CRC Industries (CRC Industries New Zealand)

Chemwatch: 4546-42

Version No: 11.1

Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

emwatch Hazard Alert Code: 4

Issue Date: 31/05/2023 Print Date: 24/10/2023 L.GHS.NZL.EN

SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	CRC 5040 Engine Start (NZ)	
Chemical Name	Not Applicable	
Synonyms	CRC 5040 Jump Start; CRC 5040 Engine Start; Jump Start	
Proper shipping name	AEROSOLS	
Chemical formula	Not Applicable	
Other means of identification	Not Available	

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

Polovant identified uses	Engine starting fluid.
Relevant identified uses	Application is by spray atomisation from a hand held aerosol pack

Details of the manufacturer or supplier of the safety data sheet

Registered company name	CRC Industries (CRC Industries New Zealand)
Address	10 Highbrook Drive East Tamaki Auckland New Zealand
Telephone	+64 9 272 2700
Fax	+64 9 274 9696
Website	www.crc.co.nz
Email	info.nz@crc.co.nz

Emergency telephone number

Association / Organisation	CRC Industries (CRC Industries New Zealand)	CHEMWATCH EMERGENCY RESPONSE (24/7)	
Emergency telephone numbers	NZ Poisons Centre 0800 POISON (0800 764 766)	+64 800 700 112	
Other emergency telephone numbers	111 (NZ Emergency Services)	+61 3 9573 3188	

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

Classification ^[1]	Aerosols Category 1, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Carcinogenicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	2.1.2A, 6.4A, 6.7B, 6.9B (narcotic effects), 9.1B

Label elements



Signal word Danger

Hazard statement(s)

H222+H229	Extremely flammable aerosol. Pressurized container: may burst if heated.
H319	Causes serious eye irritation.
H336	May cause drowsiness or dizziness.
H351	Suspected of causing cancer.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.	
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.	
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.	
P337+P313	If eye irritation persists: Get medical advice/attention.	
P391	Collect spillage.	
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.	

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
8006-61-9	30-60	gasoline
60-29-7	5-20	diethyl ether
64-17-5	1-9	ethanol
63748-98-1	1-9	mineral oil
124-38-9	1-9	carbon dioxide
Not Available		NOTE: Manufacturer has supplied full igredient
Not Available		information to allow CHEMWATCH assessment.
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L * EU IOELVs available	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	 If aerosols come in contact with the eyes: Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes 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. Transport to hospital or doctor without delay. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	 If solids or aerosol mists are deposited upon the skin: Flush skin and hair with running water (and soap if available). Remove any adhering solids with industrial skin cleansing cream. DO NOT use solvents. Seek medical attention in the event of irritation.
Inhalation	 If aerosols, fumes or combustion products are inhaled: Remove to fresh air. 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. If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor.
Ingestion	 Avoid giving milk or oils. Avoid giving alcohol. Not considered a normal route of entry.

Indication of any immediate medical attention and special treatment needed

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

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

Treat symptomatically.

for lower alkyl ethers:

BASIC TREATMENT

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

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- * Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension without signs of hypovolaemia may require vasopressors.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST),

calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.

* Ethers may produce anion gap acidosis. Hyperventilation and bicarbonate therapy might be indicated.

Haemodialysis might be considered in patients with impaired renal function.

Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L.

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

SECTION 5 Firefighting measures

Extinguishing media

SMALL FIRE: • Water spray, dry chemical or CO2 LARGE FIRE:

Water spray or fog.

Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may
The meenputisinty	result

Advice for firefighters

Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. May be violently or explosively reactive. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. If safe, switch off electrical equipment until vapour fire hazard removed. 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	 Liquid and vapour are highly flammable. Severe fire hazard when exposed to heat or flame. Vapour forms an explosive mixture with air. Severe explosion hazard, in the form of vapour, when exposed to flame or spark. Vapour may travel a considerable distance to source of ignition. Heating may cause expansion or decomposition with violent container rupture. Aerosol cans may explode on exposure to naked flames. Rupturing containers may rocket and scatter burning materials. Hazards may not be restricted to pressure effects. May emit acrid, poisonous or corrosive fumes. On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) other pyrolysis products typical of burning organic material.

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

	 Clean up all spills immediately. Avoid breathing vapours and contact with skin and eyes. Wear protective clothing impervious gloves and safety glasses.
Minor Spills	Shut off all possible sources of inplicing and increase ventilation
	 Wipe up.
	If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.
	Undamaged cans should be gathered and stowed safely.

Chemwatch: 4546-42	Page 5 of 20 Issue Date:	31/05/2023
Version No: 11.1	CRC 5040 Engine Start (NZ) Print Date:	24/10/2023
Major Spills	 Remove leaking cylinders to a safe place if possible. Release pressure under safe, controlled conditions by opening the valve. DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve. Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. May be violently or explosively reactive. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water courses No smoking, naked lights or ignition sources. Increase ventilation. Stop leak if safe to do so. Water spray or fog may be used to disperse / absorb vapour. Absorb or cover spill with sand, earth, inert materials or vermiculite. If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated undamaged cans should be gathered and stowed safely. Collect residues and seal in labelled drums for disposal. 	ıted.

