Nulon Start Ya Bastard

Nulon Products

Chemwatch Hazard Alert Code: **4**

Chemwatch: **4602-93**
Version No: **8.1.1.1**
Material Safety Data Sheet according to NOHSC and ADG requirements

Issue Date: **15/05/2014**
Print Date: **17/09/2014**
Initial Date: **Not Available**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Nulon Start Ya Bastard
Chemical Name	Not Applicable
Synonyms	SYB150; SYB350
Proper shipping name	AEROSOLS
Chemical formula	Not Applicable
Other means of identification	Not Available
CAS number	Not Applicable

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

Relevant identified uses	The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. Application is by spray atomisation from a hand held aerosol pack Starting aid for automotive and stationary engines.

Details of the manufacturer/importer

Registered company name	Nulon Products
Address	17 Yulong Close Moorebank 2170 NSW Australia
Telephone	+61 2 9608 7800
Fax	+61 2 9601 4700
Website	Not Available
Email	msds@nulon.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	Not Available
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS SUBSTANCE. DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.

CHEMWATCH HAZARD RATINGS

		Min	Max
Flammability	4		
Toxicity	1		
Body Contact	2		
Reactivity	1		
Chronic	2		

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

Poisons Schedule	S5

Risk Phrases [1]	**R36/37**	Irritating to eyes and respiratory system.
	R51/53	Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
	R66	Repeated exposure may cause skin dryness and cracking.
	R44	Risk of explosion if heated under confinement.
	R43	May cause SENSITISATION by skin contact.
	R63(3)	Possible risk of harm to the unborn child.
	R67	Vapours may cause drowsiness and dizziness.
	R40(3)	Limited evidence of a carcinogenic effect.
	R68(3)	Possible risk of irreversible effects.
	R12	Extremely flammable.
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*	

Relevant risk statements are found in section 2

Indication(s) of danger	F+, N, Xn

SAFETY ADVICE

S09	Keep container in a well ventilated place.
S13	Keep away from food, drink and animal feeding stuffs.
S15	Keep away from heat.
S16	Keep away from sources of ignition. No smoking.
S23	Do not breathe gas/fumes/vapour/spray.
S25	Avoid contact with eyes.
S26	In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.
S29	Do not empty into drains.
S33	Take precautionary measures against static discharges.
S35	This material and its container must be disposed of in a safe way.
S36	Wear suitable protective clothing.
S37	Wear suitable gloves.
S38	In case of insufficient ventilation, wear suitable respiratory equipment.
S39	Wear eye/face protection.
S40	To clean the floor and all objects contaminated by this material, use water and detergent.
S41	In case of fire and/or explosion, DO NOT BREATHE FUMES.
S43	In case of fire use...
S46	If swallowed, seek medical advice immediately and show this container or label.
S51	Use only in well ventilated areas.
S53	Avoid exposure - obtain special instructions before use.
S56	Dispose of this material and its container at hazardous or special waste collection point.
S57	Use appropriate container to avoid environmental contamination.
S61	Avoid release to the environment. Refer to special instructions/Safety data sheets.
S64	If swallowed, rinse mouth with water (only if the person is conscious).

Other hazards

	Inhalation, skin contact and/or ingestion may produce health damage*.
	Cumulative effects may result following exposure*.
	May produce skin discomfort*.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64742-89-8.	30-60	naphtha petroleum, light aliphatic solvent
115-10-6	30-60	dimethyl ether
60-29-7	10-30	diethyl ether
123-31-9	<10	hydroquinone
	NotSpec.	ingredients not contributing to the classification
	balance	ingredients not contributing to the classification

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 l/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 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 dioxide (CO2), other pyrolysis products typical of burning organic material**Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions.May emit clouds of acrid smoke

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	- Clean up all spills immediately. - Avoid breathing vapours and contact with skin and eyes. - Wear protective clothing, impervious gloves and safety glasses. - Shut off all possible sources of ignition 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.
Major Spills	- 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.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

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. ▸ Avoid contact with incompatible materials. ▸ 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 MSDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	4aer ▸ 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 MSDS.

