

Moisture Remover FG #703-2565

RS Components

Chemwatch: 4882-63
Version No: 2.1.1.1
Material Safety Data Sheet according to NOHSC and ADG requirements

Chemwatch Hazard Alert Code: 4

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

Product Identifier

Product name	Moisture Remover FG #703-2565
Chemical Name	Not Applicable
Synonyms	Manufacturer's Code: 703-2565
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	Application is by spray atomisation from a hand held aerosol pack , Lubricants.
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Details of the supplier of the safety data sheet

Registered company name	RS Components	RS Components
Address	25 Pavesi Street Smithfield 2164 NSW Australia	Units 30 & 31, 761 Great South Road Penrose 1006 Auckland New Zealand
Telephone	+1 300 656 636	+64 9 526 1600
Fax	+1 300 656 696	+64 9 579 1700
Website	Not Available	www.rsnewzealand.com
Email	Not Available	Not Available

Emergency telephone number

Association / Organisation	Not Available	Not Available
Emergency telephone numbers	1800 039 008 (24 hours),+61 3 9573 3112	Not Available
Other emergency telephone numbers	1800 039 008 (24 hours),+61 3 9573 3112	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.

Label elements



Relevant risk statements are found in section 2

Poisons Schedule	Not Applicable												
Risk Phrases ^[1]	<table><tr><td>R66</td><td>Repeated exposure may cause skin dryness and cracking.</td></tr><tr><td>R44</td><td>Risk of explosion if heated under confinement.</td></tr><tr><td>R38</td><td>Irritating to skin.</td></tr><tr><td>R67</td><td>Vapours may cause drowsiness and dizziness.</td></tr><tr><td>R41</td><td>Risk of serious damage to eyes.</td></tr><tr><td>R12</td><td>Extremely flammable.</td></tr></table>	R66	Repeated exposure may cause skin dryness and cracking.	R44	Risk of explosion if heated under confinement.	R38	Irritating to skin.	R67	Vapours may cause drowsiness and dizziness.	R41	Risk of serious damage to eyes.	R12	Extremely flammable.
R66	Repeated exposure may cause skin dryness and cracking.												
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R38	Irritating to skin.												
R67	Vapours may cause drowsiness and dizziness.												
R41	Risk of serious damage to eyes.												
R12	Extremely flammable.												
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI												
Indication(s) of danger	F+, Xi												

SAFETY ADVICE

S15	Keep away from heat.
S23	Do not breathe gas/fumes/vapour/spray.
S24	Avoid contact with skin.
S25	Avoid contact with eyes.
S26	In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.
S33	Take precautionary measures against static discharges.
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.
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.
S56	Dispose of this material and its container at hazardous or special waste collection point.
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*.
	Possible respiratory and skin sensitizer*.
	Cumulative effects may result following exposure*.
	May produce discomfort of the respiratory system*.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64742-47-8	>60	distillates, petroleum, light, hydrotreated
68608-26-4	1-5	sodium petroleum sulfonate
107-98-2	<0.1	propylene glycol monomethyl ether - alpha isomer
124-38-9	1-5	carbon dioxide

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

Eye Contact	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> ▶ 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	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ Not considered a normal route of entry. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

	Treat symptomatically.
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SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

	<p>SMALL FIRE:</p> <ul style="list-style-type: none"> ▶ Water spray, dry chemical or CO2 <p>LARGE FIRE:</p> <ul style="list-style-type: none"> ▶ Water spray or fog.
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Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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). <p>Combustion products include:</p> <ul style="list-style-type: none"> , carbon monoxide (CO) , carbon dioxide (CO2) , other pyrolysis products typical of burning organic material <p>CARE: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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.
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	<ul style="list-style-type: none"> ▶ 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.
<p style="text-align: center;">Other information</p>	<ul style="list-style-type: none"> ▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can ▶ Store in original containers in approved flammable liquid storage area. ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped. ▶ No smoking, naked lights, heat or ignition sources. ▶ Keep containers securely sealed. Contents under pressure. ▶ Store away from incompatible materials. ▶ Store in a cool, dry, well ventilated area. ▶ Avoid storage at temperatures higher than 40 deg C. ▶ Store in an upright position. ▶ Protect containers against physical damage. ▶ Check regularly for spills and leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this MSDS.