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

SECTION 7 Handling and storage

Precautions for safe handling

	 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. Avoid smoking, naked lights or ignition sources.
Safe handling	 When handling, DO NOT eat, drink or smoke. DO NOT incinerate or puncture aerosol cans. DO NOT spray directly on humans, exposed food or food utensils. Avoid physical damage to containers. Always wash hands with soap and water after handling. Work clothes should be laundered separately. 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	 Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can Store in original containers in approved flammable liquid storage area. DO NOT store in pits, depressions, basements or areas where vapours may be trapped. No smoking, naked lights, heat or ignition sources. Keep containers securely sealed. Contents under pressure. Store away from incompatible materials. Store in a cool, dry, well ventilated area. Avoid storage at temperatures higher than 40 deg C. Store in an upright position. Protect containers against physical damage. Check regularly for spills and leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	 Aerosol dispenser. Check that containers are clearly labelled.
Storage incompatibility	Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace	gasoline	Gasoline (Petrol)	300 ppm / 890	1480 mg/m3 / 500	Not	Not Available

Version No: 11.1

CRC 5040 Engine Start (NZ)

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Exposure Standards (WES)			mg/m3	ppm	Available	
New Zealand Workplace Exposure Standards (WES)	diethyl ether	Diethyl ether (Ethyl ether)	400 ppm / 1210 mg/m3	1520 mg/m3 / 500 ppm	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	ethanol	Ethanol (Ethyl alcohol)	1000 ppm / 1880 mg/m3	Not Available	Not Available	oto - Ototoxin
New Zealand Workplace Exposure Standards (WES)	mineral oil	Oil mist, mineral	5 mg/m3	10 mg/m3	Not Available	(om) - Sampled by a method that does not collect vapour
New Zealand Workplace Exposure Standards (WES)	carbon dioxide	Carbon dioxide	5000 ppm / 9000 mg/m3	54000 mg/m3 / 30000 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3	
gasoline	Not Available	Not Available	Not Available	
diethyl ether	500 ppm	3200* ppm	19000*** ppm	
ethanol	Not Available	Not Available	15000* ppm	
mineral oil	140 mg/m3	1,500 mg/m3	8,900 mg/m3	

Ingredient	Original IDLH	Revised IDLH
gasoline	Not Available	Not Available
diethyl ether	1,900 ppm	Not Available
ethanol	3,300 ppm	Not Available
mineral oil	2,500 mg/m3	Not Available
carbon dioxide	40,000 ppm	Not Available

MATERIAL DATA

Odour Safety Factor(OSF) OSF=0.042 (gasoline)

Odour threshold: 0.25 ppm.

The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m3 is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.

Odour Safety Factor (OSF)

OSF=0.042 (gasoline)

For diethyl ether:

NOTE: Detector tubes for diethyl ether, measuring in excess of 100 ppm, are commercially available.

Narcotic properties leading to anaesthesia and eye and respiratory irritation are thought to be minimised at exposures at or below the recommended TLV-TWA. Disagreement exists amongst peak bodies (notably NIOSH) as to whether this limit limits sensory limitation. Odour Safety Factor (OSF)

OSF=45 (ETHYL ETHER)

For ethanol:

Odour Threshold Value: 49-716 ppm (detection), 101 ppm (recognition)

Eye and respiratory tract irritation do not appear to occur at exposure levels of less than 5000 ppm and the TLV-TWA is thought to provide an adequate margin of safety against such effects. Experiments in man show that inhalation of 1000 ppm caused slight symptoms of poisoning and 5000 ppm caused strong stupor and morbid sleepiness. Subjects exposed to 5000 ppm to 10000 ppm experienced smarting of the eyes and nose and coughing. Symptoms disappeared within minutes. Inhalation also causes local irritating effects to the eyes and upper respiratory tract, headaches, sensation of heat intraocular tension, stupor, fatigue and a need to sleep. At 15000 ppm there was continuous lachrymation and coughing.

None assigned. Refer to individual constituents.

