#### **Motor Active**

Chernwatch: 4829-75 Version No: 8.1.1.1 Safety Data Sheet according to WHS and ADG requirements

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

#### **Product Identifier**

Product name	eguiar's G138 - NXT Hot Shine Tire Coating Aerosol	
Synonyms	Not Available	
Proper shipping name	3	
Other means of identification	Not Available	

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

Relevant identified uses	The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. Application is by spray atomisation from a hand held aerosol pack Use according to manufacturer's directions. Automotive. Tire coating / maintenance product.
--------------------------	--

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

Registered company name	Motor Active	
Address	Slough Business Park, Holker Street Silverwater NSW 2128 Australia	
Telephone	737 9422 1800 350 622	
Fax	1 2 9737 9414	
Website	www.motoractive.com.au	
Email	andrew.spira@motoractive.com.au	

### Emergency telephone number

	•		
Association / Organisation	MotorActive		
Emergency telephone numbers	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. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

#### CHEMWATCH HAZARD RATINGS

Min Max
Flammability 3
Toxicity 2
Body Contact 2
Reactivity 1
Chronic 2

Poisons Schedule	Not Applicable	
Classification [1]       Aerosols Category 1, Skin Corrosion/Irritation Category 2, Reproductive Toxicity Category 2, Specific target organ toxicity - single exposure Category 2, Inarcotic effects), Specific target organ toxicity - repeated exposure Category 2, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category		

# Label elements

GHS label elements	
 SIGNAL WORD	DANGER

Issue Date: 01/09/2015

Print Date: 22/08/2016

L.GHS.AUS.EN

Continued...

H222	Extremely flammable aerosol.		
H315	Causes skin irritation.		
H361	Suspected of damaging fertility or the unborn child.		
H336	May cause drowsiness or dizziness.		
H373	ay cause damage to organs.		
H411	c to aquatic life with long lasting effects.		
AUH044	Risk of explosion if heated under confinement		
AUH066	Repeated exposure may cause skin dryness and cracking		
Precautionary statement(s	) Prevention		
P201	Obtain special instructions before use.		
P210	Keep away from heat/sparks/open flames/hot surfaces No smoking.		
P211	Do not spray on an open flame or other ignition source.		
D251	Processized container: Do not pierce or hum, over offer upo		

P251	Pressurized container: Do not pierce or burn, even after use.	
P260	not breathe dust/fume/gas/mist/vapours/spray.	
P271	nly outdoors or in a well-ventilated area.	
P281	e personal protective equipment as required.	
P273	Avoid release to the environment.	
P280	Wear protective gloves/protective clothing/eye protection/face protection.	

### Precautionary statement(s) Response

P308+P313	E exposed or concerned: Get medical advice/attention.	
P362	off contaminated clothing and wash before reuse.	
P312	SON CENTER or doctor/physician if you feel unwell.	
P391	ct spillage.	
P302+P352	ON SKIN: Wash with plenty of soap and water.	
P304+P340	INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.	
P332+P313	f skin irritation occurs: Get medical advice/attention.	

### Precautionary statement(s) Storage

P405	Store locked up.	
P410+P412	tect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.	
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
79-20-9	30-60	methyl acetate
67-64-1	10-30	acetone
64742-49-0.	10-30	naphtha petroleum, light, hydrotreated
63148-62-9	7-13	polydimethylsiloxane
142-82-5	3-7	heptane
64742-48-9.	1-3	naphtha petroleum, heavy, hydrotreated
124-38-9	5-10	carbon dioxide

### SECTION 4 FIRST AID MEASURES

### Description of first aid measures

Eye Contact	<ul> <li>If aerosols come in contact with the eyes:</li> <li>Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water.</li> <li>Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.</li> <li>Transport to hospital or doctor without delay.</li> <li>Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
Skin Contact	<ul> <li>If solids or aerosol mists are deposited upon the skin:</li> <li>Flush skin and hair with running water (and soap if available).</li> <li>Remove any adhering solids with industrial skin cleansing cream.</li> </ul>

	<ul> <li>DO NOT use solvents.</li> <li>Seek medical attention in the event of irritation.</li> </ul>
Inhalation	<ul> <li>If aerosols, fumes or combustion products are inhaled:</li> <li>Remove to fresh air.</li> <li>Lay patient down. Keep warm and rested.</li> <li>Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>Transport to hospital, or doctor.</li> </ul>
Ingestion	<ul> <li>Avoid giving milk or oils.</li> <li>Avoid giving alcohol.</li> <li>Not considered a normal route of entry.</li> </ul>

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

#### Treat symptomatically.

for simple ketones:

#### BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for pulmonary oedema.
- Monitor and treat, where necessary, for shock
- > DO NOT use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5mL/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

#### Give activated charcoal.

-----

#### ADVANCED TREATMENT

- + Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- · Consider intubation at first sign of upper airway obstruction resulting from oedema
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT \_\_\_\_\_

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph. Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.

 Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE. P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

Following acute or short term repeated exposures to n-hexane

- + Large quantities of n-hexane are expired by the lungs after vapour exposure (50-60%). Humans exposed to 100 ppm demonstrate an n-hexane biological half life of 2 hours.
- Initial attention should be directed towards evaluation and support of respiration. Cardiac dysrhythmias are a potential complication.

#### INGESTION:

P lpecac syrup should be considered for ingestion of pure hexane exceeding 2-3ml/kg. Extreme caution must be taken to avoid aspiration since small amounts of n-hexane intratracheally, produce a severe chemical pneumonitis

#### [Ellenhorn and Barceloux: Medical Toxicology] **BIOLOGICAL EXPOSURE INDEX - BEI**

BEIs represent the levels of determinants which are most likely to be observed in specimens collected in a healthy worker who has been exposed to chemicals to the same extent as a worker with

inhalation exposure to the Exposure Standard (ES or TLV).			
Determinant	Index	Sampling Time	Comments
1. 2,5-hexanedione in urine	5 mg/gm creatinine	End of shift	NS
2. n-Hexane in end-exhaled air			SQ

2. n-Hexane in end-exhaled air

NS: Non-specific determinant; Metabolite observed following exposure to other materials.