Conditions for safe storage, including any incompatibilities

<p style="text-align: center;">Suitable container</p>	<ul style="list-style-type: none"> ▶ Glass container is suitable for laboratory quantities ▶ Aerosol dispenser. ▶ Check that containers are clearly labelled.
<p style="text-align: center;">Storage incompatibility</p>	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents ▶ Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances

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	distillates, petroleum, light, hydrotreated	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether	369 mg/m3 / 100 ppm	553 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	carbon dioxide	Carbon dioxide / Carbon dioxide in coal mines	22500 mg/m3 / 9000 mg/m3 / 12500 ppm / 5000 ppm	54000 mg/m3 / 30000 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	TEEL-0	TEEL-1	TEEL-2	TEEL-3
propylene glycol monomethyl ether - alpha isomer	100 ppm	150 ppm	300 ppm	750 ppm
carbon dioxide	5000 ppm	30000 ppm	40000 ppm	40000 ppm

Ingredient	Original IDLH	Revised IDLH
distillates, petroleum, light, hydrotreated	Not Available	Not Available
sodium petroleum sulfonate	Not Available	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available	Not Available
carbon dioxide	50,000 ppm	40,000 ppm

Exposure controls

<p style="text-align: center;">Appropriate engineering controls</p>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. 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.</p>
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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

No special equipment for minor exposure i.e. when handling small quantities.

OTHERWISE: For potentially moderate or heavy exposures:

- ▶ Safety glasses with side shields.
- ▶ **NOTE:** Contact lenses pose a special hazard; soft lenses may absorb irritants and **ALL** lenses concentrate them.

Skin protection

See Hand protection below

Hands/feet protection

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

No special equipment needed when handling small quantities.

OTHERWISE:

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

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:

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Material	CPI
BUTYL	A
NEOPRENE	A
PVC	B
NITRILE	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

Respiratory protection

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

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

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

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

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

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

up to 100	10000	-	AE-3
100+			Airline**

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

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Blue-green liquid aerosol with a characteristic odour; emulsifies with water.		
Physical state	Compressed Gas	Relative density (Water = 1)	0.83 @ 20 deg.C
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>200
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	3.9 @ 20 deg.C
Initial boiling point and boiling range (°C)	190-250	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution(1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ 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

	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation hazard is increased at higher temperatures.</p>
Inhaled	<p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C₂-C₁₂) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C₅-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may</p>

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	<p>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.</p> <p>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</p> <p>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Not normally a hazard due to physical form of product.</p> <p>Considered an unlikely route of entry in commercial/industrial environments</p>
Skin Contact	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Spray mist may produce discomfort</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. 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..</p> <p>Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.</p>
Chronic	<p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.</p> <p>Principal route of occupational exposure to the gas is by inhalation.</p> <p>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.</p> <p>Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]</p>

Moisture Remover FG #703-2565	TOXICITY	IRRITATION
	Not Available	Not Available
distillates, petroleum, light, hydrotreated	TOXICITY	IRRITATION
	Not Available	Not Available
sodium petroleum sulfonate	TOXICITY	IRRITATION
	Not Available	Not Available
propylene glycol monomethyl ether - alpha isomer	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 13000 mg/kg	Eye (rabbit) 230 mg mild
	Inhalation (rat) LC50: 10000 ppm/5 h.	Eye (rabbit) 500 mg/24 h. - mild
	Oral (rat) LD50: 3739 mg/kg	Eye (rabbit): 100 mg SEVERE
	Not Available	Skin (rabbit) 500 mg open - mild
carbon dioxide	TOXICITY	IRRITATION
	Not Available	Not Available

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

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for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m³ for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m³. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m³), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating

None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m³ (600 ppm) for PnB and 2,010 mg/m³ (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m³ (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m³ (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m³) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m³). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m³), with decreased body weights occurring at 3000 ppm (11058 mg/m³). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

No significant acute toxicological data identified in literature search.

Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.

The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.

for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates

Most chemicals of this category are not defined substances, but mixtures of homologues with different alkyl chain lengths. Alpha-olefin sulfonates are mixtures of alkene sulfonate and hydroxyl alkane sulfonates with the sulfonate group in the terminal position and the double bond, or hydroxyl group, located at a position in the vicinity of the sulfonate group.