Conditions for safe storage, including any incompatibilities

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

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

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	dimethyl ether	Dimethyl ether	760 mg/m3 / 400 ppm	950 mg/m3 / 500 ppm	Not Available	Not Available
Australia Exposure Standards	diethyl ether	Ethyl ether	1210 mg/m3 / 400 ppm	1520 mg/m3 / 500 ppm	Not Available	Not Available
Australia Exposure Standards	hydroquinone	Hydroquinone	2 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	TEEL-0	TEEL-1	TEEL-2	TEEL-3
Nulon Start Ya Bastard	Not Available	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH

naphtha petroleum, light aliphatic solvent	Not Available	Not Available
dimethyl ether	Not Available	Not Available
diethyl ether	19,000 [LEL] ppm	1,900 [LEL] ppm
hydroquinone	Unknown mg/m3 / Unknown ppm	50 mg/m3

MATERIAL DATA

Odour threshold: 0.25 ppm.
The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m3 is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.
Odour Safety Factor (OSF)
OSF=0.042 (gasoline)
The recommended TLV-TWA for hydroquinone takes into account the toxicology of hydroquinone and experience of industrial exposures to benzenediols. Exposure at or below the limit is thought to minimise the risk to workers of eye injury, dermatitis and central nervous system effects. A short-term duration exposure value has not been recommended, because no quantitative data as to the levels of hydroquinone which produce eye irritation or more serious corneal changes has been identified.

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene. Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex I.
European Union (EU) List of Dangerous Substances (Annex I) - up to the 29th ATP

Exposure controls

Appropriate engineering controls

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

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.
Provide adequate ventilation in warehouse or closed storage areas.
Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Speed:
aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s
direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should

	▸ include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. ▸ 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	▸ 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.
Thermal hazards	Not Available

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:
Nulon Start Ya Bastard

Material	CPI
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
PVC	C
TEFLON	C
VITON	C
VITON/NEOPRENE	C
##diethyl	ether

* 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. -

Respiratory protection

Type AX-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	Air-line*	AX-2 P2	AX-PAPR-2 P2 ^
up to 20 x ES	-	AX-3 P2	-
20+ x ES	-	Air-line**	-

* - Continuous-flow; ** - Continuous-flow or positive pressure demand
^ - 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)

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	22aereth Clear, colourless, flammable liquid with an aromatic odour; not miscible with water.

Physical state	Liquid	**Relative density (Water = 1)**	0.71
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	285 (hydroquinone)
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	280 (initial)	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	-45 (diethyl ether)	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Flammable.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	7.5	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1	**Volatile Component (%vol)**	100
Vapour pressure (kPa)	427 (dimethyl ether)	**Gas group**	Not Available
Solubility in water (g/L)	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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial 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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. 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. Ethers produce narcosis following inhalation. Inhalation of lower alkyl ethers may result in central nervous system depression or stimulation, intoxication, headache, dizziness, weakness, blurred vision, seizures and possible coma. Cardiovascular involvement may produce hypotension, bradycardia and cardiovascular collapse, whilst respiratory symptoms might include irritation of nose and throat, cough, laryngeal spasm, pharyngitis, irregular respiration, depression, pulmonary oedema and respiratory arrest. Nausea, vomiting and salivation might also indicate overexposure. Convulsions, respiratory distress or paralysis, asphyxia, pneumonitis, and unconsciousness are all serious manifestations of poisoning. Fatalities have been reported. Kidney and liver damage with interstitial cystitis may result from massive exposures. 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. **WARNING**:Intentional misuse by concentrating/inhaling contents may be lethal.
Ingestion	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 of alkyl ethers may produce symptoms similar to those produced following inhalation.
Skin Contact	Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Spray mist may produce discomfort Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression. Open cuts, abraded or irritated skin should not be exposed to this material The material may accentuate any pre-existing dermatitis condition Following exposure to hydroquinone contact dermatitis has been reported (NIOSHTIC); toxic effects by skin absorption have also been reported (Konica). Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Eye	Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures.. Eye contact with alkyl ethers (vapours or liquid) may produce irritation, redness and lachrymation. 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. Acute exposures to high hydroquinone dust/ vapour concentrations produce irritation, photophobia, lachrymation and corneal 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.

Chronic	On the basis, primarily, of animal experiments, concern has been expressed 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. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Principal route of occupational exposure to the gas is by inhalation. 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. Chronic exposure to alkyl ethers may result in loss of appetite, excessive thirst, fatigue, and weight loss Characteristic eye injuries are common amongst workers exposed during the manufacture of hydroquinone. The injuries develop over a number of years and consist of corneal changes in the curvature of the lens long after exposure has ceased after staining and pigmentation of the cornea has disappeared. Quinone, an agent used in the manufacture of hydroquinone, is thought to be the causative agent while hydroquinone dusts are thought to contribute to the injury. Dermal exposure results from use of cosmetic skin lighteners containing hydroquinone. EEC countries have restricted content to 2% or less. USFDA proposes 1.5 to 2.0% as limits. Some countries allow more. Mice implanted with hydroquinone-containing pellets showed a significant increase in the incidence of urinary bladder carcinomas. Skin painting studies failed to confirm the role of hydroquinone as a tumour-initiator. A further study concluded that hydroquinone had a weak inhibitory action on the carcinogenicity of topical benz[a]pyrene in mouse skin. When rats and mice received hydroquinone by gavage in drinking water in a two-year study, high dose male rats showed greater relative kidney weight and increased severity of nephropathy whilst high dose female rats showed decreased haematological indices. Relative liver weights were increased for dosed males and high dose-female mice. Thyroid gland follicular cell hyperplasia was observed in male and female mice whilst hepatic proliferative lesions were seen in male mice. Renal tubular cell adenomas were seen in male rats. Mononuclear cell leukaemia was seen in female rats. The incidence of hepatocellular neoplasms (primarily adenomas) was increased in dosed female mice. Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