SQ: Semi-quantitative determinant; Interpretation may be ambiguous - should be used as a screening test or confirmatory test. Following acute or short term repeated exposures to toluene:

- Toluene is absorbed across the alveolar barrier, the blood/air mixture being 11.2/15.6 (at 37 degrees C.) The concentration of toluene, in expired breath, is of the order of 18 ppm following sustained exposure to 100 ppm. The tissue/blood proportion is 1/3 except in adipose where the proportion is 8/10.
- Metabolism by microsomal mono-oxygenation, results in the production of hippuric acid. This may be detected in the urine in amounts between 0.5 and 2.5 g/24 hr which represents, on average 0.8 gm/gm of creatinine. The biological half-life of hippuric acid is in the order of 1-2 hours.
- Primary threat to life from ingestion and/or inhalation is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (eg cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 <50 mm Hg or pCO2 > 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial damage 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 (adrenaline) 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

#### BIOLOGICAL EXPOSURE INDEX - BEL

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

Determinant	Index	Sampling Time	Comments
o-Cresol in urine	0.5 mg/L	End of shift	В
Hippuric acid in urine	1.6 g/g creatinine	End of shift	B, NS
Toluene in blood	0.05 mg/L	Prior to last shift of workweek	

NS: Non-specific determinant: also observed after exposure to other material

B: Background levels occur in specimens collected from subjects NOT exposed

### SECTION 5 FIREFIGHTING MEASURES

### Extinguishing media

SMALL FIRE:

Water spray, dry chemical or CO2
 LARGE FIRE:

Water spray or fog.

### Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result		
Advice for firefighters			
Fire Fighting	<ul> <li>Alert Fire Brigade and tell them location and nature of hazard.</li> <li>May be violently or explosively reactive.</li> <li>Wear breathing apparatus plus protective gloves.</li> <li>Prevent, by any means available, spillage from entering drains or water course.</li> <li>If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>DO NOT approach containers suspected to be hot.</li> <li>Cool fire exposed containers surple of the name of the provided location.</li> <li>If safe to do so, remove containers from path of fire.</li> <li>Equipment should be thoroughly decontaminated after use.</li> </ul>		
Fire/Explosion Hazard	<ul> <li>Liquid and vapour are highly flammable.</li> <li>Severe fire hazard when exposed to heat or flame.</li> <li>Vapour forms an explosive mixture with air.</li> <li>Severe explosion hazard, in the form of vapour, when exposed to flame or spark.</li> <li>Vapour may travel a considerable distance to source of ignition.</li> <li>Heating may cause expansion or decomposition with violent container rupture.</li> <li>Aerosol cans may explode on exposure to naked flames.</li> <li>Rupturing containers may rocket and scatter burning materials.</li> <li>Hazards may not be restricted to pressure effects.</li> <li>May emit acid, poisonous or corrosive fumes.</li> <li>On combustion, may emit toxic fumes of carbon monoxide (CO).</li> <li>Combustion products include:carbon dioxide (CO2) silicon dioxide (SiO2) other pyrolysis products typical of burning organic materialContains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.</li> </ul>		

#### SECTION 6 ACCIDENTAL RELEASE MEASURES

#### Personal precautions, protective equipment and emergency procedures

See section 8

### **Environmental precautions**

See section 12

### Methods and material for containment and cleaning up

Minor Spills	<ul> <li>Clean up all spills immediately.</li> <li>Avoid breathing vapours and contact with skin and eyes.</li> <li>Wear protective clothing, impervious gloves and safety glasses.</li> <li>Shut off all possible sources of ignition and increase ventilation.</li> <li>Wipe up.</li> <li>If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.</li> <li>Undamaged cans should be gathered and stowed safely.</li> </ul>
Major Spills	<ul> <li>Remove leaking cylinders to a safe place if possible.</li> <li>Release pressure under safe, controlled conditions by opening the valve.</li> <li>DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve.</li> <li>Clear area of personnel and move upwind.</li> <li>Alert Fire Brigade and tell them location and nature of hazard.</li> <li>May be violently or explosively reactive.</li> <li>Wear breathing apparatus plus protective gloves.</li> <li>Prevent, by any means available, spillage from entering drains or water courses</li> <li>No smoking, naked lights or ignition sources.</li> <li>Increase ventilation.</li> <li>Stop leak if safe to do so.</li> <li>Water spray or fog may be used to disperse / absorb vapour.</li> <li>Absorb or cover spill with sand, earth, inert materials or vermiculite.</li> <li>If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.</li> <li>Undamaged cans should be gathered and stowed safely.</li> <li>Collect residues and seal in labelled drums for disposal.</li> </ul>

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

#### SECTION 7 HANDLING AND STORAGE

<ul> <li>Containers, even those that have been emptied, may contain explosive vapours.</li> </ul>
<ul> <li>Do NOT cut, drill, grind, weld or perform similar operations on or near containers.</li> </ul>
<ul> <li>Natural gases contain a contaminant, radon-222, a naturally occurring radioactive gas. During subsequent processing, radon tends to concentrate in liquefied petroleum streams and in product streams having similar boiling points. Industry experience indicates that the commercial product may contain small amounts of radon-222 and its radioactive decay products (radon daughters). The actual concentration of radon-222 and radioactive daughters in process equipment (IE lines, filters, pumps and reactor units) may reach significant levels and produce potentially damaging levels of gamma radiation. A potential external radiation hazard exists at or near any pipe, valve or vessel containing a radon enriched stream or containing internal deposits of radioactive material. Field studies, however, have not shown that conditions exist that expose the worker to cumulative exposures in excess of general population limits. Equipment containing gamma-emitting decay products should be presumed to be internally contaminated with alpha-emitting decay products which may be hazardous if inhaled or ingested. During maintenance operations that require the opening of contaminated process equipment, the flow of gas should be stopped and a four hour delay enforced to allow gamma-radiation to drop to backforround levels. Protective equipment (including high efficiency particulate respirators (P3) suitable for radionucleotides or supplied air) should be worn by personnel entering a vessel or working on contaminated process equipment to prevent skin contamination or inhalation of any residue containing alpha-radiation. Airborne contamination may be minimised by handling scale and/or contaminated materials in a wet state. (<i>TEXACO</i>)</li> <li>Avoid all personal contact, including inhalation.</li> <li>Wear protective clothing when risk of exposure occurs.</li> <li>Use in a well-ventilated area.</li> <li>Prevent concentration in hollows and sumps.</li> <li>Do NOT enter confined spaces until atmosphere has been checked.</li> <li>Avoid s</li></ul>
<ul> <li>Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
<ul> <li>Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can</li> <li>Store in original containers in approved flammable liquid storage area.</li> <li>DO NOT store in pits, depressions, basements or areas where vapours may be trapped.</li> <li>No smoking, naked lights, heat or ignition sources.</li> <li>Keep containers securely sealed. Contents under pressure.</li> <li>Store in a cool, dry, well ventilated area.</li> <li>Avoid storage at temperatures higher than 40 deg C.</li> <li>Store in an upright position.</li> <li>Protect containers against physical damage.</li> </ul>