Common physical and/or biological pathways result in structurally similar breakdown products, and are, together with the surfactant properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

Acute toxicity: These substances are well absorbed after ingestion; penetration through the skin is however poor. After absorption, these chemicals are distributed mainly to the liver.

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Acute oral LD50 values of alkyl sulfates in rats and/or mice were (in mg/kg):

C10-; 290-580

C10-16-, and C12-; 1000-2000

C12-14, C12-15, C12-16, C12-18 and C16-18-; >2000

C14-18, C16-18-; >5000

The clinical signs observed were non-specific (piloerection, lethargy, decreased motor activity and respiratory rate, diarrhoea). At necropsy the major findings were irritation of the gastrointestinal tract and anemia of inner organs.

Based on limited data, the acute oral LD50 values of alkane sulfonates and alpha-olefin sulfonates of comparable chain lengths are assumed to be in the same range.

The counter ion does not appear to influence the toxicity in a substantial way.

Acute dermal LD50 values of alkyl sulfates in rabbits (mg/ kg):

C12-; 200

C12-13 and C10-16-; >500

Apart from moderate to severe skin irritation, clinical signs included tremor, tonic-clonic convulsions, respiratory failure, and body weight loss in the study with the C12- alkyl sulfate and decreased body weights after administration of the C10-16- alkyl sulfates. No data are available for alkane sulfonates but due to a comparable metabolism and effect concentrations in long-term studies effect concentrations are expected to be in the same range as found for alkyl sulfates.

There are no data available for acute inhalation toxicity of alkyl sulfates, alkane sulfonates or alpha-olefin sulfonates.

In skin irritation tests using rabbits (aqueous solutions, OECD TG 404):

C8-14 and C8-16 (30%), C12-14 (90%), C14-18 (60%)- corrosive

Under occlusive conditions:

C12, and C12-14 (25%), C12-15-, C13-15 and C15-16 (5-7%) - moderate to strong irritants

Comparative studies investigating skin effects like transepidermal water loss, epidermal electrical conductance, skin swelling, extraction of amino acids and proteins or development of erythema in human volunteers consistently showed a maximum of effects with C12-alkyl sulfate, sodium; this salt is routinely used as a positive internal control giving borderline irritant reactions in skin irritation studies performed on humans. As the most irritant alkyl sulfate it can be concluded that in humans 20% is the threshold concentration for irritative effects of alkyl sulfates in general. No data were available with regard to the skin irritation potential of alkane sulfonates. Based on the similar chemical structure they are assumed to exhibit similar skin irritation properties as alkyl sulfates or alpha-olefin sulfonates of comparable chain lengths.

In eye irritation tests, using rabbits, C12-containing alkyl sulfates (>10% concentration) were severely irritating and produced irreversible corneal effects. With increasing alkyl chain length, the irritating potential decreases, and C16-18 alkyl sulfate sodium, at a concentration of 25%, was only a mild irritant.

Concentrated C14-16- alpha-olefin sulfonates were severely irritating, but caused irreversible effects only if applied as undiluted powder. At concentrations below 10% mild to moderate, reversible effects, were found. No data were available for alkane sulfonates

Alkyl sulfates and C14-18 alpha-olefin sulfonates were not skin sensitizers in animal studies. No reliable data were available for alkane sulfonates. Based on the similar chemical structure, no sensitisation is expected.

However anecdotal evidence suggests that sodium lauryl sulfate causes pulmonary sensitisation resulting in hyperactive airway dysfunction and pulmonary allergy accompanied by fatigue, malaise and aching. Significant symptoms of exposure can persist for more than two years and can be activated by a variety of non-specific environmental stimuli such as an exhaust, perfumes and passive smoking.

Absorbed sulfonates are quickly distributed through living systems and are readily excreted. Toxic effects may result from the effects of binding to proteins and the ability of sulfonates to translocate potassium and nitrate (NO₃-) ions from cellular to interstitial fluids. Airborne sulfonates may be responsible for respiratory allergies and, in some instances, minor dermal allergies. Repeated skin contact with some sulfonated surfactants has produced sensitisation dermatitis in predisposed individuals

Repeat dose toxicity: After repeated oral application of alkyl sulfates with chain lengths between C12 and C18, the liver was the only target organ for systemic toxicity. Adverse effects on this organ included an increase in liver weight, enlargement of liver cells, and elevated levels of liver enzymes. The LOAEL for liver toxicity (parenchymal hypertrophy and an increase in comparative liver weight) was 230 mg/kg/day (in a 13 week study with C16-18 alkyl sulfate, sodium). The lowest NOAEL in rats was 55 mg/kg/day (in a 13 week study with C12-alkyl sulfate, sodium).

