Graffiti Remover #823-2621

RS Components

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5155-87**
Version No: **2.1.1.1**
Material Safety Data Sheet according to NOHSC and ADG requirements

Issue Date: **25/11/2014**
Print Date: **26/11/2014**
Initial Date: **Not Available**
S.Local.AUS.EN

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

Product Identifier

Product name	Graffiti Remover #823-2621
Chemical Name	Not Applicable
Synonyms	Manufacturer's Code: 823-2621
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 Cleaners - Heavy duty.

Details of the manufacturer/importer

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.

Poisons Schedule	Not Applicable	
Risk Phrases [1]	**R36/38**	Irritating to eyes and skin.
	R44	Risk of explosion if heated under confinement.
	R67	Vapours may cause drowsiness and dizziness.
	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+, Xi

SAFETY ADVICE

S09	Keep container in a well ventilated place.
S15	Keep away from heat.
S16	Keep away from sources of ignition. No smoking.
S23	Do not breathe gas/fumes/vapour/spray.

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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.
S29	Do not empty into drains.
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 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.
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*.
	Cumulative effects may result following exposure*.
	May produce discomfort of the respiratory system*.
	May affect fertility*.
	May be harmful to the foetus/ embryo*.
	Repeated exposure potentially causes skin dryness and cracking*.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1119-40-0	25-50	dimethyl glutarate
107-98-2	10-25	propylene glycol monomethyl ether - alpha isomer
67-63-0	10-25	isopropanol
106-65-0	5-10	dimethyl succinate
627-93-0	5-10	dimethyl adipate
64-17-5	5-10	ethanol
124-38-9	1-5	carbon dioxide

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	▸ 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.

SECTION 5 FIREFIGHTING MEASURES

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Extinguishing media

	▸ Alcohol stable foam. ▸ Dry chemical powder. ▸ BCF (where regulations permit). ▸ Carbon dioxide. ▸ Water spray or fog - Large fires only.

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), aldehydes, other pyrolysis products typical of burning organic material**WARNING**: Long standing in contact with air and light may result in the formationof potentially explosive peroxides.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	Slippery when spilt. ▸ 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	▸ **DO NOT** allow clothing wet with material to stay in contact with skin The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe ▸ **DO NOT** concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential. ▸ Any static discharge is also a source of hazard. ▸ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina. ▸ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage. ▸ Add inhibitor to any distillate as required. ▸ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely. The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example. Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised. ▸ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date.

Continued...

	▸ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▸ Unopened containers received from the supplier should be safe to store for 18 months. ▸ Opened containers should not be stored for more than 12 months. ▸ 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	▸ 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

Suitable container	▸ Aerosol dispenser. ▸ Check that containers are clearly labelled.
Storage incompatibility	▸ 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	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	isopropanol	Isopropyl alcohol	983 mg/m3 / 400 ppm	1230 mg/m3 / 500 ppm	Not Available	Not Available
Australia Exposure Standards	ethanol	Ethyl alcohol	1880 mg/m3 / 1000 ppm	Not Available	Not Available	Not Available
Australia Exposure Standards	carbon dioxide	Carbon dioxide / Carbon dioxide in coal mines	9000 mg/m3 / 22500 mg/m3 / 5000 ppm / 12500 ppm	54000 mg/m3 / 30000 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether; (Ucar Triol HG-170)	150 ppm	150 ppm	470 ppm
isopropanol	Isopropyl alcohol	400 ppm	400 ppm	12000 ppm
dimethyl succinate	Butanedioic acid, dimethyl ester; (Succinic acid, dimethyl ester)	2.5 ppm	28 ppm	170 ppm
ethanol	Ethyl alcohol; (Ethanol)	Not Available	Not Available	Not Available
carbon dioxide	Carbon dioxide	30,000 ppm	30000 ppm	50000 ppm

Ingredient	Original IDLH	Revised IDLH
dimethyl glutarate	Not Available	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available	Not Available
isopropanol	12,000 ppm	2,000 [LEL] ppm
dimethyl succinate	Not Available	Not Available
dimethyl adipate	Not Available	Not Available
ethanol	15,000 ppm	3,300 [LEL] ppm

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carbon dioxide	50,000 ppm	40,000 ppm

Exposure controls

Appropriate engineering controls

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

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

- 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
NEOPRENE	A
NITRILE	B

Respiratory protection

Type A-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

Continued...

PVC	B

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

up to 5 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	A-2 P2	A-PAPR-2 P2
up to 50 x ES	-	A-3 P2	-
50+ 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)

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Colourless highly flammable liquid aerosol with a solvent odour; insoluble in water.