### Conditions for safe storage, including any incompatibilities

Suitable container	<ul> <li>Aerosol dispenser.</li> <li>Check that containers are clearly labelled.</li> </ul>
Storage incompatibility	<ul> <li>Avoid reaction with oxidising agents</li> </ul>

### SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

### **Control parameters**

### OCCUPATIONAL EXPOSURE LIMITS (OEL)

### INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	methyl acetate	Methyl acetate	606 mg/m3 / 200 ppm	757 mg/m3 / 250 ppm	Not Available	Not Available
Australia Exposure Standards	acetone	Acetone	1185 mg/m3 / 500 ppm	2375 mg/m3 / 1000 ppm	Not Available	Not Available
Australia Exposure Standards	heptane	Heptane (n-Heptane)	1640 mg/m3 / 400 ppm	2050 mg/m3 / 500 ppm	Not Available	Not Available
Australia Exposure Standards	naphtha petroleum, heavy, hydrotreated	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	carbon dioxide	Carbon dioxide / Carbon dioxide in coal mines	9000 mg/m3 / 22500 mg/m3 / 5000 ppm / 12500 ppm	54000 mg/m3 / 30000 ppm	Not Available	Not Available

### EMERGENCY LIMITS

Ingredient	Material name		TEEL-1	TEEL-2	TEEL-3
methyl acetate	Methyl acetate	2	250 ppm	250 ppm	10000 ppm
acetone	Acetone	Ν	Not Available	Not Available	Not Available
polydimethylsiloxane	Dimethyl siloxane; (Dimethylpolysiloxane; Syltherm XLT; Syltherm 800; Silicone	360) 1	1.5 mg/m3	16 mg/m3	990 mg/m3
heptane	Heptane	4	140 ppm	440 ppm	5000 ppm
naphtha petroleum, heavy, hydrotreated	Naphtha, hydrotreated heavy; (Isopar L-rev 2)	1	171 ppm	171 ppm	570 ppm
carbon dioxide	Carbon dioxide	3	30,000 ppm	30000 ppm	50000 ppm
Ingredient	Original IDLH	Revised IDLH			

methyl acetate	10,000 ppm	3,100 [LEL] ppm
acetone	20,000 ppm	2,500 [LEL] ppm
naphtha petroleum, light, hydrotreated	Not Available	Not Available
polydimethylsiloxane	Not Available	Not Available
heptane	5,000 ppm	750 ppm
naphtha petroleum, heavy, hydrotreated	Not Available	Not Available
carbon dioxide	50,000 ppm	40,000 ppm

#### MATERIAL DATA

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)

Saturation vapour concentration: 237000 ppm @ 20 C

NOTE: Detector tubes measuring in excess of 40 ppm, are available.

Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.

Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.

A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.

Odour Safety Factor(OSF)

OSF=38 (ACETONE)

#### For butane:

Odour Threshold Value: 2591 ppm (recognition)

Butane in common with other homologues in the straight chain saturated aliphatic hydrocarbon series is not characterised by its toxicity but by its narcosis-inducing effects at high concentrations. The TLV is based on analogy with pentane by comparing their lower explosive limits in air. It is concluded that this limit will protect workers against the significant risk of drowsiness and other narcotic effects.

Odour Safety Factor(OSF) OSF=0.22 (n-BUTANE)

For n-hexane:

#### Odour Threshold Value: 65 ppm

NOTE: Detector tubes for n-hexane, measuring in excess of 100 ppm, are available commercially.

Occupational polyneuropathy may result from exposures as low as 500 ppm (as hexane), whilst nearly continuous exposures of 250 ppm have caused neurotoxic effects in animals. Many literature reports have failed to distinguish hexane from n-hexane and on the assumption that the commercial hexane contains 30% n-hexane, a worst case recommendation for TLV is assumed to reduce the risk of peripheral neuropathies (due to the metabolites 2,5-heptanedione and 3,6-octanedione) and other adverse neuropathic effects.

Concurrent exposure to chemicals (including MEK) and drugs which induce hepatic liver oxidative metabolism can reduce the time for neuropathy to appear.

Odour Safety Factor(OSF) OSF=0.15 (n-HEXANE)

For propane Odour Safety Factor(OSF) OSF=0.16 (PROPANE)

Exposed individuals are NOT reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows

Class OSF Description

- A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
- B 26-550 As "A" for 50-90% of persons being distracted
- C 1-26 As "A" for less than 50% of persons being distracted
- D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
- E <0.18 As "D" for less than 10% of persons aware of being tested
- Odour Safety Factor(OSF) OSF=0.042 (solvent naphtha, medium aliphatic)

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. General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate

	protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, det required to effectively remove the contaminant.	ermine the "capture veloc	cities" of fresh circulating air	
	Type of Contaminant:		Speed:	
	aerosols, (released at low velocity into zone of active generation)		0.5-1 m/s	
	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid	air motion)	1-2.5 m/s (200-500 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 o	ir currents	
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high	n toxicity	
	3: Intermittent, low production.	3: High production, hear	vy use	
	4: Large hood or large air mass in motion	4: Small hood-local con	ntrol only	
	Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple ext of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point distance from the contaminating source. The air velocity at the extraction fan, for example, should be solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerat apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when	t should be adjusted, acco a minimum of 1-2 m/s (20 ions, producing performar	ordingly, after reference to 00-400 f/min.) for extraction of nce deficits within the extraction	
Personal protection				
Eye and face protection	No special equipment for minor exposure i.e. when handling small quantities. <b>OTHERWISE:</b> For potentially moderate or heavy exposures: • Safety glasses with side shields. • <b>NOTE:</b> Contact lenses pose a special hazard; soft lenses may absorb irritants and <b>ALL</b> lenses concentrate them.			
Skin protection	See Hand protection below			
Hands/feet protection	<ul> <li>No special equipment needed when handling small quantities.</li> <li>OTHERWISE:</li> <li>For potentially moderate exposures:</li> <li>Wear general protective gloves, eg. light weight rubber gloves.</li> <li>For potentially heavy exposures:</li> <li>Wear chemical protective gloves, eg. PVC. and safety footwear.</li> </ul>			
Body protection	See Other protection below			
Other protection	Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recomm For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs Non sparking safety or conductive footwear should be considered. Conductive footwear describes a chemically bound to the bottom components, for permanent control to electrically ground the foot an possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 c to the room in which they are worn. Personnel who have been issued conductive footwear should not return. No special equipment needed when handling small quantities. <b>OTHERWISE:</b> • Overalls. • Kin cleansing cream. • Eyewash unit. • Do not spray on hot surfaces.	s or pockets). boot or shoe with a sole n shall dissipate static elect hms. Conductive shoes s	nade from a conductive compound tricity from the body to reduce the should be stored in lockers close	
Thermal hazards	Not Available			
incina nazalus				