C14- and C14-16-alpha-olefin sulfonates produced NOAELs of 100 mg/kg/day (in 6 month- and 2 year studies). A reduction in body weight gain was the only adverse effect identified in these studies.

No data were available with regard to the repeated dose toxicity of alkane sulfonates. Based on the similarity of metabolic pathways between alkane sulfonates, alkyl sulfates and alkyl-olefin sulfonates, the repeated dose toxicity of alkane sulfonates is expected to be similar with NOAEL and LOAEL values in the same range as for alkyl sulfates and alpha-olefin sulfonates, i.e. 100 and 200-250 mg/kg/day, respectively, with the liver as potential target organ.

Genotoxicity: Alkyl sulfates of different chain lengths and with different counter ions were not mutagenic in standard bacterial and mammalian cell systems both in the absence and in the presence of metabolic activation. There was also no indication for a genotoxic potential of alkyl sulfates in various in vivo studies on mice (micronucleus assay, chromosome aberration test, and dominant lethal assay). alpha-Olefin sulfonates were not mutagenic in the Ames test, and did not induce chromosome aberrations in vitro. No genotoxicity data were available for alkane sulfonates. Based on the overall negative results in the genotoxicity assays with alkyl sulfates and alpha-olefin sulfonates, the absence of structural elements indicating mutagenicity, and the overall database on different types of sulfonates, which were all tested negative in mutagenicity assays, a genotoxic potential of alkane sulfonates is not expected.

Carcinogenicity: Alkyl sulfates were not carcinogenic in feeding studies with male and female Wistar rats fed diets with C12-15 alkyl sulfate sodium for two years (corresponding to doses of up to 1125 mg/kg/day).

alpha-Olefin sulfonates were not carcinogenic in mice and rats after dermal application, and in rats after oral exposure.

No carcinogenicity studies were available for the alkane sulfonates.

Reproductive toxicity: No indication for adverse effects on reproductive organs was found in various oral studies with different alkyl sulfates. The NOAEL for male fertility was 1000 mg/kg/day for sodium dodecyl sulfate. In a study using alpha-olefin sulfonates in male and female rats, no adverse effects were identified up to 5000 ppm.

Developmental toxicity: In studies with various alkyl sulfates (C12 up to C16-18- alkyl) in rats, rabbits and mice, effects on litter parameters were restricted to doses that caused significant maternal toxicity (anorexia, weight loss, and death).

The principal effects were higher foetal loss and increased incidences of total litter losses. The incidences of malformations and visceral and skeletal anomalies were unaffected apart from a higher incidence of delayed ossification or skeletal variation in mice at > 500 mg/kg bw/day indicative of a delayed development. The lowest reliable NOAEL for maternal toxicity was about 200 mg/kg/day in rats, while the lowest NOAELs in offspring were 250 mg/kg/day in rats and 300 mg/kg/day for mice and rabbits.

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For alpha-olefin sulfonates (C14-16-alpha-olefin sulfonate, sodium) the NOAEL was 600 mg/kg/day both for maternal and developmental toxicity.

No data were available for the reproductive and developmental toxicity of alkane sulfonates. Based on the available data, the similar toxicokinetic properties and a comparable metabolism of the alkyl sulfates and alkane sulfonates, alkane sulfonates are not considered to be developmental toxicants.

Although the database for category members with C<12 is limited, the available data are indicating no risk as the substances have comparable toxicokinetic properties and metabolic pathways. In addition, longer-term studies gave no indication for adverse effects on reproductive organs with different alkyl sulfates

For "kerosenes"

Acute toxicity: Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg. The dermal LD50s of the same three kerosenes were all >2.0 g/kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No. 8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same material produced an LC50 of >6.4 mg/l

When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced "moderate" to "severe" irritation. Six additional skin irritation studies on a range of kerosenes produced "mild" to "severe" irritation.