	TOXICITY	IRRITATION
Nulon Start Ya Bastard	Not Available	Not Available

	TOXICITY	IRRITATION
naphtha petroleum, light aliphatic solvent	Not Available	Not Available

	TOXICITY	IRRITATION
dimethyl ether	Inhalation (rat) LC50: 308000 mg/m3	
	Not Available	Not Available

	TOXICITY	IRRITATION
diethyl ether	Inhalation (human) TDLo: 200 ppm.	Eye (rabbit): 100 mg - moderate
	Inhalation (rat) LC50: 73000 ppm/2 h	Skin (rabbit):360 mg (open)-mild
	Oral (man) LDLo: 260 mg/kg.	
	Oral (rat) LD50: 1215 mg/kg.	
	Not Available	Not Available

	TOXICITY	IRRITATION
hydroquinone		

	Oral (human) LDLo: 29 mg/kg	Skin (human): 2% - mild
	Oral (human) TDLo: 170 mg/kg	Skin (human): 5% - SEVERE
	Oral (rat) LD50: 320 mg/kg	
	Not Available	Not Available

** Value obtained from manufacturer's msds*
unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances

Nulon Start Ya Bastard

The following information refers to contact allergens as a group and may not be specific to this product.
Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
for petroleum:
This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.
This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.
This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents
Carcinogenicity: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.
Mutagenicity: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.
Reproductive Toxicity: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.
Human Effects: Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.

Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.
The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.
Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
Hydroquinone is rapidly and extensively absorbed from the gut and lungs of animals. Absorption via the skin is slow but may be accelerated with vehicles such as alcohols. Hydroquinone distributes rapidly and widely among tissues. It is metabolized to 1,4-benzoquinone and other oxidised products, and is detoxified by conjugation to monoglucuronide, monosulfate, and mercapturic derivatives. The excretion of hydroquinone and its metabolites is rapid, and occurs primarily via the urine.
Hydroquinone exhibits moderate acute oral toxicity for animals. Limited data suggest that powdered hydroquinone causes transient eye irritation and corneal opacity in dogs and guinea-pigs; in rabbits powdered hydroquinone induced slight irritation of the eye. Hydroquinone may be a skin sensitiser in animals. The ability to induce sensitization has been found to vary from "weak'' to "strong'' depending on the test procedure and vehicle used.
Repeated oral dosing caused tremors and reduced activity .>=64 mg/kg), reduced body weight gain (>=200 mg/kg), convulsions (..400 mg/kg), and nephropathy in F-344 rats (>=100 mg/kg). No adverse effects on the kidneys were reported in Sprague-Dawley rats treated for the same length of time with the same dose levels. Effects in mice include