### Recommended material(s)

#### GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the: "Forsberg Clothing Performance Index".

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

Meguiar's G138 - NXT Hot Shine Tire Coating Aerosol

Material	CPI
BUTYL	С
BUTYL/NEOPRENE	С
CPE	С
HYPALON	С
NATURAL RUBBER	С
NATURAL+NEOPRENE	С
NEOPRENE	C
NITRILE	С
NITRILE+PVC	C
PE/EVAL/PE	С
PVA	С

### **Respiratory protection**

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	AX-AUS / Class1 P2	-
up to 50	1000	-	AX-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	AX-2 P2
up to 100	10000	-	AX-3 P2
100+			Airline**

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

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = (1 + 1)

PVC	С
PVDC/PE/PVDC	С
SARANEX-23	С
SARANEX-23 2-PLY	С
TEFLON	С
VITON/NEOPRENE	С

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

NOTE: As a series of factors will influence the actual performance of the glove, a final

selection must be based on detailed observation. -

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

### SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

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.

- Positive pressure, full face, air-supplied breathing apparatus should be used for work in enclosed spaces if a leak is suspected or the primary containment is to be opened (e.g. for a cylinder change)
- Air-supplied breathing apparatus is required where release of gas from primary containment is either suspected or demonstrated.

#### Information on basic physical and chemical properties

Appearance	Supplied as an aerosol pack. Contents under <b>PRESSURE</b> . Contains highly flammable hydrocarbon propellant. [Clear highly flammable liquid with a slight chemical odour; not miscible with water.			
Physical state	Liquid	Relative density (Water = 1)	0.78	
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 Applicable	Viscosity (cSt)	Not Available	
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable	
Flash point (°C)	-7 (PMCC)	Taste	Not Available	
Evaporation rate	Not Available	Explosive properties	Not Available	
Flammability	HIGHLY FLAMMABLE.	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 = 62%	
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	<ul> <li>Elevated temperatures.</li> <li>Presence of open flame.</li> <li>Product is considered stable.</li> <li>Hazardous polymerisation will not occur.</li> </ul>
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

	Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.
Inhaled	Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, 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. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects,

## Meguiar's G138 - NXT Hot Shine Tire Coating Aerosol

	slowed reaction time, slurred speech and may progress to un	consciousness. Serious poisonings may result in respiratory depression and may be fatal.		
	Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. 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 WARNING:Intentional misuse by concentrating/inhaling contents may be lethal. Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms.			
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. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments			
Skin Contact	<ul> <li>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</li> <li>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</li> <li>The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either: <ul> <li>produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or</li> <li>produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.</li> </ul> </li> <li>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.</li> <li>Spray mist may produce discomfort</li> <li>Open cuts, abraded or irritated skin should not be exposed to this material</li> <li>The material may accentuate any pre-existing dermatitis condition</li> <li>Excessive use or prolonged contact may lead to defatting, drying and irritation of sensitive skin</li> </ul>			
Eye	Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures 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. Evidence exists, or practical experience predicts, that the material may cause severe 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. Eye contact may cause significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. When the eyes of human subjects where exposed to silicone fluids, there was evidence of transitory conjunctival irritation within a few hours; this resolved within 24 hours. When applied to the eyes of rabbits, silicone fluids produced transitory irritation which lasted no longer than 48 hours. Injection into the various structures of the eye of animals produced corneal scarring, degenerative changes in the retina, foreign body reaction and cataracts.			
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. 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, particularly melanoma. Other studies have been unable to confirm this finding. Chronic inhalation or skin exposure to n-hexane may cause peripheral neuropathy, which is damage to nerve ends in extremities, e.g. fingers, with loss of sensation and characteristic thickening. Nerve damage has been documented with chronic exposures of greater than 500 ppm. Improvement in condition does not immediately follow removal from exposure and symptoms may progress for two or three months. Recovery may take a year or more depending on severity of exposure, and may not always be complete. Exposure to n-hexane with methyl ethyl ketone (MEK) will accelerate the appearance of damage, but MEK alone will not cause the nerve damage. Other i			
Manufarla 0420 NVT list	ΤΟΧΙϹΙΤΥ	IRRITATION		
Meguiar's G138 - NXT Hot Shine Tire Coating Aerosol	Not Available	Not Available		
methyl acetate	Not Available     Not Available       TOXICITY     IRRITATION       dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Eye (rabbit):100 mg/24h-moderate       Oral (rat) LD50: >5000 mg/kg <sup>[2]</sup> Skin (rabbit): 20 mg/24h - mild       Skin (rabbit): 500 mg/24h - mild     Skin (rabbit): 500 mg/24h - mild			
acetone	TOXICITY           Dermal (rabbit) LD50: 20000 mg/kg <sup>[2]</sup> Inhalation (rat) LC50: 50.1 mg/L/8 hr <sup>[2]</sup> Oral (rat) LD50: 5800 mg/kg <sup>[2]</sup>	IRRITATION         Eye (human): 500 ppm - irritant         Eye (rabbit): 20mg/24hr -moderate         Eye (rabbit): 3.95 mg - SEVERE         Skin (rabbit): 500 mg/24hr - mild         Skin (rabbit): 395 mg (open) - mild		

	ΤΟΧΙΟΙΤΥ	IRRITATION
naphtha petroleum, light,	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	Not Available
hydrotreated		
	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	
	ΤΟΧΙΟΙΤΥ	IRRITATION
polydimethylsiloxane	Dermal (rabbit) LD50: >2000 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg/1h - mild
	Oral (rat) LD50: >17000 mg/kg <sup>[2]</sup>	
	ΤΟΧΙΟΙΤΥ	IRRITATION
heptane	Inhalation (rat) LC50: 103 mg/L/4hr <sup>[2]</sup>	Nil reported
	ΤΟΧΙΟΙΤΥ	IRRITATION
naphtha petroleum, heavy,	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	[CCINFO-Shell]
hydrotreated	Oral (rat) LD50: >4500 mg/kg <sup>[1]</sup>	[EXXON]
		None reported
	ΤΟΧΙΟΙΤΥ	IRRITATION
	Inhalation (mouse) LC50: 200000 ppm/2hr <sup>[2]</sup>	Not Available
carbon dioxide	Inhalation (mouse) LC50: 361 mg/L/2hr <sup>[2]</sup>	
	Inhalation (rat) LC50: 470000 ppm/30M <sup>[2]</sup>	
Legend:	1. Value obtained from Europe ECHA Registered Substances - , extracted from RTECS - Register of Toxic Effect of chemical Su	Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data

### for methyl acetate

Acute toxicity:

Methyl acetate is a water soluble substance with high volatility. The substance has narcotic properties if inhaled at concentrations of 34 mg/l (mice) and 56 mg/l (cats) with a short duration of the narcotic action after cessation of exposure.