An eye irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed and washed eyes) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurised kerosene and jet fuel. These materials produced more irritation in the unwashed eyes at 1 hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but by day 7 had resolved.

Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurised kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in guinea pigs

Repeat-Dose toxicity: Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects

Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits. The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24 respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from "slight" to "moderate" in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss. Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related:

- increased relative heart weights for the mid- and high- dose males and females,
- increased absolute and relative spleen weights in treated females, and
- differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stress-related and therefore, indirectly related to treatment).

Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group. These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes.

In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Ophthalmological examination of all animals also found no treatment-related effects. There were no treatment-related effects on growth rates, hematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin.

A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study. In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m³ kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues.

Carcinogenicity: In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation. If the equivalent dose of kerosene was applied to the skin in a manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred. Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration. The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor. A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice. Animal survivals were not affected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity.

In-Vitro (Genotoxicity): The potential *in vitro* genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation. Modified Ames assays on four kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation. The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results. Hydrodesulfurised kerosene tested in a sister chromatid exchange assay produced negative results (with/without activation)

In-Vivo Genotoxicity: Multiple *in vivo* genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in *in vivo* bone marrow cytogenetic tests in Sprague-Dawley rats. One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay. Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation.

Reproductive/Developmental Toxicity Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days pre-mating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day. Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported. There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or

food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds.

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In-Vivo Genotoxicity: Multiple *in vivo* genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in *in vivo* bone marrow cytogenetic tests in Sprague-Dawley rats. One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay. Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation.

Reproductive/Developmental Toxicity Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days pre-mating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day.

Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported. There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds.

No significant acute toxicological data identified in literature search.

**DISTILLATES, PETROLEUM, LIGHT,
HYDROTREATED**

SODIUM PETROLEUM SULFONATE

for alkaryl sulfonate petroleum additives:

Mammalian Toxicology - Acute. Existing data on acute mammalian toxicity indicates a low concern for acute toxicity.

Acute oral toxicity: In all but one studies, there were no deaths that could be attributed to treatment with the test material when administered at the limit dose of 2000 or 5000 mg/kg. In some studies, the primary clinical observations were diarrhea and reduced food consumption (without a change in body weight). These effects are consistent with the gastrointestinal irritant properties of detergents in an oil-based vehicle. In other studies, decreased body weight gain or ruffled fur was observed. In one study where deaths occurred, animals were administered dose levels well above the 2000 mg/kg limit dose. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.

Acute dermal toxicity: No mortality was observed for any tested substance when administered at the limit dose of 2000 or 5000 mg/kg. The principal clinical observation was erythema and/or edema at the site of dermal application. In some cases, the cutaneous findings included dry, flaky skin, desquamation and hyperkeratosis. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.

Acute inhalation toxicity: One member of the petroleum additive alkaryl sulfonate category (CAS RN: 6878396-0) was tested for acute inhalation toxicity (OECD Guideline 403, *Acute Inhalation Toxicity*). Rats were exposed whole-body to an aerosol of the substance at a nominal atmospheric concentration of 1.9 mg/L for four hours. This was the maximum attainable concentration due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. Clinical signs of toxicity during exposure included reduced activity, matted coat, and closed eyes. Clinical signs of toxicity observed post exposure included lacrimation, nasal discharge, salivation rates, matted coat, hunched appearance, soft stools and closed eyes. No treatment-related macroscopic findings were noted. The lack of mortality at a concentration just below the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for this substance.

Mammalian Toxicology - Subchronic Toxicity. Existing data from repeated-dose toxicity studies indicates minimal signs of toxicity following repeated oral exposure. Adverse effects at the site of contact were observed following repeated dermal exposure (injury to the skin) and repeated inhalation (injury to the lungs).

NOAELs range from 49.5 mg/m³ to 1000 mg/kg/day

Mammalian Toxicology - Reproductive and Developmental Toxicity. A one-generation reproductive toxicity test was conducted on one member of the category (CAS # 115733-09-0). Exposure to the alkaryl sulfonate did not significantly impact reproduction or development and these results were bridged to the remainder of the category.

Mammalian Toxicology - Mutagenicity. Existing data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies indicate a low concern for mutagenicity.

No significant acute toxicological data identified in literature search.