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

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

The Odour Safety Factor (OSF) is defined as:

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

550

working activities

A

CRC 5040 Engine Start (NZ)

Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by

B 26-550As "A" for 50-90% C 1-26 As "A" for less tha D 0.18-1 10-50% of person E <0.18 As "D" for less tha NOTE P: The classification as Note E shall also apply when th	of persons being distracted n 50% of persons being distracted s aware of being tested perceive by smell that the Exposure Standard is being reached in 10% of persons aware of being tested a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7 he substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI. armoniced classification and labelling hazardous substances. Table 3.1. Appay VI. Regulation (EC) No 1272/2008 (CLP) - up to the
latest ATP	
Exposure controls	
Appropriate engineering controls	 Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulate area. Work should be undertaken in an isolated system such as a "glove-box". Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. Open-vessel systems are prohibited. Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear
Individual protection measures, such as personal protective equipment	
Eye and face protection	No special equipment for minor exposure i.e. when handling small quantities. OTHERWISE: For potentially moderate or heavy exposures: Safety glasses with side shields. NOTE: Contact lenses pose a special hazard; soft lenses may absorb irritants and ALL lenses concentrate them.
Skin protection	See Hand protection below
Hands/feet protection	 No special equipment needed when handling small quantities. OTHERWISE: For potentially moderate exposures: Wear general protective gloves, eg. light weight rubber gloves. For potentially heavy exposures: Wear chemical protective gloves, eg. PVC. and safety footwear.
Body protection	See Other protection below
Other protection	 Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent]

on the same level with locations where direct exposure is likely.

Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave

protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood.

- Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton.
- Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost.

BRETHERICK: Handbook of Reactive Chemical Hazards.

- Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.
- For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).
- Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

No special equipment needed when handling small quantities.

- OTHERWISE:
- Overalls.
- Skin cleansing cream.
- Eyewash unit.
- Do not spray on hot surfaces.

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:

CRC 5040 Engine Start (NZ)

Material	CPI
PE/EVAL/PE	A
BUTYL	С
NATURAL RUBBER	С
NATURAL+NEOPRENE	С
NEOPRENE	С
NEOPRENE/NATURAL	С
NITRILE	С
NITRILE+PVC	С
PVA	С
PVC	С
TEFLON	С
VITON	C
VITON/NEOPRENE	С

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

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

Respiratory protection

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear water white flammable liquid with a under PRESSURE. Contains carbon diox Supplied as an aerosol pack. Contents un	n ethereal odour; partially miscible with water. Su kide propellant. nder PRESSURE . Contains highly flammable hyd	oplied in an aerosol pack. Contents rocarbon propellant.
Physical state	Liquid	Relative density (Water = 1)	0.75
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	<500
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	35	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	-45	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	48.0	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.9	Volatile Component (%vol)	95
Vapour pressure (kPa)	39.9	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	>1	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Diethyl ether has pronounced narcotic / anaesthetic effect and a severe acute inhalation exposure may cause rapid loss of consciousness. If exposure is continued respiratory paralysis and possible death may follow. At 200 ppm, mild nasal irritation occurs, and at 2,000 ppm, dizziness may be experienced. Minor vapour exposure may produce headache, nausea, vomiting. Ether anesthesia can result in vomiting at the end of anesthesia, caused by a direct irritation of the gastric mucosa. Concentrations ranging from 100,000 ppm to 150,000 ppm are required to produce anaesthesia however exposures to these
---------	---

concentrations may be fatal due to respiratory arrest.

Diethyl ether has an irritant action on the mucous membrane of the respiratory tract, it stimulates salivation and increases bronchial secretion; laryngeal spasm may occur. It causes vasodilation which may lead to a severe fall in blood pressure, it reduces blood flow to the kidneys and increases capillary bleeding. The bleeding time is unchanged but the prothrombin time may be prolonged.

Leucocytosis occurs after ether anesthesia and convulsions occasionally occur in children or young adults under deep ether anesthesia. Recovery is slow from prolonged anesthesia and postoperative vomiting commonly occurs. Acute overdosage of ether is characterized by respiratory failure followed by cardiac arrest.

Isolated cases of centrilobular liver necrosis and fatty degeneration of liver lobular are described, but a typical damage of liver or kidney tissue is not reported. Ether anesthesia can result in a metabolic acidosis followed by hyperglycaemia Inhalation hazard is increased at higher temperatures.

High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations, Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. WARNING:Intentional misuse by concentrating/inhaling contents may be lethal.

The symptoms of exposure to high vapour concentrations of benzene include confusion, dizziness, tightening of the leg muscles and pressure over the forehead followed by a period of excitement. If exposure continues, the casualty quickly becomes stupefied and lapses into a coma with narcosis. In non-fatal cases, recovery is usual. Effects of inhalation may include nausea, vomiting, headache, dizziness, drowsiness, weakness, sometimes preceded by brief periods of ataxia, staggering, weak and rapid pulse, chest pain and tightness with breathlessness, pallor, cyanosis of the lips and fingertips and tinnitus. Severe exposures may produce blurred vision, shallow rapid breathing, delirium, cardiac arrhythmias, unconsciousness, deep anaesthesia, paralysis and coma characterised by motor restlessness, tremors and hyperreflexia (occasionally preceded by convulsions). Polyneuritis and persistent nausea, anorexia, muscular weakness, headache, drowsiness, insomnia and agitation may also occur. Two to three weeks after exposure, nervous irritability, breathlessness and unsteady gait may still persist; cardiac distress and unusual discolouration of the skin may be evident for up to four weeks. Haemotoxicity is not usually a feature of acute exposures although anaemia, thrombocytopenia, petechial haemorrhage, and spontaneous internal bleeding have been reported. Fatal exposures may result in asphyxia, central nervous system depression, cardiac and respiratory failure and circulatory collapse; sudden ventricular fibrillation may also be fatal. Death may be sudden or may be delayed for 24 hours. Central nervous system, respiratory or haemorrhagic complications may occur up to five days after exposure and may be lethal; pathological findings include congestion, cerebral oedema, and lung haemorrhage, renal congestion, cerebral oedema and extensive petechial haemorrhage in the brain, pleurae, pericardium, urinary tract, mucous membrane and skin. Exposure to toxic levels has also produced chromosomal damage.