	tremors and convulsions (400 mg/kg), increased liver weight (>=25 mg/kg), and irritation of the forestomach (>=200 mg/kg). A functional observational battery and neuropathological examinations of rats failed to give any evidence of persistent or structural neurotoxicity after repeated dosing for 90 days. A NOEL for all effects was 20 mg/kg per day. Fourteen days of repeated dermal dosing caused reduced body weights of male rats at the 3840 mg/kg dose level (6% relative to the controls), but the body weights of female rats at this dose level and of mice at 4800 mg/kg were comparable to controls. There were no clinical signs of toxicity in either species. Prolonged dermal dosing over 13 weeks with 2.0, 3.5, or 5.0% hydroquinone in an oil-in-water emulsion cream resulted in minimal to minor dermal irritation, but no overt toxicity. No adverse effects or compound-related effects occurred in organ weight, clinical pathology, or histopathology. A NOEL was not determined because of the dermal irritation in all treated groups, but the NOAEL was the highest dose level of 5% hydroquinone (74 mg/kg in males and 110 mg/kg in females) based on the lack of systemic effects. **Reproductive effects:** A two-generation reproduction study was conducted in rats. The NOAEL for reproductive effects through two generations was 150 mg/kg per day (the highest dose tested). **Genetic toxicity:** Numerous genotoxicity studies of hydroquinone have been conducted. Hydroquinone is not mutagenic in the *Salmonella*/microsome test. Other data indicate that hydroquinone induces structural chromosome aberrations and c-mitotic effects *in vivo* in mouse bone-marrow cells following ip injection. *In vitro* studies with various cell lines showed that hydroquinone was capable of inducing gene mutations, structural chromosome aberrations, sister-chromatid exchange, and DNA damage. Hydroquinone produces adducts with DNA *in vitro*, but recent *in vivo* studies were unable to produce DNA adducts. While several experiments with hydroquinone have shown mutagenic effects; the relevance of these results to human risk is uncertain . The majority of positive mutagenicity studies use routes of exposure (parenteral or *in vitro*) which are not relevant to human exposures. A dominant lethal assay in rats was negative. **Carcinogenicity:** Sprague-Dawley rats treated for two-years with hydroquinone in the diet showed "atrophy of the liver cord cells, lymphoid tissue of the spleen, adipose tissue, and striated muscle together with superficial ulceration and hemorrhage of the stomach mucosa" but no carcinogenesis. Two-year studies performed by the NTP reported that hydroquinone exposure was associated with some evidence of carcinogenicity in F-344 rats and B6C3F1 mice. In the NTP study, renal tubular cell adenomas occurred in male rats and mononuclear cell leukemia in female rats, and hepatocellular neoplasms, mainly adenomas, in female mice. The NTP concluded that these data indicated "some evidence of carcinogenic activity" in male and female rats and in female mice. In an another study using F-344 rats and B6C3F1 mice, renal tubular cell adenomas were also noted in male rats; hepatocellular adenomas and renal cell hyperplasia were noted in male mice; and hyperplasia of the forestomach was noted in both male and female mice fed 0.8% hydroquinone diets for two years. The evidence provided by cancer bioassay studies is considered limited A U.S.E.P.A. review of the NTP bioassay found the bioassay results provide limited evidence of carcinogenicity in animals. **Mechanisms:** Covalent binding and oxidative stress are mechanisms postulated to be associated with hydroquinone-induced toxicity. Oxidised hydroquinone metabolites may covalently bind cellular macromolecules or alkylate low molecular weight nucleophiles (e.g., glutathione (GSH)) resulting in enzyme inhibition, alterations in nucleic acids and oxidative stress; however, redox cycling is not likely to contribute significantly to oxidative stress. The reaction of hydroquinone metabolites with GSH results in the formation of conjugates which can be further processed to cysteine conjugates which are postulated to cause kidney toxicity Cell proliferation associated nephrotoxicity in a sensitive strain and species of animal (male F344 rat) has been postulated to be involved in the production of renal tumors in rats. **Interaction with Phenols:** A number of studies reporting interactive effects between hydroquinone and other phenolic compounds. Coadministration of hydroquinone and phenol (75 mg/kg), when given by intraperitoneal injection twice per day, produced a synergistic decrease in bone marrow cellularity in B6C3F1 mice that was similar to that induced by benzene. This compound treatment was significantly more myelotoxic than that observed when either hydroquinone or phenol was administered separately. Associated *in vitro* studies suggested that this interactive effect was due to a phenol-induced stimulation of the myeloperoxidase-mediated conversion of hydroquinone to 1,4-benzoquinone in the bone marrow. Subsequent studies have indicated that interactions between hydroquinone and other phenolic compounds can result in a variety of cytotoxic, immunotoxic and genotoxic effects.
NAPHTHA PETROLEUM, LIGHT ALIPHATIC SOLVENT	**for petroleum:** This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic. This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents **Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans. **Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays. **Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed. **Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.