Methyl acetate is absorbed via the lungs in animals and humans, absorption via the oral route is demonstrated. After absorption the substance undergoes hydrolysis to methanol and acetic acid.

From the available *in vitro* data it may be anticipated that the half-life of methyl acetate in blood ranges between 2 and 4 hours. Immediately after stopping a 6-hour inhalation exposure to rats (2,000 ppm (6,040 mg/m3)) blood concentrations below the limit of quantification (less than 4.6 mg/l) were determined indicating rapid hydrolysis and high clearance of the substance. It appears from these data that the systemic availability of methyl acetate is low. The main metabolite is methanol which itself is metabolised to formic acid. Formate is introduced into C1-metabolism after activation by reacting with

tetrahydrofolate. Humans as well as monkeys are more sensitive to methanol poisoning compared with rats because of a lower tetrahydrofolate content in liver. Therefore interspecies differences in the metabolism were considered mainly of concern at dose levels leading to acute toxicity. Thus rat is a useful model to indicate subacute/subchronic toxic effects below sublethal dosages.

Assessment of the available animal toxicology data indicates that methyl acetate is of low acute toxicity (rats LD50 oral: 6,482 mg/kg bw, dermal: >2,000 mg/kg bw, LC50 inhalative >49 mg/l/4h). After oral application and after inhalation of substance vapours, animals showed narcotic symptoms, spasms, dyspnea and vomiting; inhalation of vapours in addition caused irritation of eyes and upper respiratory tract. The narcotic concentration for mice starts at 34 mg/l and for cats with 56 mg/l inhaled.

In humans, accidental inhalation of vapours of methyl acetate caused severe headache and considerable somnolence.

Methyl acetate has proven to cause only weak skin irritation in humans and in rabbits (no oedema, erythema with maximum grade 1 reversible within 48 hours). Eye irritation however, was strong but reversible within 7 days in a Draize eye test with rabbits. Exposure to methyl acetate vapours causes irritation to eyes and respiratory tract of humans.

Taking into account the long experience with human exposure to the substance, methyl acetate is not supposed to exhibit skin sensitising properties although no relevant human or animal date are available.

#### Sensitisation:

METHYL ACETATE

Relevant human data are not available. In a maximisation test with 25 volunteers no sensitisation was observed after exposure to 10% methyl acetate in petrolatum (Kligman, 1976). Taking into account the long experience with human exposure to the substance, and the absence of any reports on contact allergy in exposed persons, methyl acetate is not expected to exhibit skin sensitising properties, especially since the substance is hydrolysed in contact with water by non-specific tissue esterases to methanol and acetic acid. For these substances a skin sensitisation potential is either absent (methanol, or restricted to a few cases (acetic acid).

#### Repeat dose toxicity:

Overall, reliable experimental animal data on the local and systemic effects after repeated administration of methyl acetate are restricted to the inhalation exposure. After nose-only inhalation during a 28-day treatment period, methyl acetate induced degeneration/necrosis of the rat olfactory mucosa at a concentration of 2,000 ppm on 6 hours/day, 5 days/week (6,040 mg/m3). There was some concern on minimal effects of systemic toxicity at this concentration diverses, minimal liver cell dysfunction, adrenal weight increase, and reduced serum cholesterol concentrations).

There are no adequate data from human experience on repeated or prolonged exposure.

Based on general experience that acute and long-time or repeated exposure to methyl acetate defats skin and cause dryness and cracking of the skin. No-observed-adverse-effect-level (NOAEL)

#### Inhalation route

The NOAEC for local effects on the respiratory tract derived from an accurate 28-day inhalation study in rats was 350 ppm (1,057 mg/m3). The NOAEC for systemic effects also derived from a 28-day inhalation study was 350 ppm (1,057 mg/m3).

#### Mutagenicity

Methyl acetate is negative in a bacterial mutation test and a rat bone marrow micronucleus test. Furthermore, the hydrolysis products methanol and acetic acid do not reveal evidence for a mutagenic potential. There is no concern with respect to mutagenicity. Methyl acetate should not be classified as a mutagen. **Reproductive toxicity:** 

There are no data on reproductive toxicity of methyl acetate. However, due to the rapid hydrolysis of this compound it is justified to base hazard assessment with respect to reproduction on the toxicological properties of the immediate metabolites. Concerning the metabolites of methyl acetate, acetic acid appears to be of less significance, since there are no indications of a foetotoxic or teratogenic potential, whereas for methanol some embryo-/foetotoxic and teratogenic effects were demonstrated in rodents, however at relatively high concentrations, respectively maternal toxic concentrations only. A NOEC/fertility for methanol of 1,000 ppm (1,300 mg methanol/m3) was derived from a 2-generation inhalation study in rats. With the assumption that methyl acetate is immediately degraded to methanol at a molar ratio of 1, this value can be converted to NOAEC/fertility of about 3,000 mg methyl acetate/m3. A NOAEC/developmental toxicity for methanol of 1,000 ppm (1,300 mg methanol/m3) was derived from two studies in mice and rats from intermittent as well as from continuous inhalatory exposure,

	which can be converted to a NOAEC/developmental toxicity of about 3,000 mg methyl acetate/m3. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ACETONE	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.
NAPHTHA PETROLEUM, LIGHT, HYDROTREATED	No significant acute toxicological data identified in literature search. 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.
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 (decamethylcyclotetrasiloxane) and the short-linear HMDS (hexamethyldisiloxane). These three siloxanes have a relatively low order of acute toxicity by order 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 rifect. 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 antioestrogen
CARBON DIOXIDE	- pulmonary effects IDLH: 50,000 ppm
Meguiar's G138 - NXT Hot Shine Tire Coating Aerosol & NAPHTHA PETROLEUM, LIGHT, HYDROTREATED & POLYDIMETHYLSILOXANE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
Meguiar's G138 - NXT Hot Shine Tire Coating Aerosol & METHYL ACETATE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
Meguiar's G138 - NXT Hot Shine Tire Coating Aerosol & ACETONE	for acetone: The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone- induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals. The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies
NAPHTHA PETROLEUM, LIGHT, HYDROTREATED & NAPHTHA PETROLEUM, HEAVY, HYDROTREATED	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