Accidental ingestion of the material may be damaging to the health of the individual.

Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.

Ingestion

Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Spray mist may produce discomfort

Open cuts, abraded or irritated skin should not be exposed to this material

Skin Contact

Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.

Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability.

The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either: produces severe inflammation of the skin in a substantial number of individuals following direct contact, and/or

produces significant and severe inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. NOTE: Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. Limited evidence or practical experience suggests, that the material may cause moderate eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged exposure may cause moderate inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Eve Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures ... Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation. On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence to provide a presumption that human exposure to the material may result in impaired fertility on the basis of: some evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Principal route of occupational exposure to the gas is by inhalation. Diethyl ether is used as a medical anaesthetic. While medically "safe", the hazard is high volatility and high potential of vapour explosion, fire. Abuse of ether by repeated inhalation may lead to ether habit', with symptoms resembling chronic alcoholism. Repeated exposures by workers in industry (often intentional) produced loss of appetite, exhaustion, headache, sleepiness, dizziness, excitation and psychic disturbances. Albuminuria and polycythemia may also result. A degree of tolerance may be acquired 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 Chronic routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding. Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties 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.

Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents.

Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size. Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number

of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1). (1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996

Chronic exposure to benzene may cause headache, fatigue, loss of appetite and lassitude with incipient blood effects including anaemia and blood changes. Benzene is a myelotoxicant known to suppress bone- marrow cell proliferation and to induce haematologic disorders in humans and animals. Signs of benzene-induced aplastic anaemia include suppression of leukocytes (leukopenia), red cells (anaemia), platelets (thrombocytopenia) or all three cell types (pancytopenia). Classic symptoms include weakness, purpura, and haemorrhage. The most significant toxic effect is insidious and often reversible injury to the blood forming tissue. Leukaemia may develop. Occupational exposures have shown a relationship between exposure to benzene and production of myelogenous leukaemia. There may also be a relationship between benzene exposure and the production of lymphoma and multiple myeloma. In chronic exposure, workers exhibit signs of central nervous system lesions and impairment of hearing.

Benzene haemotoxicity and leukaemogenicity involve metabolism, growth factor regulation, oxidative stress, DNA damage, cell regulation, and apoptosis. (Yoon et al Environmental Health Perspectives, 111, pp 1411-1420, 2003) Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

Not Available	Not Available
TOXICITY	IRRITATION
Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Eye (man): 500ppm/1h moderate
Inhalation(Rat) LC50: >4.42 mg/L4h ^[1]	Eye (man): 140ppm/8h mild
Oral (Rat) LD50: >4500 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Skin: adverse effect observed (irritating) ^[1]
TOXICITY	IRRITATION
Dermal (rabbit) LD50: >14280 mg/kg ^[2]	Eye (rabbit): 100 mg - moderate
Inhalation(Rat) LC50: 32000 ppm4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
Oral (Rat) LD50: 1215 mg/kg ^[2]	Skin (rabbit):360 mg (open)-mild
	Skin: no adverse effect observed (not irritating) ^[1]
TOXICITY	IRRITATION
Dermal (rabbit) LD50: 17100 mg/kg ^[1]	Eye (rabbit): 500 mg SEVERE
Inhalation(Rat) LC50: 64000 ppm4h ^[2]	Eye (rabbit):100mg/24hr-moderate
Oral (Rat) LD50: 7060 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
	Skin (rabbit):20 mg/24hr-moderate
	Skin (rabbit):400 mg (open)-mild
	Skin: no adverse effect observed (not irritating) ^[1]
тохісіту	IRRITATION
Not Available	Not Available
TOXICITY	IRRITATION
Not Available	Not Available
	Inhalation(Rat) LC50: >4.42 mg/L4h ^[1] Oral (Rat) LD50: >4500 mg/kg ^[1] TOXICITY Dermal (rabbit) LD50: >14280 mg/kg ^[2] Inhalation(Rat) LC50: 32000 ppm4h ^[1] Oral (Rat) LD50: 1215 mg/kg ^[2] TOXICITY Dermal (rabbit) LD50: 17100 mg/kg ^[1] Inhalation(Rat) LC50: 64000 ppm4h ^[2] Oral (Rat) LD50: 7060 mg/kg ^[2] TOXICITY Not Available TOXICITY Not Available