DIETHYL ETHER	The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
HYDROQUINONE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. Hydroquinone is rapidly and extensively absorbed from the gut and lungs of animals. Absorption via the skin is slow but may be accelerated with vehicles such as alcohols. Hydroquinone distributes rapidly and widely among tissues. It is metabolized to 1,4-benzoquinone and other oxidised products, and is detoxified by conjugation to monoglucuronide, monosulfate, and mercapturic derivatives. The excretion of hydroquinone and its metabolites is rapid, and occurs primarily via the urine. Hydroquinone exhibits moderate acute oral toxicity for animals. Limited data suggest that powdered hydroquinone causes transient eye irritation and corneal opacity in dogs and guinea-pigs; in rabbits powdered hydroquinone induced slight irritation of the eye. Hydroquinone may be a skin sensitiser in animals. The ability to induce sensitization has been found to vary from "weak'' to "strong'' depending on the test procedure and vehicle used. Repeated oral dosing caused tremors and reduced activity .>=64 mg/kg), reduced body weight gain (>=200 mg/kg), convulsions (..400 mg/kg), and nephropathy in F-344 rats (>=100 mg/kg). No adverse effects on the kidneys were reported in Sprague-Dawley rats treated for the same length of time with the same dose levels. Effects in mice include tremors and convulsions (400 mg/kg), increased liver weight (>=25 mg/kg), and irritation of the forestomach (>=200 mg/kg). A functional observational battery and neuropathological examinations of rats failed to give any evidence of persistent or structural neurotoxicity after repeated dosing for 90 days. A NOEL for all effects was 20 mg/kg per day. Fourteen days of repeated dermal dosing caused reduced body weights of male rats at the 3840 mg/kg dose level (6% relative to the controls), but the body weights of female rats at this dose level and of mice at 4800 mg/kg were comparable to controls. There were no clinical signs of toxicity in either species. Prolonged dermal dosing over 13 weeks with 2.0, 3.5, or 5.0% hydroquinone in an oil-in-water emulsion cream resulted in minimal to minor dermal irritation, but no overt toxicity. No adverse effects or compound-related effects occurred in organ weight, clinical pathology, or histopathology. A NOEL was not determined because of the dermal irritation in all treated groups, but the NOAEL was the highest dose level of 5% hydroquinone (74 mg/kg in males and 110 mg/kg in females) based on the lack of systemic effects. **Reproductive effects:** A two-generation reproduction study was conducted in rats. The NOAEL for reproductive effects through two generations was 150 mg/kg per day (the highest dose tested). **Genetic toxicity:** Numerous genotoxicity studies of hydroquinone have been conducted. Hydroquinone is not mutagenic in the *Salmonella*/microsome test. Other data indicate that hydroquinone induces structural chromosome aberrations and c-mitotic effects *in vivo* in mouse bone-marrow cells following ip injection. *In vitro* studies with various cell lines showed that hydroquinone was capable of inducing gene mutations, structural chromosome aberrations, sister-chromatid exchange, and DNA damage. Hydroquinone produces adducts with DNA *in vitro*, but recent *in vivo* studies were unable to produce DNA adducts. While several experiments with hydroquinone have shown mutagenic effects; the relevance of these results to human risk is uncertain . The majority of positive mutagenicity studies use routes of exposure (parenteral or *in vitro*) which are not relevant to human exposures. A dominant lethal assay in rats was negative. **Carcinogenicity:** Sprague-Dawley rats treated for two-years with hydroquinone in the diet showed "atrophy of the liver cord cells, lymphoid tissue of the spleen, adipose tissue, and striated muscle together with superficial ulceration and hemorrhage of the stomach mucosa" but no carcinogenesis. Two-year studies performed by the NTP reported that hydroquinone exposure was associated with some evidence of carcinogenicity in F-344 rats and B6C3F1 mice. In the NTP study, renal tubular cell adenomas occurred in male rats and mononuclear cell leukemia in female rats, and hepatocellular neoplasms, mainly adenomas, in female mice. The NTP concluded that these data indicated "some evidence of carcinogenic activity'' in male and female rats and in female mice. In an another study using F-344 rats and B6C3F1 mice, renal tubular cell adenomas were also noted in male rats; hepatocellular adenomas and renal cell hyperplasia were noted in male mice; and hyperplasia of the forestomach was noted in both male and female mice fed 0.8% hydroquinone diets for two years. The evidence provided by cancer bioassay studies is considered limited A U.S.E.P.A. review of the NTP bioassay found the bioassay results provide limited evidence of carcinogenicity in animals. **Mechanisms:** Covalent binding and oxidative stress are mechanisms postulated to be associated with hydroquinone-induced toxicity. Oxidised hydroquinone metabolites may covalently bind cellular macromolecules or alkylate low molecular weight nucleophiles (e.g., glutathione (GSH)) resulting in enzyme inhibition, alterations in nucleic acids and oxidative stress; however, redox cycling is not likely to contribute significantly to oxidative stress. The reaction of hydroquinone metabolites with GSH results in the formation of conjugates which can be further processed to cysteine conjugates which are postulated to cause kidney toxicity Cell proliferation associated nephrotoxicity in a sensitive strain and species of animal (male F344 rat) has been postulated to be involved in the production of renal tumors in rats. **Interaction with Phenols:** A number of studies reporting interactive effects between hydroquinone and other phenolic compounds. Coadministration of hydroquinone and phenol (75 mg/kg), when given by intraperitoneal injection twice per day, produced a synergistic decrease in bone marrow cellularity in B6C3F1 mice that was similar to that induced by benzene. This compound

	treatment was significantly more myelotoxic than that observed when either hydroquinone or phenol was administered separately. Associated *in vitro* studies suggested that this interactive effect was due to a phenol-induced stimulation of the myeloperoxidase-mediated conversion of hydroquinone to 1,4-benzoquinone in the bone marrow. Subsequent studies have indicated that interactions between hydroquinone and other phenolic compounds can result in a variety of cytotoxic, immunotoxic and genotoxic effects. 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.

