

Meguiars G181 Clear Coat Safe Polishing Compound (21-61A)

Motor Active

Chemwatch Hazard Alert Code: 2

Chemwatch: 4799-45

Version No: 5.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Issue Date: 05/09/2019

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

Product Identifier

Product name	Meguiars G181 Clear Coat Safe Polishing Compound (21-61A)
Synonyms	Product Code: 21-61A, G18110, G18116
Other means of identification	Not Available

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

Relevant identified uses	Automotive; polishing agent / burnishing compound. Use according to manufacturer's directions.
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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	
Toxicity	1	
Body Contact	2	
Reactivity	1	
Chronic	2	

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

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Skin Sensitizer Category 1, Carcinogenicity Category 2, Reproductive Toxicity Category 2, Specific target organ toxicity - repeated exposure Category 2, Aspiration Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.
H361d	Suspected of damaging the unborn child.

Continued...

H373	May cause damage to organs through prolonged or repeated exposure.
H304	May be fatal if swallowed and enters airways.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P321	Specific treatment (see advice on this label).
P331	Do NOT induce vomiting.
P362	Take off contaminated clothing and wash before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P314	Get medical advice/attention if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.

Precautionary statement(s) Storage

P405	Store locked up.
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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
93821-35-3	7-13	<u>calcined flint clay</u>
64742-88-7	7-13	<u>solvent naphtha petroleum, medium aliphatic</u>
64742-46-7	1-5	<u>distillates, petroleum, middle, hydrotreated</u>
56-81-5	1-5	<u>glycerol</u>
8001-79-4	0.5-1.5	<u>castor oil</u>
102-71-6	0.1-1	<u>triethanolamine</u>
Not Available	70-90	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES**Description of first aid measures**

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ 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.

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Ingestion

- ▶ If swallowed do **NOT** induce vomiting.
- ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- ▶ Observe the patient carefully.
- ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- ▶ Seek medical advice.
- ▶ Avoid giving milk or oils.
- ▶ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

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

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

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

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

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 dioxide (CO₂)
nitrogen oxides (NO_x)
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

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills

- ▶ Clean up all spills immediately.
- ▶ Avoid breathing vapours/ aerosols/ or dusts and avoid contact with skin and eyes.
- ▶ Control personal contact with the substance, by using protective equipment.
- ▶ Contain and absorb spill with sand, earth, inert material or vermiculite.
- ▶ Place in a suitable, labelled container for waste disposal.

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).

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- ▶ 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	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Keep dry. ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials. ▶ Protect containers against physical damage. ▶ Check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid 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	solvent naphtha petroleum, medium aliphatic	Oil mist, refined mineral	5 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	distillates, petroleum, middle, hydrotreated	Oil mist, refined mineral	5 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	glycerol	Glycerin mist	10 mg/m ³	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	triethanolamine	Triethanolamine	5 mg/m ³	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
glycerol	Glycerine (mist); (Glycerol; Glycerin)	45 mg/m ³	860 mg/m ³	2,500 mg/m ³
triethanolamine	Triethanolamine; (Trihydroxytriethylamine)	15 mg/m ³	240 mg/m ³	1,500 mg/m ³
Ingredient	Original IDLH	Revised IDLH		
calcined flint clay	Not Available	Not Available		
solvent naphtha petroleum, medium aliphatic	2,500 mg/m ³	Not Available		
distillates, petroleum, middle, hydrotreated	2,500 mg/m ³	Not Available		

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glycerol	Not Available	Not Available
castor oil	Not Available	Not Available
triethanolamine	Not Available	Not Available

MATERIAL DATA

for benzene

Odour Threshold Value: 34 ppm (detection), 97 ppm (recognition)

NOTE: Detector tubes for benzene, measuring in excess of 0.5 ppm, are commercially available. The relative quality of epidemiological data and quantitative health risk assessments related to documented and theoretical leukaemic deaths constitute the basis of the TLV-recommendation.