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER

for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m³ for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m³. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m³), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating

None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m³ (600 ppm) for PnB and 2,010 mg/m³ (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m³ (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m³ (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m³) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m³). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m³), with decreased body weights occurring at 3000 ppm (11058 mg/m³). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some

Moisture Remover FG #703-2565

	<p>anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.</p> <p>The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. <i>In vitro</i>, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic <i>in vivo</i>. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.</p> <p>NOTE: For PGE - mixed isomers: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.</p>
CARBON DIOXIDE	- pulmonary effects IDLH: 50,000 ppm

Acute Toxicity	⊘	Carcinogenicity	⊘
Skin Irritation/Corrosion	✓	Reproductivity	⊘
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	⊘	STOT - Repeated Exposure	⊘
Mutagenicity	⊘	Aspiration Hazard	⊘

CMR STATUS

Not Applicable

SECTION 12 ECOLOGICAL INFORMATION**Toxicity****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	<ul style="list-style-type: none"> ▶ 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.
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SECTION 14 TRANSPORT INFORMATION**Labels Required**

	
Marine Pollutant	NO
HAZCHEM	2YE

Land transport (ADG)

UN number	1950
Packing group	Not Available
UN proper shipping name	AEROSOLS

Environmental hazard	No relevant data
Transport hazard class(es)	Class : 2.1 Subrisk :
Special precautions for user	Special provisions : 63 190 277 327 Limited quantity : See SP 277

Air transport (ICAO-IATA / DGR)

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

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture**

distillates, petroleum, light, hydrotreated(64742-47-8) is found on the following regulatory lists	"International Maritime Dangerous Goods Requirements (IMDG Code)", "International Council of Chemical Associations (ICCA) - High Production Volume List", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5", "Australia Exposure Standards", "International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index", "Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions", "United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)", "Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes", "OECD List of High Production Volume (HPV) Chemicals", "Australia Inventory of Chemical Substances (AICS)", "OSPAR National List of Candidates for Substitution - Norway", "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)", "Sigma-AldrichTransport Information", "Australia High Volume Industrial Chemical List (HVICL)", "United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)", "OECD Existing Chemicals Database", "Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List", "International Air Transport Association (IATA) Dangerous Goods Regulations", "Australia Hazardous Substances Information System - Consolidated Lists", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)", "International Fragrance Association (IFRA) Survey: Transparency List"
sodium petroleum sulfonate(68608-26-4) is found on the following regulatory lists	"International Council of Chemical Associations (ICCA) - High Production Volume List", "OECD List of High Production Volume (HPV) Chemicals", "Australia Inventory of Chemical Substances (AICS)", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "IMO IBC Code Chapter 17: Summary of minimum requirements"
propylene glycol monomethyl ether - alpha isomer(107-98-2) is found on the following regulatory lists	"International Maritime Dangerous Goods Requirements (IMDG Code)", "International Council of Chemical Associations (ICCA) - High Production Volume List", "Australia Exposure Standards", "International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index", "FisherTransport Information", "United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)", "Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes", "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)", "OSPAR National List of Candidates for Substitution - Norway", "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 National Pollutant Inventory", "Sigma-AldrichTransport Information", "United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)", "OECD

	Existing Chemicals Database", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "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", "International Fragrance Association (IFRA) Survey: Transparency List"
carbon dioxide(124-38-9) is found on the following regulatory lists	"International Maritime Dangerous Goods Requirements (IMDG Code)", "Australia Approved Active Constituents for Agricultural Chemical Products", "Australia Exposure Standards", "International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index", "United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)", "Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes", "OECD List of High Production Volume (HPV) Chemicals", "Australia Inventory of Chemical Substances (AICS)", "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 Numbering System for Food Additives", "UNECE - Kiev Protocol on Pollutant Release and Transfer Registers - Annex II", "Sigma-Aldrich Transport Information", "Australia Dangerous Goods Code (ADG Code) - Packing Instruction - Liquefied and Dissolved Gases", "Australia Australian Pesticides and Veterinary Medicines Authority (APVM) Record of approved active constituents", "Australia High Volume Industrial Chemical List (HVICL)", "United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (Spanish)", "Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List", "Australia Hazardous Substances Information System - Consolidated Lists", "International Air Transport Association (IATA) Dangerous Goods Regulations", "CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP", "Acros Transport Information"

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.

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