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 required to make classification available*
✘ *– Data available but does not fill the criteria for classification*
⊘ *– Data Not Available to make classification*

CMR STATUS

Not Applicable

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
naphtha petroleum, light aliphatic solvent	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
dimethyl ether	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
diethyl ether	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
hydroquinone	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

Toxic to aquatic organisms.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
May cause long-term adverse effects in the aquatic environment.
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 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.).

Hydroquinone is a naturally occurring substance found in several foods (e.g., wheat products, fruits) and beverages (e.g., brewed coffee, some teas, beer, red wine). Hydroquinone is formed as a byproduct of metabolism in several bacteria and marine species. It is estimated that approximately 5 x 104 kg of hydroquinone is generated per year during cigarette smoking.

Hydroquinone is considered to be readily biodegradable and photodegradable.

The aquatic toxicity of hydroquinone to fresh water fish, Daphnia, and algae was between 0.050-0.335 mg/l; the predicted chronic values for these fresh water taxa were calculated to be < 0.100.

The 84 hr LC50 for the salt water shrimp, *C. septemspinosa*, was selected as the only salt water species for analysis. Based on these data and on the predicted aquatic toxicity values, the USEPA identified concern concentrations or predicted no effect concentrations (PNECs) at 1.0 ug/l for fresh water species and 8.0 ug/l for salt water species. Alternatively, a PNEC can be derived using the assessment factors recommended in the SIDS Manual. As only acute effect data for fish and daphnids are available, an assessment factor of 100-1000 would be appropriate. Due to the large available database, a factor of 100 would be acceptable. Applied to the lowest experimental value of 0.044 mg/l (fathead minnow), a PNEC of 0.44 ug/l can be derived.

Physicochemical parameters:

Water solubility: 73 g/l (25 C)
log Kow: 0.50-0.61
Vapour pressure: 2.34 x 10-3 Pa (25 C)
BOD5: 1.0 g O2/g
COD5: 1.83 gO2/g
BOD5/COD5: 0.55

Degradation: Hydroquinone degrades by both biotic and abiotic mechanisms. Biodegradation is affected by pH, temperature, aerobic/anaerobic conditions, and acclimation of the microorganisms involved. Under aerobic conditions 74% of the radioactivity from the incubation of activated sludge and 14C- hydroquinone was recovered as carbon dioxide in 5-10 days. Small amounts of 1,4-benzoquinone, 2-hydroxy-1,4-benzoquinone, and beta-ketoadipic acid are formed as metabolites of hydroquinone. A maximum concentration of 0.11% (1.05 mg/L) 1,4-benzoquinone was detected at 2 hr during incubation of 950 mg/L hydroquinone by yeast cultures. At later time points 1,4-benzoquinone levels were lower and benzoquinone was not detected in the effluent from the activated sludge unit. In another study 82% of hydroquinone was converted to CO2 in a 28-day Sturm test ; 97% of the dissolved organic carbon was removed in 28 days. Thus hydroquinone is primarily converted to CO2 or mineralised during aerobic degradation.

Under anaerobic conditions, hydroquinone is metabolized through phenol, instead of 1,4-benzoquinone, prior to mineralisation.

As the organisms which biodegrade hydroquinone are widely distributed in the environment in sludges, soils, sediments, and composts, hydroquinone is expected to readily biodegrade in soils and water.

Photolysis: Due to its intrinsic properties, hydroquinone is relatively rapidly photodegraded; phototransformations may occur from direct excitation or from induced or photocatalytic reactions.

Bioaccumulation: With measured partition coefficients log Pow = 0.50-0.61, hydroquinone is not considered to undergo bioaccumulation. Bioaccumulation factors of 40 have been determined for algae and fish .

Distribution between environmental compartments and occurrence in the environment: The environmental transport of hydroquinone can be partially predicted based on its physicochemical properties. With a melting point of 169 C, a vapor pressure of 2.34 x 10-3 Pa at 25 C and a relative vapor density of 3.81 (air=1), it is not expected to transport into the atmosphere. A calculation of fugacity using Mackay's model I indicates that hydroquinone will be distributed to the water compartment (99.6%) when released into the environment.