One study [Dow Chemical] demonstrates a significant fourfold increase in myelogenous leukaemia for workers exposed to average benzene concentrations of about 5 ppm for an average of 9 years and that 2 out of four individuals in the study who died from leukaemia were characterised as having been exposed to average benzene levels below 2 ppm. Based on such findings the estimated risk of leukaemia in workers exposed at daily benzene concentrations of 10 ppm for 40 years is 155 times that of unexposed workers; at 1 ppm the risk falls to 1.7 times whilst at 0.1 ppm the risk is about the same in the two groups. A revision of the TLV-TWA to 0.1 ppm was proposed in 1990 but this has been revised upwards as result of industry initiatives.

Typical toxicities displayed following inhalation:

- ▶ At 25 ppm (8 hours): no effect
- ▶ 50-150 ppm: signs of intoxication within 5 hours
- ▶ 500-1500 ppm: signs of intoxication within 1 hour
- ▶ 7500 ppm: severe intoxication within 30-60 minutes
- ▶ 20000 ppm: fatal within 5-10 minutes

Some jurisdictions require that health surveillance be conducted on occupationally exposed workers. Some surveillance should emphasise (i) demography, occupational and medical history and health advice (ii) baseline blood sample for haematological profile (iii) records of personal exposure.

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. Beaudry et al: Journal of Occupational and Environmental Hygiene 10: 71-77; 2013

for triethanolamine:

Exposure at or below the TLV-TWA is thought to minimise the potential for skin and eye irritation, and acute effects (including liver, kidney and nerve damage) and chronic effects (including cancer and allergic contact dermatitis).

Odour Safety Factor (OSF)

OSF=0.77 (triethanolamine)

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p>																			
	<p>General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear 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, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	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.)	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
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Personal protection																				

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Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

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:

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Material	CPI
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
PVA	C
PVC	C

* 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.

Respiratory protection

Type AK-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	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - Full-face

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

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ 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 paste with sweet chemical odour; partly mixes with water.		
Physical state	Free-flowing Paste	Relative density (Water = 1)	1.01
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	8.30	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Applicable	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	193	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>93 (CC)	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available

Continued...

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Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	VOC = 11.08% (by wt)
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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</p>
Ingestion	<p>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.</p> <p>Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p>
Skin Contact	<p>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.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>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.</p>
Eye	<p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p>
Chronic	<p>Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>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.</p>
	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent</p>

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following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

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

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

Meguiars G181 Clear Coat Safe Polishing Compound (21-61A)	TOXICITY	IRRITATION
	Not Available	Not Available
calcined flint clay	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[1]	
	Oral (rat) LD50: >2000 mg/kg ^[1]	
solvent naphtha petroleum, medium aliphatic	TOXICITY	IRRITATION
	dermal (rat) LD50: 28000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: >5000 mg/kg ^[2]	Skin: adverse effect observed (irritating) ^[1]
distillates, petroleum, middle, hydrotreated	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (rat) LC50: 7.64 mg/l4 h ^[1]	Skin: adverse effect observed (irritating) ^[1]
	Oral (rat) LD50: >5000 mg/kg ^[2]	
glycerol	TOXICITY	IRRITATION
	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available
castor oil	TOXICITY	IRRITATION
	Oral (rat) LD50: >4600 mg/kg ^[1]	Eye (rabbit): 500 mg mild
		Skin (human): 50 mg/48h mild Skin (rabbit): 100 mg/24h SEVERE
triethanolamine	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Eye (rabbit): 0.1 ml -
	Oral (rat) LD50: 4190 mg/kg ^[2]	Eye (rabbit): 10 mg - mild
		Eye (rabbit): 5.62 mg - SEVERE
		minor conjunctival irritation
		no irritation *
		Skin (human): 15 mg/3d (int)-mild Skin (rabbit): 4 h occluded Skin (rabbit): 560 mg/24 hr- mild

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

**SOLVENT NAPHTHA
PETROLEUM, MEDIUM
ALIPHATIC**

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.

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This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.

This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents

Carcinogenicity: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.

Mutagenicity: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.

Reproductive Toxicity: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.

Human Effects: Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.

Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.

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.

For toluene:

Acute Toxicity

Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.

Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.

Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.

Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.

Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea. Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death

Toluene can also strip the skin of lipids causing dermatitis

Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days

Subchronic/Chronic Effects:

Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.

Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin.

Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L

Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day).

Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely affect the developing offspring in laboratory animals.

Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy

Animals - Sterebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m³. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.

Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene.

Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues.

Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.

DISTILLATES, PETROLEUM, MIDDLE, HYDROTREATED

The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives;

The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:

- The adverse effects of these materials are associated with undesirable components, and
- The levels of the undesirable components are inversely related to the degree of processing;
- Distillate base oils receiving the same degree or extent of processing will have similar toxicities;
- The potential toxicity of *residual base oils* is independent of the degree of processing the oil receives.
- The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing.

The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential.

Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and

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severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.

Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing

Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).

Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class (Other Lubricant Base Oils).

Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class (Other Lubricant Base Oils))

Germ cell mutagenicity: The tests performed within the "in vivo" studies regarding gene mutation at mice micronuclei indicated negative results

(CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class (Other Lubricant Base Oils)).

Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance

is not toxic for reproduction.

STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m³ and for systemic effects NOAEL > 980 mg/m³.

Sub-chronic toxicity

90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies).

Repeat dose toxicity:

Oral

NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.

Inhalation

The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m³. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m³.

Dermal

In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.

Toxicity to reproduction:

Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.

Developmental toxicity, teratogenicity:

Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.

The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic

Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.

Highly and Severely Refined Distillate Base Oils

Acute toxicity: Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to > 4 mg/l.

When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating"

Testing in guinea pigs for sensitization has been negative

Repeat dose toxicity: Several studies have been conducted with these oils. The weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil's toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/or the peculiarities of the study.

- ▶ The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,
- ▶ The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and
- ▶ The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.

Reproductive and developmental toxicity: A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study's authors.

A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed fetuses were found among three litters. The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.

Genotoxicity:

In vitro (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay. Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.

In vivo (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.

Carcinogenicity: Highly & severely refined base oils are not carcinogens, when given either orally or dermally. typical for isoparaffinic hydrocarbons: isoparaffinic hydrocarbon:

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GLYCEROL	<p>For glycerol:</p> <p>Acute toxicity: Glycerol is of a low order of acute oral and dermal toxicity with LD50 values in excess of 4000 mg/kg bw. At very high dose levels, the signs of toxicity include tremor and hyperaemia of the gastro-intestinal tract. Skin and eye irritation studies indicate that glycerol has low potential to irritate the skin and the eye. The available human and animal data, together with the very widespread potential for exposure and the absence of case reports of sensitisation, indicate that glycerol is not a skin sensitiser.</p> <p>Repeat dose toxicity: Repeated oral exposure to glycerol does not induce adverse effects other than local irritation of the gastro-intestinal tract. The overall NOEL after prolonged treatment with glycerol is 10,000 mg/kg bw/day (20% in diet). At this dose level no systemic or local effects were observed. For inhalation exposure to aerosols, the NOAEC for local irritant effects to the upper respiratory tract is 165 mg/m³ and 662 mg/m³ for systemic effects.</p> <p>Genotoxicity: Glycerol is free from structural alerts, which raise concern for mutagenicity. Glycerol does not induce gene mutations in bacterial strains, chromosomal effects in mammalian cells or primary DNA damage <i>in vitro</i>. Results of a limited gene mutation test in mammalian cells were of uncertain biological relevance. <i>In vivo</i>, glycerol produced no statistically significant effect in a chromosome aberrations and dominant lethal study. However, the limited details provided and the absence of a positive control, prevent any reliable conclusions to be drawn from the <i>in vivo</i> data. Overall, glycerol is not considered to possess genotoxic potential.</p> <p>Carcinogenicity: The experimental data from a limited 2 year dietary study in the rat does not provide any basis for concerns in relation to carcinogenicity. Data from non-guideline studies designed to investigate tumour promotion activity in male mice suggest that oral administration of glycerol up to 20 weeks had a weak promotion effect on the incidence of tumour formation.</p> <p>Reproductive and developmental toxicity: No effects on fertility and reproductive performance were observed in a two generation study with glycerol administered by gavage (NOAEL 2000 mg/kg bw/day). No maternal toxicity or teratogenic effects were seen in the rat, mouse or rabbit at the highest dose levels tested in a guideline comparable teratogenicity study (NOEL 1180 mg/kg bw/day).</p>
CASTOR OIL	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.</p> <p>Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p> <p>Some tumorigenic effects have been reported in animal studies The castor seed contains ricin, a toxic protein. Heating during the oil extraction process denatures and inactivates the protein. However, harvesting castor beans may not be without risk. Allergenic compounds found on the plant surface can cause permanent nerve damage, making the harvest of castor beans a human health risk. The United States Food and Drug Administration (FDA) has categorized castor oil as "generally recognized as safe and effective" (GRASE) for over-the-counter use as a laxative with its major site of action the small intestine where it is digested into ricinoleic acid. Despite castor oil being widely used to start labor in pregnant women, to date there is not enough research to show whether it is effective to ripen the cervix or induce labour Due to its foul taste a heavy dose of castor oil was formerly used as a humiliating punishment for children and adults. Victims of this treatment did sometimes die, as the dehydrating effects of the oil-induced diarrhea; however, even those victims who survived had to bear the humiliation of the laxative effects resulting from excessive consumption of the oil.</p>
TRIETHANOLAMINE	<p>While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.</p> <ul style="list-style-type: none"> ▶ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis. ▶ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient. <p>Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.</p> <p>Inhalation:</p> <p>Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs.</p> <p>Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.</p> <p>Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies.</p> <p>While most polyurethane amine catalysts are not sensitizers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease.</p> <p>Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.</p> <p>Skin Contact:</p> <p>Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.</p> <p>Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.</p> <p>Eye Contact:</p> <p>Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations.</p> <p>Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.)</p> <p>Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.</p> <p>The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases.</p> <p>Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.</p> <p>Ingestion:</p> <p>The oral toxicity of amine catalysts varies from moderately to very toxic.</p> <p>Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract.</p> <p>Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.</p> <p>Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.</p> <p>Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000 Alliance for Polyurethanes Industry</p> <p>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.</p> <p>For triethanolamine (and its salts):</p> <p>Acute toxicity: Triethanolamine is of low toxicity by the oral, dermal and inhalation routes of exposure. Oral LD50 values have been shown to range from</p>

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	<p>approximately 5-10 g/kg. The dermal LD50 is greater than 2 g/kg. The inhalation LC50 is greater than a saturated atmosphere</p> <p>Repeat Dose Toxicity: The studies to determine toxicity of triethanolamine from repeated exposure were conducted for a duration of 91 days or 2 years. In both studies the NOAEL was at least 1000 mg/kg. There was no evidence of gross or histopathological change that could be attributed to treatment. Also, triethanolamine was shown to be non-carcinogenic.</p> <p>Genetic Toxicity: Mutation (bacterial); This endpoint has been satisfied by two studies using 4 strains (TA 98, TA 100, TA 1535 and TA 1537) of <i>Salmonella typhimurium</i>. Triethanolamine was not mutagenic in any of the tester strains.</p> <p>Chromosomal aberration (mammalian, <i>in vitro</i>) – This endpoint was satisfied by a cytogenetic assay using Chinese hamster lung cells. Triethanolamine did not induce chromosome aberrations in this test system.</p> <p>Reproductive Toxicity: No studies have been conducted to specifically evaluate the effect of triethanolamine on reproductive performance. However, based on consideration of the repeat dose toxicity studies of at least 90 days duration, there were no abnormalities noted in the histopathological examination of reproductive organs. This fact, and the lack of effects on foetal development, allow the conclusion that triethanolamine would not be expected to produce adverse effects to reproductive performance and fertility.</p> <p>Developmental Toxicity: This endpoint was satisfied using a developmental toxicity screening study according to the Chernoff-Kavlock method. Based on the results from this test, triethanolamine does not impair development of the fetus.</p> <p>551teapcp</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA. Lachrymation, diarrhoea, convulsions, urinary tract changes, changes in bladder weight, changes in testicular weight, changes in thymus weight, changes in liver weight, dermatitis after systemic exposure, kidney, ureter, bladder tumours recorded. Equivocal tumourigen by RTECS criteria. Dermal rabbit value quoted above is for occluded patch in male or female animals * Union Carbide</p>
Meguiars G181 Clear Coat Safe Polishing Compound (21-61A) & TRIETHANOLAMINE	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
Meguiars G181 Clear Coat Safe Polishing Compound (21-61A) & CALCINED FLINT CLAY	No significant acute toxicological data identified in literature search.
SOLVENT NAPHTHA PETROLEUM, MEDIUM ALIPHATIC & DISTILLATES, PETROLEUM, MIDDLE, HYDROTREATED	<p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.</p>
SOLVENT NAPHTHA PETROLEUM, MEDIUM ALIPHATIC & TRIETHANOLAMINE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
GLYCEROL & TRIETHANOLAMINE	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>