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. <b>Reproductive Toxicity</b> : Repeated exposure of pregnant rats effects, such as lower birth weight and developmental neurotor vapour condensate, no adverse effects on the foetus were obse <b>Human Effects</b> : Prolonged/ repeated contact may cause defa and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogeni male rats as a consequence of accumulation of the alpha2-mit accumulation represents lysosomal overload and leads to chro tubules and necrosis. A sustained regenerative proliferation or alpha2-microglobulin is produced under the influence of hormore	kicity, on the foetus. However, in a tw aved. atting of the skin which can lead to de city although the relevance to human croglobulin protein in hyaline droplet onic renal tubular cell degeneration, ccurs in epithelial cells with subsequ	o-generation reproductive study in rats exposed to gasoline ermatitis and may make the skin more susceptible to irritation hs has been questioned. Gasoline induces kidney cancer in s in the male (but not female) rat kidney. Such abnormal accumulation of cell debris, mineralisation of renal medullary ent neoplastic transformation with continued exposure. The
Acute Toxicity	$\otimes$	Carcinogenicity	$\otimes$
Skin Irritation/Corrosion	✓	Reproductivity	×
Serious Eye Damage/Irritation	$\otimes$	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	0	STOT - Repeated Exposure	✓
Mutagenicity	$\odot$	Aspiration Hazard	$\odot$
		Legend: 🗙	- Data available but does not fill the criteria for classification

- Data required to make classification available
- N Data Not Available to make classification

#### **SECTION 12 ECOLOGICAL INFORMATION**

#### Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
methyl acetate	EC50	48	Crustacea	=1026.7mg/L	1
methyl acetate	EC50	96	Algae or other aquatic plants	6.261mg/L	3
methyl acetate	LC50	96	Fish	83.513mg/L	3
methyl acetate	EC10	72	Algae or other aquatic plants	>120mg/L	1
methyl acetate	NOEC	96	Fish	=100mg/L	1
acetone	EC50	384	Crustacea	97.013mg/L	3
acetone	EC50	48	Crustacea	>100mg/L	4
acetone	EC50	96	Algae or other aquatic plants	20.565mg/L	4
acetone	LC50	96	Fish	>100mg/L	4
acetone	NOEC	96	Algae or other aquatic plants	4.950mg/L	4
naphtha petroleum, light, hydrotreated	LC50	96	Fish	2.1-61.1mg/L	2
naphtha petroleum, light, hydrotreated	EC50	48	Crustacea	4.7mg/L	2
naphtha petroleum, light, hydrotreated	EC50	72	Algae or other aquatic plants	12.4mg/L	2
naphtha petroleum, light, hydrotreated	EC50	96	Algae or other aquatic plants	1.6-16.3mg/L	2
naphtha petroleum, light, hydrotreated	NOEC	72	Algae or other aquatic plants	6.47mg/L	2
polydimethylsiloxane	LC50	96	Fish	3.16mg/L	4
heptane	EC50	384	Crustacea	0.213mg/L	3
heptane	EC50	96	Algae or other aquatic plants	1.323mg/L	3
heptane	LC50	96	Fish	0.854mg/L	3
heptane	EC50	48	Crustacea	0.64mg/L	2
heptane	NOEC	504	Crustacea	0.17mg/L	2
naphtha petroleum, heavy, hydrotreated	EC50	96	Algae or other aquatic plants	64mg/L	2
carbon dioxide	EC50	384	Crustacea	12.472mg/L	3
carbon dioxide	EC50	96	Algae or other aquatic plants	237.138mg/L	3
carbon dioxide	LC50	96	Fish	53.413mg/L	3

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

It is well accepted that polydimethylsiloxane fluids become permanent residents of sediment but should not exert adverse environmental effects. 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

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

#### For ketones:

Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds

Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrateThe higher molecular weight ketones do no form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions

Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (*e.g.*, ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable.

Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.

For butane: log Kow: 2.89 Koc: 450-900

BCF: 1.9

**Environmental Fate** 

Environmental Fate

Terrestrial Fate: An estimated Koc value of 900, determined from a log Kow of 2.89 indicates that n-butane is expected to have low mobility in soil. Volatilisation of n-butane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 0.95 atm-cu m/mole, derived from its vapor pressure, 1820 mm Hg and water solubility, 61.2 mg/l. The potential for volatilisation of n-butane from dry soil surfaces may exist based upon its vapor pressure. While volatilistion from soil surfaces is expected to be the predominant fate process of n-butane released to soil, this compound is also susceptible to biodegradation. In one soil, a biodegradation rate of 1.8 mgC/day/kg dry soil was reported.

Aquatic fate: The estimated Koc value indicates that n-butane may adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 2.2 hours and 3 days, respectively. An estimated BCF of 33 derived from the log Kow suggests the potential for bioconcentration in aquatic organisms is moderate. While volatilisation from water surfaces is expected to be the major fate process for n-butane released to water, biodegradation of this compound is also expected to occur. In a screening study, complete biodegradation was reported in 34 days. In a second study using a defined microbial culture, it was reported that n-butane was degraded to 2-butanone and 2-butanol. Photolysis of n-butane in aquatic systems is not expected to be important.

Atmospheric fate: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and the vapour pressure, n-butane, is expected to exist solely as a gas in the ambient atmosphere. Gas-phase n-butane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 6.3 days, calculated from its rate constant of 2.54x10-12 cu cm/molecule-sec at 25 deg. Based on data for iso-octane and n-hexane, n-butane is not expected to absorb UV light in the environmentally significant range, >290 nm and probably will not undergo direct photolysis in the atmosphere. Experimental data showed that 7.7% of the n-butane fraction in a dark chamber reacted with nitrogen oxide to form the corresponding alkyl nitrate, suggesting nightime reactions with radical species and nitrogen oxides may contribute to the atmospheric transformation of n-butane.

For propane:

**Environmental Fate** 

Terrestrial fate:: An estimated Koc value of 460 determined from a log Kow of 2.36 indicates that propane is expected to have moderate mobility in soil. Volatilisation of propane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 7.07x10-1 atm-cu m/mole, derived from its vapor pressure, 7150 mm Hg, and water solubility, 62.4 mg/L. Propane is expected to volatilise from dry soil surfaces based upon its vapor pressure. Using cell suspensions of microorganisms isolated from soil and water, propane was oxidised to acetone within 24 hours, suggesting that biodegradation may be an important fate process in soil and sediment.

Aquatic fate: The estimated Koc value indicates that propane is expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant. Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 41 minutes and 2.6 days, respectively. An estimated BCF of 13.1 using log Kow suggests the potential for bioconcentration in aquatic organisms is low. After 192 hr, the trace concentration of propane contained in gasoline remained unchanged for both a sterile control and a mixed culture sample collected from ground water contaminated with gasoline. This indicates that biodegradation may not be an important fate process in water.