Air: Hydroquinone is essentially non-volatile in its solid form. Its solubility in water (which increases with temperature), low vapor pressure, and high relative vapor density, and low Henry's law constant (3.84x10-11 atm-m-3/mole) indicate that hydroquinone will not evaporate from water into the atmosphere. The half-life of hydroquinone in the air is 14 hr (U.S.E.P.A., 1990).

In its dry solid form, hydroquinone is stable and darkens only slowly if exposed to the air. In the presence of moisture and ambient levels of oxygen, hydroquinone can undergo oxidation to 1,4-benzoquinone which is more likely to volatilise because of its higher vapor pressure. As this potential reaction is well recognised, manufacturing plants do not let hydroquinone powders stand in open environments prior to bagging or drumming operations. For the same reason, hydroquinone-containing products such as photographic developers contain stabilizers such as sodium sulfite to prevent or retard oxidation.

Water: Due to its physical chemical properties, hydroquinone can be expected to partition to the water compartment. As its melting point is 169 C, vapor pressure is low, Henry's law constant is relatively low, and its solubility in water increases with temperature, hydroquinone is not likely to be volatilised to the air compartment from water. In waste water, hydroquinone would be expected to be readily biodegradable. If hydroquinone were present in an open body of water, it would be expected to both biodegrade and photodegrade. Hydroquinone half-life in surface water is 20 hr. While 1,4-benzoquinone would be expected to be one of the degradation products of hydroquinone, its ready degradation would not be expected to impact the toxicity of a hydroquinone release.

Soil: Hydroquinone released to the soil would be expected to mineralize as organisms which can degrade hydroquinone are commonly found in soils and compost. Half-life in soil is 2-14 days and depends on photo-oxidation and bacterial degradation. Hydroquinone present in soil could be expected to partition to water in the soil and be mobile. Half-life values in ground water are 4-14 days (aerobic conditions) and up to a month (anaerobic conditions).

However, hydroquinone and its immediate degradation product, 1,4-benzoquinone may also be absorbed to the soil. Since hydroquinone and 1,4-benzoquinone are electron donor and electron acceptor molecules respectively, they could form charge transfer complexes with soil particles. Hydroquinone and its biodegradation products may contribute to the formation of humic acids which are polymerisation products of polyphenols commonly formed during the biodegradation of plants. Much of the naturally occurring hydroquinone in plants may be reincorporated into soils in this manner.

Effects on the Environment

Aquatic effects

Fish LC50 (96 h): Pimephales promelas 0.044 mg/l

Daphnia magna LC50 (48 h): 0.096 mg/l (interpolated results from several researchers)

Salt water shrimp (Crangon septemspinosa) LC50 (84 h):0.833 mg/l

Algal EC50 (3 d): S. capricormutum 0.355 mg/l

Based on these numbers hydroquinone has a high acute toxicity for aquatic organisms

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
Not Available	Not Available	Not Available

Bioaccumulative potential

Ingredient	Bioaccumulation
Not Available	Not Available

Mobility in soil

Ingredient	Mobility
Not Available	Not Available

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Consult State Land Waste Management Authority for 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.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	
HAZCHEM	Not Applicable

Land transport (ADG)

UN number	1950
Packing group	Not Applicable
UN proper shipping name	AEROSOLS
Environmental hazard	No relevant data
Transport hazard class(es)	Class: 2.1 Subrisk: Not Applicable
Special precautions for user	Special provisions: 63 190 277 327 344 Limited quantity: See SP 277

Air transport (ICAO-IATA / DGR)

UN number	1950
Packing group	Not Applicable
UN proper shipping name	Aerosols, flammable
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class: 2.1 ICAO / IATA Subrisk: Not Applicable ERG Code: 10L
Special precautions for user	Special provisions: A145A167A802 Cargo Only Packing Instructions: 203 Cargo Only Maximum Qty / Pack: 150 kg Passenger and Cargo Packing Instructions: 203 Passenger and Cargo Maximum Qty / Pack: 75 kg Passenger and Cargo Limited Quantity Packing Instructions: Y203 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	1950
Packing group	Not Applicable
UN proper shipping name	AEROSOLS
Environmental hazard	No relevant data
Transport hazard class(es)	IMDG Class: 2.1 IMDG Subrisk: See SP63
Special precautions for user	EMS Number: F-D , S-U Special provisions: 63 190 277 327 344 959 Limited Quantities: See SP277

Inland waterways transport (ADNR / River Rhine): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	diethyl ether	Z

