric fate: Alkanes, isoalkanes, and cycloalkanes have half-lives on the order of 1-10 days, whereas alkenes, cycloalkenes, and substituted benzenes have half-lives of 1 day or less.

Photochemical oxidation products include aldehydes, hydroxy compounds, nitro compounds, and peroxyacyl nitrates. Alkenes, certain substituted aromatics, and naphthalene are potentially susceptible to direct photolysis.

Ecotoxicity:

Hydrocarbons are hydrophobic (high log Kow and low water solubility). Such substances produce toxicity in aquatic organisms by a mechanism referred to as "non-polar narcosis" or "baseline" toxicity. The hydrophobicity increases and water solubility decreases with increasing carbon number for a particular class of hydrocarbon. Substances with the same carbon number show increased hydrophobicity and decreased solubility with increasing saturation. Quantitative structure activity relationships (QSAR), relating both solubility and toxicity to Kow predict that the water solubility of single chemical substances decreases more rapidly with increasing Kow than does the acute toxicity.

Based on test results, as well as theoretical considerations, the potential for bioaccumulation may be high. Toxic effects are often observed in species such as blue mussel, daphnia, freshwater green algae, marine copepods and amphipods.

The values of log Kow for individual hydrocarbons increase with increasing carbon number within homologous series of generic types. Quantitative structure activity relationships (QSAR), relating log Kow values of single hydrocarbons to toxicity, show that water solubility decreases more rapidly with increasing Kow than does the concentration causing effects. This relationship varies somewhat with species of hydrocarbon, but it follows that there is a log Kow limit for hydrocarbons, above which, they will not exhibit acute toxicity; this limit is at a log Kow value of about 4 to 5. It has been confirmed experimentally that for fish and invertebrates, paraffinic hydrocarbons with a carbon number of 10 or higher (log Kow >5) show no acute toxicity and that alkylbenzenes with a carbon number of 14 or greater (log Kow >5) similarly show no acute toxicity.

QSAR equations for chronic toxicity also suggest that there should be a point where hydrocarbons with high log Kow values become so insoluble in water that they will not cause chronic toxicity, that is, that there is also a solubility cut-off for chronic toxicity. Thus, paraffinic hydrocarbons with carbon numbers of greater than 14 (log Kow >7.3) should show no measurable chronic toxicity. Experimental support for this cut-off is demonstrated by chronic toxicity studies on lubricant base oils and one "heavy" solvent grade (substances composed of paraffins of C20 and greater) which show no effects after exposures to concentrations well above solubility.

The initial criteria for classification of substances as dangerous to the aquatic environment are based upon acute toxicity data in fish, daphnids and algae. However, for substances that have low solubility and show no acute toxicity, the possibility of a long-term or chronic hazard to the environment is recognised in the R53 phrase or so-called "safety net". The R53 assignment for possible long-term harm is a surrogate for chronic toxicity test results and is triggered by substances that are both bioaccumulative and persistent. The indicators of bioaccumulation and persistence are taken as a BCF > 100 (or log Kow > 3 if no BCF data) and lack of ready biodegradability. For low solubility substances which have direct chronic toxicity data demonstrating no chronic toxicity at 1 mg/L or higher, these data take precedence such that no classification for long term toxicity is required.

Drinking Water Standards: hydrocarbon total: 10 ug/l (UK max.).

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
isoparaffins petroleum hydrotreated HFP	LOW (BCF = 159)

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 DISPOSAL CONSIDERATIONS**Waste treatment methods**

Product / Packaging disposal	▶ 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.
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Continued...

▶ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO Not Applicable
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

PETROLEUM DISTILLATES HFP(64742-48-9.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO
Australia Inventory of Chemical Substances (AICS)	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)	International FOSFA List of Banned Immediate Previous Cargoes

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

Australia Inventory of Chemical Substances (AICS)	IMO IBC Code Chapter 17: Summary of minimum requirements
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)	IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C	IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4	

CALCINED FLINT CLAY(93821-35-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

ISOPARAFFINS PETROLEUM HYDROTREATED HFP(64742-47-8.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List	IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes	IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO
Australia Exposure Standards	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International FOSFA List of Banned Immediate Previous Cargoes
Australia Inventory of Chemical Substances (AICS)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Chinese)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)
IMO IBC Code Chapter 17: Summary of minimum requirements	

MONTAN WAX ACIDS, ETHYLENE ESTERS(73138-45-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

CARNAUBA WAX(8015-86-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)	

CALCINED CHINA CLAY, LOW QUARTZ(92704-41-1.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

POLYETHYLENE/ POLYPROPYLENE GLYCOL GLYCERYL ETHER(9082-00-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)	IMO IBC Code Chapter 17: Summary of minimum requirements
GESAMP/EHS Composite List - GESAMP Hazard Profiles	

National Inventory Status

National Inventory	Status
Australia - AICS	No (calcined china clay, low quartz; conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
Canada - DSL	No (conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients

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Canada - NDSL	No (petroleum distillates HFP; polydimethylsiloxane; polyethylene/ polypropylene glycol glyceryl ether; montan wax acids, ethylene esters; calcined china clay, low quartz; carnauba wax; isoparaffins petroleum hydrotreated HFP; calcined flint clay; conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
China - IECSC	No (conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
Europe - EINEC / ELINCS / NLP	No (polydimethylsiloxane; polyethylene/ polypropylene glycol glyceryl ether; conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
Japan - ENCS	No (petroleum distillates HFP; polydimethylsiloxane; polyethylene/ polypropylene glycol glyceryl ether; montan wax acids, ethylene esters; calcined china clay, low quartz; carnauba wax; calcined flint clay; conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
Korea - KECI	No (conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
New Zealand - NZIoC	No (conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
Philippines - PICCS	No (conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
USA - TSCA	No (conditioners, trade secret; ingredients determined not to be hazardous [Mfr]) Non-disclosed ingredients
Legend:	Yes = All ingredients are on the inventory No = 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

Revision Date	25/01/2019
Initial Date	18/11/2006

SDS Version Summary

Version	Issue Date	Sections Updated
9.1.1.1	23/06/2014	Acute Health (skin), Acute Health (swallowed), Chronic Health, Classification, Engineering Control, Environmental, Exposure Standard, Fire Fighter (fire/explosion hazard), First Aid (skin), Personal Protection (Respirator), Storage (storage incompatibility), Toxicity and Irritation (Other), Transport Information
10.1.1.1	25/01/2019	One-off system update. NOTE: This may or may not change the GHS classification

Other information

Ingredients with multiple cas numbers

Name	CAS No
petroleum distillates HFP	64742-48-9, 64742-88-7
calcined flint clay	93821-35-3, 66402-68-4, 39429-16-8, 85995-95-5, 90669-61-7, 90669-64-0, 90669-65-1, 90669-66-2, 90669-67-3, 90669-68-4, 90669-69-5, 90669-70-8, 90669-71-9, 90669-72-0, 93763-83-8, 94114-16-6, 94114-17-7, 94551-76-5, 94552-04-2, 92704-41-1
isoparaffins petroleum hydrotreated HFP	64742-47-8, 64742-82-1, 8052-41-3, 1030262-12-4, 101795-05-5.
montan wax acids, ethylene esters	73138-45-1, 73138-46-2
carnauba wax	8015-86-9, 85712-28-3

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC — TWA: Permissible Concentration-Time Weighted Average
 PC — STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit,
 IDLH: Immediately Dangerous to Life or Health Concentrations
 OSF: Odour Safety Factor
 NOAEL :No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index

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