Oral (unspec) LD50: 500 - 5000 mg/kg [Manufacturer] Substance has been investigated as a tumorigen. Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cycloparaffins.

absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and

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

GASOLINE

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. For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable. WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. 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 DIETHYL ETHER 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. 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 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 MINERAL OIL

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

Continued...

orally. Inhalation

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

CRC 5040 Engine Start (NZ)

	survivability, body weights, organ weights (particularly the liver and thymus), and parameters in exposed animals. Histopathological changes which were treatmen bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on material is less than 30 mg/kg/day. Toxicity to reproduction: Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore this study is =1000 mg/kg/day and no LOAEL was determined. Developmental toxicity, teratogenicity: Heavy paraffinic distillate furfural extract produced maternal, reproductive and fore vaginal discharge (dose-related), body weight decrease, reduction in thymus weig and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg, effects was shown by an increased number of dams with resorptions and intraute developmentally toxic regardless of exposure duration as indicated by increased Furthermore, when exposures were increased to 1000 mg/kg/day and given only palate and ossification delays were observed. Cleft palate was considered to indi The following Oil Industry Note (OIN) has been applied: OIN 8 - The classification	variety of haematology and serum chemistry t-related were most prominent in the adrenals, the results of this study, the NOAEL for the test with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an ore, the reproductive/developmental NOAEL for etal toxicity. Maternal toxicity was exhibited as ght and increase in liver weight (125 mg/kg/day /kg/day). Evidence of potential reproductive orine death. Distillate aromatic extract (DAE) was resorptions and decreased foetal body weights. during gestation days 10 through 12, cleft cate a potential teratogenic effect of DAE. as as a reproductive toxicant category 2; H361d
	(Suspected of damaging the unborn child) and specific target organ toxicant cate through prolonged or repeated exposure) need not apply if the substance is not of Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of intestine is related to carbon chain length; hydrocarbons with smaller chain length with a longer chain length. The majority of an oral dose of mineral hydrocarbon is faeces. Distribution of mineral hydrocarbons following absorption has been obser Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Bas disposition profiles, the data indicate inherent strain differences in the total system F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C different strain sensitivities to the formation of liver granulomas and MLN histiocy.	gory 1; H372 (Causes damage to organs lassified as carcinogenic other lubricant base oils across the small n are more readily absorbed than hydrocarbons not absorbed and is excreted unchanged in the ved in liver, fat, kidney, brain and spleen. sed on the pharmacokinetic parameters and nic exposure (~4 fold greater systemic dose in 26H52, which may be associated with the tosis.
GASOLINE & ETHANOL	(Suspected of damaging the unborn child) and specific target organ toxicant cate through prolonged or repeated exposure) need not apply if the substance is not of Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of intestine is related to carbon chain length; hydrocarbons with smaller chain length with a longer chain length. The majority of an oral dose of mineral hydrocarbon is faeces. Distribution of mineral hydrocarbons occurs via the faeces and urine. Bas disposition profiles, the data indicate inherent strain differences in the total system F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C different strain sensitivities to the formation of liver granulomas and MLN histiccy. The material may cause skin irritation after prolonged or repeated exposure and rates form of dermatitis is often characterised by skin redness (erythema) and swe intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of	gory 1; H372 (Causes damage to organs lassified as carcinogenic other lubricant base oils across the small a are more readily absorbed than hydrocarbons not absorbed and is excreted unchanged in the ved in liver, fat, kidney, brain and spleen. sed on the pharmacokinetic parameters and nic exposure (~4 fold greater systemic dose in 26H52, which may be associated with the tosis. may produce a contact dermatitis (nonallergic). elling the epidermis. Histologically there may be the epidermis.
GASOLINE & ETHANOL Acute Toxicity	(Suspected of damaging the unborn child) and specific target organ toxicant cate through prolonged or repeated exposure) need not apply if the substance is not of Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of intestine is related to carbon chain length; hydrocarbons with smaller chain length with a longer chain length. The majority of an oral dose of mineral hydrocarbon is faeces. Distribution of mineral hydrocarbons following absorption has been obser Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Bas disposition profiles, the data indicate inherent strain differences in the total syster F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C different strain sensitivities to the formation of liver granulomas and MLN histiocyl The material may cause skin irritation after prolonged or repeated exposure and n This form of dermatitis is often characterised by skin redness (erythema) and swe intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of	gory 1; H372 (Causes damage to organs lassified as carcinogenic other lubricant base oils across the small in are more readily absorbed than hydrocarbons not absorbed and is excreted unchanged in the ved in liver, fat, kidney, brain and spleen. sed on the pharmacokinetic parameters and nic exposure (~4 fold greater systemic dose in 26H52, which may be associated with the tosis. may produce a contact dermatitis (nonallergic). elling the epidermis. Histologically there may be the epidermis.
GASOLINE & ETHANOL Acute Toxicity Skin Irritation/Corrosion	(Suspected of damaging the unborn child) and specific target organ toxicant cate through prolonged or repeated exposure) need not apply if the substance is not of Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of intestine is related to carbon chain length; hydrocarbons with smaller chain length with a longer chain length. The majority of an oral dose of mineral hydrocarbon is faeces. Distribution of mineral hydrocarbons following absorption has been obser Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Bas disposition profiles, the data indicate inherent strain differences in the total syster F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C different strain sensitivities to the formation of liver granulomas and MLN histiocyt The material may cause skin irritation after prolonged or repeated exposure and r This form of dermatitis is often characterised by skin redness (erythema) and swe intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of X Carcinogenicity X Reproductivity	gory 1; H372 (Causes damage to organs lassified as carcinogenic other lubricant base oils across the small n are more readily absorbed than hydrocarbons not absorbed and is excreted unchanged in the ved in liver, fat, kidney, brain and spleen. sed on the pharmacokinetic parameters and nic exposure (~4 fold greater systemic dose in 26H52, which may be associated with the tosis. may produce a contact dermatitis (nonallergic). elling the epidermis. Histologically there may be the epidermis.
GASOLINE & ETHANOL Acute Toxicity Skin Irritation/Corrosion Serious Eye Damage/Irritation	(Suspected of damaging the unborn child) and specific target organ toxicant cate through prolonged or repeated exposure) need not apply if the substance is not or Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of intestine is related to carbon chain length; hydrocarbons with smaller chain length with a longer chain length. The majority of an oral dose of mineral hydrocarbon is faeces. Distribution of mineral hydrocarbons occurs via the faeces and urine. Bas disposition profiles, the data indicate inherent strain differences in the total syster F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C different strain sensitivities to the formation of liver granulomas and MLN histiocyl The material may cause skin irritation after prolonged or repeated exposure and r This form of dermatitis is often characterised by skin redness (erythema) and swe intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the spongy layer (spongiosis) and intracellular oedema of the spongy layer (Spongiosis) and intracellular oedema of STOT - Single Exposure	gory 1; H372 (Causes damage to organs lassified as carcinogenic other lubricant base oils across the small in are more readily absorbed than hydrocarbons not absorbed and is excreted unchanged in the ved in liver, fat, kidney, brain and spleen. sed on the pharmacokinetic parameters and nic exposure (~4 fold greater systemic dose in 26H52, which may be associated with the tosis. may produce a contact dermatitis (nonallergic). elling the epidermis. Histologically there may be the epidermis.