Atmospheric fate:: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and vapour pressure, propane is expected to exist solely as a gas in the ambient atmosphere. Gas-phase propane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 14 days, calculated from its rate constant of 1.15x10-12 cu cm/molecule-sec at 25 deg C. Propane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight. DO NOT discharge into sewer or waterways.

For n-hexane: log Kow: 3.17-3.94 BOD 5 if unstated: 2.21 COD: 0.04 ThOD: 3.52

#### Environmental fate:

Transport and Partitioning: The physical properties of *n*-hexane that affect its transport and partitioning in the environment are: water solubility of 9.5 mg/L; log[Kow] (octanol/water partition coefficient), estimated as 3.29; Henry's law constant, 1.69 atm-m3 mol; vapor pressure, 150 mm Hg at 25 C; and log[Koc] in the range of 2.90 to 3.61. As with many alkanes, experimental methods for the estimation of the Koc parameter are lacking, so that estimates must be made based on theoretical considerations.

The dominant transport process from water is volatilization. Based on mathematical models the half-life for *n*-hexane in bodies of water with any degree of turbulent mixing (e.g., rivers) would be less than 3 hours. For standing bodies of water (e.g. small ponds), a half-life no longer than one week (6.8 days) is estimated Based on the log octanol/water partition coefficient (i.e. log[Koc]) and the estimated log sorption coefficient (i.e. log[Koc]) *n*-hexane is not expected to become concentrated in biota. A calculated bioconcentration factor (BCF) of 453 for a fathead minnow further suggests a low potential for *n*-hexane to bioconcentrate or bioaccumulate in trophic food chains.

In soil, the dominant transport mechanism for *n*-hexane present near the surface probably is volatilisation (based on its Henry's law constant, water solubility, vapor pressure, and Koc). While its estimated Koc values suggest a moderate ability to sorb to soil particles, *n*-hexane has a density (0.6603 g/mL at 20 C) well below that of water and a very low water solubility of 9.5 mg/L. *n*-Hexane would, therefore, be viewed as a light nonaqueous phase liquid (LNAPL), which would suggest a low potential for leaching into the lower soil depths since the *n*-hexane would tend to float on the top of the saturated zone of the water table. *n*-Hexane would generally stay near the soil surface and, if not appreciably sorbed into the soil matrix, would be expected eventually to volatilise to the atmosphere. Exceptions would involve locations with shallow groundwater tables where there were large spills of hexane products. In such cases, the *n*-hexane could spread out to contaminant a large volume of soil materials.

Air: *n*-Hexane does not absorb ultraviolet (UV) light at 290 nm and is thus not expected to undergo direct photolysis reactions. The dominant tropospheric removal mechanism for *n*-hexane is generally regarded to be decomposition by hydroxyl radicals. Calculations assuming typical hydroxyl radical concentrations suggest a half-life of approximately 2.9 days. While *n*-hexane can react with nitrogen oxides to produce ozone precursors under controlled laboratory conditions, the smog-producing potential of *n*-hexane is very low compared to that of other alkanes or chlorinated VOCs. Hydroxyl ion reactions in the upper troposphere, therefore, are probably the primary mechanisms for *n*-hexane degradation in the atmosphere. As with most alkanes, *n*-hexane is resistant to hydrolysis

Water: Although few data are available dealing explicitly with the biodegradation of *n*-hexane in water, neither hydrolysis nor biodegradation in surface waters appears to be rapid compared with volatilization. In surface waters, as in the atmosphere, alkanes such as *n*-hexane would be resistant to hydrolysis. Biodegradation is probably the most significant degradation mechanism in groundwater. The ability of *Pseudomonas mendocina* bacteria to metabolise *n*-hexane in laboratory microcosms simulating groundwater conditions has been documented. Mixed bacterial cultures as well as pure cultures are documented as capable of metabolizing *n*-hexane under aerobic conditions. In general, linear alkanes (such as *n*-hexane) are viewed as the most readily biodegradable fractions in petroleum, particularly when oxygen is present in solution. Once introduced into groundwater, *n*-hexane my be fairly persistent since its degradation by chemical hydrolysis is slow and opportunities for biodegradation may be limited under anoxic conditions or where nutrients such as nitrogen or phosphorus are in limited supply.

Sediment and Soil: The most important biodegradation processes involve the conversion of the *n*-hexane to primary alcohols, aldehydes and, ultimately, into fatty acids. Similar processes are encountered with other light hydrocarbons such as heptane. In general, unless the *n*-hexane is buried at some depth within a soil or sediment, volatilisation is generally assumed to occur at a much more rapid rate than chemical or biochemical degradation processes. Once introduced into deeper sediments, *n*-hexane may be fairly persistent. Ecotoxicity:

Fish LC50 (96 h): Oncorhyncus mykiss 4.14 mg/l; Pimephales promelus 2.5 mg/l (flow through); Lepomis macrochirus 4.12 mg/l Daphnia EC50 (48 h): 3.87 mg/l for acetone:

log Kow: -0.24 Half-life (hr) air: 312-1896 Half-life (hr) H2O surface water: 20 Henry's atm m3 /mol: 3.67E-05 BOD 5: 0.31-1.76.46-55%

### Issue Date: 01/09/2015 Print Date: 22/08/2016

### Meguiar's G138 - NXT Hot Shine Tire Coating Aerosol

COD: 1.12-2.07 ThOD: 2.2 BCF 0.69 Environmental fate: Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source. Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades. Acetone does not concentrate in the food chain. Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period Drinking Water Standard: none available Soil Guidelines: none available Air Quality Standards: none available Ecotoxicity: Testing shows that acetone exhibits a low order of toxicity Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l Bird LC0 (5 day): Japanese guail, ring-neck pheasant 40,000 mg/ Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l

Aquatic invertebrate 2100 - 16700 mg/l

Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephestia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m3. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

#### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methyl acetate	LOW	LOW
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
heptane	LOW	LOW
carbon dioxide	LOW	LOW

#### **Bioaccumulative potential**

Ingredient	Bioaccumulation
methyl acetate	LOW (LogKOW = 0.18)
acetone	LOW (BCF = 0.69)
heptane	HIGH (LogKOW = 4.66)
carbon dioxide	LOW (LogKOW = 0.83)

#### Mobility in soil

Ingredient	Mobility
methyl acetate	MEDIUM (KOC = 3.324)
acetone	HIGH (KOC = 1.981)
heptane	LOW (KOC = 274.7)
carbon dioxide	HIGH (KOC = 1.498)