SECTION 15 REGULATORY INFORMATION

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

naphtha petroleum, light aliphatic solvent(64742-89-8.) is found on the following regulatory lists	"Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5","International Council of Chemical Associations (ICCA) - High Production Volume List","International Maritime Dangerous Goods Requirements (IMDG Code)","OSPAR List of Chemicals for Priority Action","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia GHS Hazardous Chemical Information List (Draft)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","OECD List of High Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","OSPAR National List of Candidates for Substitution – Norway","International Society of Automotive Engineers (SAE) Declarable Substances Chemical List - ARP9536","International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","International Chemical Secretariat (ChemSec) SIN List (*Substitute It Now!)","Australia High Volume Industrial Chemical List (HVICL)","OECD Existing Chemicals Database","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","Australia Hazardous Substances Information System - Consolidated Lists","International Air Transport Association (IATA) Dangerous Goods Regulations","Acros Transport Information"
dimethyl ether(115-10-6) is found on the following regulatory lists	"Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5","International Council of Chemical Associations (ICCA) - High Production Volume List","International Maritime Dangerous Goods Requirements (IMDG Code)","Australia Exposure Standards","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia GHS Hazardous Chemical Information List (Draft)","Australia Dangerous Goods Code (ADG Code) - List of

	Emergency Action Codes","Australia Therapeutic Goods Administration (TGA) Substances that may be used in Listed medicines","Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","OECD List of High Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","Australia National Pollutant Inventory","Australia Dangerous Goods Code (ADG Code) - Packing Instruction - Liquefied and Dissolved Gases","Sigma-AldrichTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","Australia Hazardous Substances Information System - Consolidated Lists","International Air Transport Association (IATA) Dangerous Goods Regulations","IMO IBC Code Chapter 17: Summary of minimum requirements"
diethyl ether(60-29-7) is found on the following regulatory lists	"Australia - New South Wales Protection of the Environment Operations (Waste) Regulation 2005 - Waste transported within NSW or interstate and required to be tracked","Australia Illicit Drug Reagents/Essential Chemicals - Category III","International Maritime Dangerous Goods Requirements (IMDG Code)","IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)","International Council of Chemical Associations (ICCA) - High Production Volume List","Australia Customs (Prohibited Exports) Regulations 1958 - Schedule 9 Precursor substances - Part 2","Australia Crimes (Traffic in Narcotic Drugs and Psychotropic Substances) Act - Schedule 1 - United Nations Convention Against Illicit Traffic In Narcotic Drugs And Psychotropic Substances - Table II","United Nations Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances - Table II","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4","Australia GHS Hazardous Chemical Information List (Draft)","Australia Exposure Standards","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions","FisherTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","Australia Therapeutic Goods Administration (TGA) Substances that may be used in Listed medicines","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2","IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances","United Nations Consolidated List of Products Whose Consumption and/or Sale Have Been Banned, Withdrawn, Severely Restricted or Not Approved by Governments","OSPAR National List of Candidates for Substitution – Norway","OECD List of High Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","Australia National Pollutant Inventory","Sigma-AldrichTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","GESAMP/EHS Composite List - GESAMP Hazard Profiles","Australia Hazardous Substances Information System - Consolidated Lists","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","International Air Transport Association (IATA) Dangerous Goods Regulations","Joint FAO/WHO Expert Committee on Food Additives (JECFA) - Compendium of Food Additive Specifications - Extraction solvents","International Fragrance Association (IFRA) Survey: Transparency List","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6","IMO IBC Code Chapter 17: Summary of minimum requirements","United Nations List of Precursors and Chemicals Frequently used in the Illicit Manufacture of Narcotic Drugs and Psychotropic Substances Under International Control (Red List) - Table II"
hydroquinone(123-31-9) is found on the following regulatory lists	"International Maritime Dangerous Goods Requirements (IMDG Code)","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)","IOFI Global Reference List of Chemically Defined Substances","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4","Australia GHS Hazardous Chemical Information List (Draft)","Australia Exposure Standards","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions","FisherTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)","Australia Therapeutic Goods Administration (TGA) Substances that may be used in Listed medicines","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2","United Nations Consolidated List of Products Whose Consumption and/or Sale Have Been Banned, Withdrawn, Severely Restricted or Not Approved by Governments","OECD List of High Production Volume (HPV) Chemicals","Australia Inventory of Chemical Substances (AICS)","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)","OECD Existing Chemicals Database","Sigma-AldrichTransport Information","United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)","Australia Hazardous Substances Information System - Consolidated Lists","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","International Air Transport Association (IATA) Dangerous Goods Regulations","Australia - New South Wales Protection of the Environment Operations (Waste) Regulation 2005 - Characteristics of trackable wastes","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6"

SECTION 16 OTHER INFORMATION

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

The (M)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.