SECTION 12 Ecological information

Mutagenicity X

CBC 5040 Engine Start	Endpoint	Test Duration (hr)	Species	Value	Source
(NZ)	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	6.5mg/l	1
gasoline	EC50	96h	Algae or other aquatic plants	64mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	<0.1mg/l	1
	Endpoint	Test Duration (hr)	Species	Value	Source
Contract Street	LC50	96h	Fish	2560mg/l	4
diethyl ether	NOEC(ECx)	504h	Crustacea	100mg/l	2
1 1 2 3 4	BCF	1008h	Fish	0.9-1.4	7

Legend:

Aspiration Hazard X

🛹 – Data available to make classification

🗙 – Data either not available or does not fill the criteria for classification

	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	1378.63m	ıg/L 5
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	275mg/l	2
	EC50	48h	Crustacea	2mg/l	4
ethanol	EC50	96h	Algae or other aquatic plants	<0.001m	ig/L 4
	LC50	96h	Fish	42mg/l	4
	EC50(ECx)	96h	Algae or other aquatic plants	<0.001m	ıg/L 4
	Endpoint	Test Duration (hr)	Species	Value	Source
mineral oil	Not Available	Not Available	Not Available	Not Availat	Not le Available
	Endpoint	Test Duration (hr)	Species	Val	ue Source
carbon dioxide	LC50	96h	Fish	35r	ng/l 1
Legend:	Extracted from 4. US EPA, Ec Bioconcentrati	n 1. IUCLID Toxicity Data 2. Europ cotox database - Aquatic Toxicity I con Data 7. METI (Japan) - Biocor	ne ECHA Registered Substances - Ecotoxicologi Data 5. ECETOC Aquatic Hazard Assessment D Incentration Data 8. Vendor Data	ical Information - Pata 6. NITE (Jap	Aquatic Toxicity an) -

Most ethers are very resistant to hydrolysis, and the rate of cleavage of the carbon-oxygen bond by abiotic processes is expected to be insignificant. Direct photolysis will not be an important removal process since aliphatic ethers do not absorb light at wavelengths >290 nm

For hydrocarbons: Environmental fate:

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

Some hydrocarbon will become associated with benthic sediments, and it is likely to be spread over a fairly wide area of sea floor. Marine sediments may be either aerobic or anaerobic. The material, in probability, is biodegradable, under aerobic conditions (isomerised olefins and alkenes show variable results). Evidence also suggests that the hydrocarbons may be degradable under anaerobic conditions although such degradation in benthic sediments may be a relatively slow process. Under aerobic conditions hydrocarbons degrade to water and carbon dioxide, while under anaerobic processes they produce water, methane and carbon dioxide. Alkenes have low log octanol/water partition coefficients (Kow) of about 1 and estimated bioconcentration factors (BCF) of about 10; aromatics have intermediate values (log Kow values of 2-3 and BCF values of 20-200), while C5 and greater alkanes have fairly high values (log Kow values of about 3-4.5 and BCF values of 100-1.500

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

Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds. Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media. The final products of microbial degradation are carbon dioxide, water, and microbial biomass. The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics, and the aromatics in the C10-C22 range are the most readily biodegradable; n-alkanes, n-alkyl aromatics, and aromatics in the C5-C9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilisation and thus are unavailable in most environments; n-alkanes in the C1-C4 ranges are biodegradable only by a narrow range of specialised hydrocarbon degraders; and n-alkanes, n-alkyl aromatics, and aromatics above C22 are generally not available to degrading microorganisms. Hydrocarbons with condensed ring structures, such as PAHs with four or more rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is, greater than 7.

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

Atmospheric fate: Alkanes, isoalkanes, and cycloalkanes have half-lives on the order of 1-10 days, whereas alkenes, cycloalkenes, and substituted benzenes have half-lives of 1 day or less. Photochemical oxidation products include aldehydes, hydroxy compounds, nitro compounds, and peroxyacyl nitrates. Alkenes, certain substituted aromatics, and naphthalene are potentially susceptible to direct photolysis.

Ecotoxicity:

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

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

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

Page 16 of 20

CRC 5040 Engine Start (NZ)

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	
diethyl ether	LOW	LOW	
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)	
carbon dioxide	LOW	LOW	

Bioaccumulative potential

Ingredient	Bioaccumulation
diethyl ether	LOW (BCF = 9.1)
ethanol	LOW (LogKOW = -0.31)
carbon dioxide	LOW (LogKOW = 0.83)

Mobility in soil

Ingredient	Mobility
diethyl ether	LOW (KOC = 4.395)
ethanol	HIGH (KOC = 1)
carbon dioxide	HIGH (KOC = 1.498)

SECTION 13 Disposal considerations

	• 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 sever may be subject to local laws and regulations and these should be considered first.
Desident / Desident	 Where in doubt contact the responsible authority.
Product / Packaging	Consult State Land Waste Management Authority for disposal.
disposal	 Discharge contents of damaged aerosol cans at an approved site.
	 Allow small quantities to evaporate.
	 DO NOT incinerate or puncture aerosol cans.
	Bury residues and emptied aerosol cans at an approved site.

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.

DO NOT deposit the hazardous substance into or onto a landfill or a sewage facility.

Burning the hazardous substance must happen under controlled conditions with no person or place exposed to

(1) a blast overpressure of more than 9 kPa; or

(2) an unsafe level of heat radiation.

The disposed hazardous substance must not come into contact with class 1 or 5 substances.

1

Page 17 of 20

CRC 5040 Engine Start (NZ)

SECTION 14 Transport information

Marine Pollutant		
HAZCHEM	Not Applicable	

Land transport (UN)

14.1. UN number or ID number	1950		
14.2. UN proper shipping name	AEROSOLS		
14.3. Transport hazard class(es)	Class	2.1	
	Subsidiary Hazard	Not Applicable	
14.4. Packing group	Not Applicable		
14.5. Environmental hazard	Environmentally haza	dous	
14.6. Special precautions for user	Special provisions	63; 190; 277; 327; 344; 381	
	Limited quantity	1000ml	

Air transport (ICAO-IATA / DGR)

14.1. UN number	1950			
14.2. UN proper shipping name	Aerosols, flammable (engine starting fluid)			
	ICAO/IATA Class	2.1		
14.3. Transport hazard	ICAO / IATA Subsidiary Hazard	Not Applicable		
0.000(00)	ERG Code	10L		
14.4. Packing group	Not Applicable			
14.5. Environmental hazard	Environmentally hazardous			
	Special provisions		A1 A145 A167 A802	
	Cargo Only Packing Instructions		203	
	Cargo Only Maximum Qty / Pack		150 kg	
14.6. Special precautions for user	Passenger and Cargo Packing Instructions		Forbidden	
	Passenger and Cargo Maximum Qty / Pack		Forbidden	
	Passenger and Cargo Limited Quantity Packing Instructions		Forbidden	
	Passenger and Cargo Limited Ma	aximum Qty / Pack	Forbidden	

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1950			
14.2. UN proper shipping name	AEROSOLS			
14.3. Transport hazard	IMDG Class	2.1		
class(es)	IMDG Subsidiary Hazard	Not Applicable		
14.4. Packing group	Not Applicable			