### SECTION 13 DISPOSAL CONSIDERATIONS

Product / Packaging disposal	<ul> <li>DO NOT allow wash water from cleaning or process equipment to enter drains.</li> <li>It may be necessary to collect all wash water for treatment before disposal.</li> <li>In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>Where in doubt contact the responsible authority.</li> <li>Consult State Land Waste Management Authority for disposal.</li> <li>Discharge contents of damaged aerosol cans at an approved site.</li> <li>Allow small quantities to evaporate.</li> <li>DO NOT incinerate or puncture aerosol cans.</li> <li>Bury residues and emptied aerosol cans at an approved site.</li> </ul>	
---------------------------------	---	--

#### **SECTION 14 TRANSPORT INFORMATION**

	FLANMARLL GAS		
Marine Pollutant			
HAZCHEM	Not Applicable		
Land transport (ADG)			
UN number	1950		
UN proper shipping name	AEROSOLS		
Transport hazard class(es)	Class     2.1       Subrisk     Not Applicable		
Packing group	Not Applicable	Not Applicable	
Environmental hazard	Not Applicable		
Special precautions for user	Special provisions63 190 277 327 344Limited quantity1000ml		
Air transport (ICAO-IATA / D	DGR)		
UN number	1950		
UN proper shipping name	Aerosols, flammable; Aerosols, flammable (engine starting fluid)		
Transport hazard class(es)	ICAO/IATA Class2.1ICAO / IATA SubriskNot ApplicableERG Code10L		
Packing group	Not Applicable		
Environmental hazard	Not Applicable		
	Special provisions Cargo Only Packing Instructions Cargo Only Maximum Qty / Pack	A145A167A802; A1A145A167A802 203 150 kg	
Special precautions for user	Passenger and Cargo Packing Instructions	203; Forbidden	
	Passenger and Cargo Maximum Qty / Pack	75 kg; Forbidden	

### Sea transport (IMDG-Code / GGVSee)

	· ·	
UN number	1950	
UN proper shipping name	AEROSOLS	
Transport hazard class(es)	IMDG Class     2.1       IMDG Subrisk     Not Applicable	
Packing group	Not Applicable	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS NumberF-D, S-USpecial provisions63 190 277 327 344 959Limited Quantities1000ml	

Y203; Forbidden

30 kg G; Forbidden

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

Passenger and Cargo Limited Quantity Packing Instructions

Passenger and Cargo Limited Maximum Qty / Pack

### METHYL ACETATE(79-20-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards Australia Hazardous Substances Information System - Consolidated Lists

Australia Exposure Standards	ID ON THE FOLLOWING REGULATORY LISTS	Australia Inventory of Chemical Substances (AICS)
Australia Exposure Standards Australia Hazardous Substances Information System - Consolidated Lists		Australia Inventory of Chemical Substances (AICS)
	GHT, HYDROTREATED(64742-49-0.) IS FOUND ON THE	
Australia Hazardous Substance	es Information System - Consolidated Lists	Australia Inventory of Chemical Substances (AICS)
POLYDIMETHYLSILOXANE(	63148-62-9) IS FOUND ON THE FOLLOWING REGULAT	TORY LISTS
Australia Inventory of Chemical	Substances (AICS)	
HEPTANE(142-82-5) IS FOUI	ND ON THE FOLLOWING REGULATORY LISTS	
Australia Exposure Standards		Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substance	es Information System - Consolidated Lists	
NAPHTHA PETROLEUM, HE	AVY, HYDROTREATED(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
CARBON DIOXIDE(124-38-9)	) IS FOUND ON THE FOLLOWING REGULATORY LIST	S
•	) IS FOUND ON THE FOLLOWING REGULATORY LIST	S Australia Inventory of Chemical Substances (AICS)
Australia Exposure Standards	) IS FOUND ON THE FOLLOWING REGULATORY LIST	
Australia Exposure Standards Australia Hazardous Substance	-	
Australia Exposure Standards Australia Hazardous Substance National Inventory	es Information System - Consolidated Lists	
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS	es Information System - Consolidated Lists Status	
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS Canada - DSL	Status     Y     Y	
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS Canada - DSL Canada - NDSL	Status     Y     Y	Australia Inventory of Chemical Substances (AICS)
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS Canada - DSL Canada - NDSL China - IECSC Europe - EINEC / ELINCS /	es Information System - Consolidated Lists          Status         Y         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphth	Australia Inventory of Chemical Substances (AICS)
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS Canada - DSL Canada - NDSL China - IECSC Europe - EINEC / ELINCS / NLP	es Information System - Consolidated Lists          Status         Y         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphth         Y	Australia Inventory of Chemical Substances (AICS)
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS Canada - DSL Canada - NDSL China - IECSC Europe - EINEC / ELINCS / NLP Japan - ENCS	es Information System - Consolidated Lists          Status         Y         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphth         Y         N (polydimethylsiloxane)	Australia Inventory of Chemical Substances (AICS)
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS Canada - DSL Canada - NDSL China - IECSC Europe - EINEC / ELINCS / NLP Japan - ENCS Korea - KECI	es Information System - Consolidated Lists          Status         Y         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphthere         Y         N (polydimethylsiloxane)         N (polydimethylsiloxane; naphtha petroleum, heavy, hydromethylsiloxane;	Australia Inventory of Chemical Substances (AICS)
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS Canada - DSL Canada - DSL Canada - NDSL China - IECSC Europe - EINEC / ELINCS / NLP Japan - ENCS Korea - KECI New Zealand - NZIoC	es Information System - Consolidated Lists          Status         Y         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphth         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphth         Y         N (polydimethylsiloxane; naphtha petroleum, heavy, hydro         Y	Australia Inventory of Chemical Substances (AICS)
Australia Exposure Standards Australia Hazardous Substance National Inventory Australia - AICS	es Information System - Consolidated Lists          Status         Y         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphth         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphth         Y         N (polydimethylsiloxane; acetone; carbon dioxide; naphth         Y         N (polydimethylsiloxane)         N (polydimethylsiloxane; naphtha petroleum, heavy, hydro         Y         Y         Y         Y         Y         Y	Australia Inventory of Chemical Substances (AICS)

### **SECTION 16 OTHER INFORMATION**

#### Other information

#### Ingredients with multiple cas numbers

Name	CAS No
heptane	142-82-5, 31394-54-4
naphtha petroleum, heavy, hydrotreated	64742-48-9., 101795-02-2.

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 LOAEL: Lowest Observed Adverse Effect Level LODEL: Imit of Detection OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index

This document is copyright. Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH. TEL (+61 3) 9572 4700.