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

Meguiars G181 Clear Coat Safe Polishing Compound (21-61A)	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
calcined flint clay	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.57mg/L	2
	EC50	48	Crustacea	1.5mg/L	2

Continued...

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	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
solvent naphtha petroleum, medium aliphatic	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	18mg/L	2
	EC50	48	Crustacea	1.4mg/L	2
	EC50	72	Algae or other aquatic plants	3.7mg/L	2
distillates, petroleum, middle, hydrotreated	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.13mg/L	2
	EC50	48	Crustacea	2mg/L	2
	EC50	72	Algae or other aquatic plants	1.714mg/L	2
	NOEC	48	Crustacea	=10mg/L	1
glycerol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>0.011-mg/L	2
	EC50	96	Algae or other aquatic plants	77712.039mg/L	3
castor oil	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>100mg/L	2
	NOEC	72	Algae or other aquatic plants	100mg/L	2
triethanolamine	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	11-800mg/L	2
	EC50	48	Crustacea	609.88mg/L	2
	EC50	96	Algae or other aquatic plants	169mg/L	1
	EC0	24	Crustacea	1-530mg/L	2
	NOEC	504	Crustacea	16mg/L	1

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

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 > methyl naphthalenes > 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 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

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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
glycerol	LOW	LOW
triethanolamine	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
glycerol	LOW (LogKOW = -1.76)
triethanolamine	LOW (BCF = 3.9)

Mobility in soil

Ingredient	Mobility
glycerol	HIGH (KOC = 1)
triethanolamine	LOW (KOC = 10)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

Safety, health and environmental regulations / legislation specific for the substance or mixture**CALCINED FLINT CLAY IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)

SOLVENT NAPHTHA PETROLEUM, MEDIUM ALIPHATIC IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List

Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes

Australia Exposure Standards

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Air Transport Association (IATA) Dangerous Goods Regulations

International FOSFA List of Banned Immediate Previous Cargoes

International Maritime Dangerous Goods Requirements (IMDG Code)

United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

DISTILLATES, PETROLEUM, MIDDLE, HYDROTREATED IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International FOSFA List of Banned Immediate Previous Cargoes

GLYCEROL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

GESAMP/EHS Composite List - GESAMP Hazard Profiles

IMO IBC Code Chapter 17: Summary of minimum requirements

IMO IBC Code Chapter 18: List of products to which the Code does not apply

IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances

CASTOR OIL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

GESAMP/EHS Composite List - GESAMP Hazard Profiles

IMO IBC Code Chapter 17: Summary of minimum requirements

IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk

TRIETHANOLAMINE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

GESAMP/EHS Composite List - GESAMP Hazard Profiles

IMO IBC Code Chapter 17: Summary of minimum requirements

IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSDL	No (solvent naphtha petroleum, medium aliphatic; glycerol; triethanolamine; castor oil; distillates, petroleum, middle, hydrotreated; calcined flint clay)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (distillates, petroleum, middle, hydrotreated; calcined flint clay)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	05/09/2019
Initial Date	21/01/2013

SDS Version Summary

Version	Issue Date	Sections Updated
5.1.1.1	05/09/2019	Appearance, Physical Properties

Other information

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

Meguiars G181 Clear Coat Safe Polishing Compound (21-61A)

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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