Page 18 of 20

14.5 Environmental hazard Marine Pollutant

14.6. Special precautions for user	EMS Number	F-D, S-U
	Special provisions	63 190 277 327 344 381 959
	Limited Quantities	1000 ml

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

Not Applicable

14.7.2. Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
gasoline	Not Available
diethyl ether	Not Available
ethanol	Not Available
mineral oil	Not Available
carbon dioxide	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
gasoline	Not Available
diethyl ether	Not Available
ethanol	Not Available
mineral oil	Not Available
carbon dioxide	Not Available

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002517	Aerosols Flammable Carcinogenic Group Standard 2020
HSR002552	Cosmetic Products Group Standard 2020

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

gasoline is found on the following regulatory lists	
Chemical Footprint Project - Chemicals of High Concern List	New Zealand Hazardous Substances and New Organisms (HSNO) Act -
International Agency for Research on Cancer (IARC) - Agents Classified by	Classification of Chemicals
the IARC Monographs	New Zealand Inventory of Chemicals (NZIoC)
International Agency for Research on Cancer (IARC) - Agents Classified by	New Zealand Workplace Exposure Standards (WES)
the IARC Monographs - Group 2B: Possibly carcinogenic to humans	
New Zealand Approved Hazardous Substances with controls	
diethyl ether is found on the following regulatory lists	
New Zealand Approved Hazardous Substances with controls	New Zealand Inventory of Chemicals (NZIoC)
New Zealand Hazardous Substances and New Organisms (HSNO) Act -	New Zealand Workplace Exposure Standards (WES)
Classification of Chemicals	
New Zealand Hazardous Substances and New Organisms (HSNO) Act -	
Classification of Chemicals - Classification Data	
ethanol is found on the following regulatory lists	
New Zealand Approved Hazardous Substances with controls	New Zealand Inventory of Chemicals (NZIoC)
New Zealand Hazardous Substances and New Organisms (HSNO) Act -	New Zealand Workplace Exposure Standards (WES)
Classification of Chemicals	
New Zealand Hazardous Substances and New Organisms (HSNO) Act -	
Classification of Chemicals - Classification Data	

4					
Chemwatch: 4546-42		Page 19	of 20	Issue Date: 31/05/2023	
Version No: 11.1		CRC 5040 Engine Start (NZ)		Print Date: 24/10/2023	
Chemical Footprint Project -	Chemicals of High Conc	ern List	New Zealand Approved Hazardous Su	ibstances with controls	
International Agency for Resetted the IARC Monographs	earch on Cancer (IARC)	- Agents Classified by	New Zealand Hazardous Substances a Classification of Chemicals	and New Organisms (HSNO) Act -	
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans			New Zealand Workplace Exposure Standards (WES)		
International Agency for Reset the IARC Monographs - Not (earch on Cancer (IARC) Classified as Carcinoger	- Agents Classified by nic			
carbon dioxide is found on	the following regulato	ory lists			
FEI Equine Prohibited Substa	ances List - Controlled M	ledication	New Zealand Hazardous Substances a	and New Organisms (HSNO) Act -	
FEI Equine Prohibited Substa	ances List (EPSL)		Classification of Chemicals - Classifica	ation Data	
New Zealand Hazardous Sub	stances and New Organ	nisms (HSNO) Act -	New Zealand Inventory of Chemicals (NZIOC)	
Classification of Chemicals			New Zealand Workplace Exposure Sta	andards (WES)	

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantity (Closed Containers)	Quantity (Open Containers)
2.1.2A	3 000 L (aggregate water capacity)	3 000 L (aggregate water capacity)

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
2.1.2A				1L (aggregate water capacity)

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status			
Australia - AIIC / Australia Non-Industrial Use	No (mineral oil)			
Canada - DSL	No (mineral oil)			
Canada - NDSL	No (gasoline; diethyl ether; ethanol; mineral oil; carbon dioxide)			
China - IECSC	No (mineral oil)			
Europe - EINEC / ELINCS / NLP	No (mineral oil)			
Japan - ENCS	No (gasoline)			
Korea - KECI	No (mineral oil)			
New Zealand - NZIoC	No (mineral oil)			
Philippines - PICCS	No (mineral oil)			
USA - TSCA	No (mineral oil)			
Taiwan - TCSI	No (mineral oil)			
Mexico - INSQ	No (mineral oil)			
Vietnam - NCI	No (mineral oil)			
Russia - FBEPH	No (mineral oil)			
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.			

Page 20 of 20

SECTION 16 Other information

Revision Date	31/05/2023
Initial Date	12/11/2001

SDS Version Summary

Version	Date of Update	Sections Updated
10.1	10/12/2021	Classification change due to full database hazard calculation/update.
11.1	10/03/2023	Classification change due to full database hazard calculation/update.

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 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
- ES: Exposure Standard
- 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- * AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- * EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- * TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- * FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

This document is copyright.

Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH.

TEL (+61 3) 9572 4700.