Physical state	Liquid	**Relative density (Water = 1)**	0.94 @ 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)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	12 (CC)	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Flammable.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	900 g/l (VOC)
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water (g/L)	Immiscible	**pH as a solution(1%)**	Not Applicable
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Inhalation of 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. 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. Inhalation hazard is increased at higher temperatures. **WARNING**:Intentional misuse by concentrating/inhaling contents may be lethal.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments

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Skin Contact	The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. 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. 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 Open cuts, abraded or irritated skin should not be exposed to this material 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.
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.. Evidence exists, or practical experience predicts, that the material may cause 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 eye contact may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	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. Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

Graffiti Remover #823-2621	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
dimethyl glutarate	**TOXICITY**	**IRRITATION**
	Oral (mouse) LD50: 2227 mg/kg	[Manuf. DU]
	Oral (rat) LD50: 5000 mg/mg	Eye (rabbit): Irritant
		Skin (human): Irritant
	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
		Skin (rabbit) 500 mg open - mild
	Not Available	Not Available
isopropanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 12800 mg/kg	Eye (rabbit): 10 mg - moderate
	Inhalation (Mouse) LC50: 53000 mg/m3/4h	Eye (rabbit): 100 mg - SEVERE
	Inhalation (Rat) LC50: 72600 mg/m3/4h	Eye (rabbit): 100mg/24hr-moderate
	Intraperitoneal (Guinea pig) LD50: 2560 mg/kg	Skin (rabbit): 500 mg - mild
	Intraperitoneal (Mouse) LD50: 4477 mg/kg	
	Intraperitoneal (Rabbit) LD50: 667 mg/kg	
	Intraperitoneal (Rat) LD50: 2735 mg/kg	
	Intravenous (Mouse) LD50: 1509 mg/kg	
	Intravenous (Rabbit) LD50: 1184 mg/kg	
	Intravenous (Rat) LD50: 1088 mg/kg	
	Oral (Mouse) LD50: 3600 mg/kg	
	Oral (Rabbit) LD50: 6410 mg/kg	
	Oral (Rat) LD50: 5000 mg/kg	
	Oral (rat) LD50: 5045 mg/kg	
	Not Available	Not Available
dimethyl succinate	**TOXICITY**	**IRRITATION**
	Dermal (rat) LD50: >5000 mg/kg	Nil reported
	Oral (rat) LD50: >5000 mg/kg	
	Not Available	Not Available

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dimethyl adipate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2500 mg/kg	Eye (rabbit): Irritant
	Inhalation (rat) LC50: 10.7 mg/l/1h	Skin (human): SEVERE
	Inhalation (rat) LC50: 11 mg/l/4h	
	Oral (rat) LD50: 8191 mg/kg	
	Not Available	Not Available
ethanol	**TOXICITY**	**IRRITATION**
	Inhalation (rat) LC50: 20,000 ppm/10h	Eye (rabbit): 500 mg SEVERE
	Inhalation (rat) LC50: 64000 ppm/4h	Eye (rabbit):100mg/24hr-moderate
	Oral (rat) LD50: 7060 mg/kg	Skin (rabbit):20 mg/24hr-moderate
		Skin (rabbit):400 mg (open)-mild
	Not Available	Not Available
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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The family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CAS No. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixture DBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produce DMS, DMG, and DMA and three other fractions that are mixtures of these esters generally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbon straight-chain dicarboxylic acid dimethyl esters differing incrementally by one carbon atom. The four members of the category produce similar levels of acute and repeated-dose toxicity in experimental animals
DBEs have very low acute oral toxicities with LD50 s in rats generally > 5,000 mg/kg (with two exceptions reported as >500 and <5,000 mg/kg b.wt. for DBE (the mixture) and DMS
By skin absorption, DBEs have a low order of acute toxicity to rabbits with dermal LD50s of 3,000 mg/kg . Based upon the most recent GLP studies DBEs are not considered to produce primary dermal irritation as defined in EPA Guidelines . Earlier studies did show moderate irritation in one of six rabbits, but these results were not repeated in later studies. All four DBE materials are considered to produce eye irritation as defined by EPA Guidelines. Mild to moderate irritation involving the cornea was observed in rabbits with recovery by 7 days. DBEs are not skin sensitisers, and are not harmful via skin or inhalation exposures. DBE is slightly toxic by inhalation with 1-and 4-hour LC50s in rats of > 10.7 and > 11 mg/L, respectively. In subchronic inhalation studies with all four DBEs, degeneration of the olfactory epithelium of the nose was observed. This change in the nasal tissues is related to enzymatic hydrolysis of DBE within the nasal cavity. However, risk to human nasal tissue due to DBE toxicity is likely to be reduced when compared to rats since DBEs are hydrolysed more slowly in humans. No information is available on the carcinogenic potential of DBEs. A range of studies with DMS, DMG, DMA and DBE did not produce genetic damage in animals or bacterial cell cultures. DBE was positive in one study with cultured mammalian cells, but the positive findings were not apparent when the assay was repeated. Testing in rats indicates DBEs are not developmental or reproductive toxicants.
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 sensitisers.
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

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	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. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
DIMETHYL GLUTARATE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. The family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CAS No. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixture DBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produce DMS, DMG, and DMA and three other fractions that are mixtures of these esters generally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbon straight-chain dicarboxylic acid dimethyl esters differing incrementally by one carbon atom. The four members of the category produce similar levels of acute and repeated-dose toxicity in experimental animals DBEs have very low acute oral toxicities with LD50 s in rats generally > 5,000 mg/kg (with two exceptions reported as >500 and <5,000 mg/kg b.wt. for DBE (the mixture) and DMS By skin absorption, DBEs have a low order of acute toxicity to rabbits with dermal LD50s of 3,000 mg/kg . Based upon the most recent GLP studies DBEs are not considered to produce primary dermal irritation as defined in EPA Guidelines . Earlier studies did show moderate irritation in one of six rabbits, but these results were not repeated in later studies. All four DBE materials are considered to produce eye irritation as defined by EPA Guidelines. Mild to moderate irritation involving the cornea was observed in rabbits with recovery by 7 days. DBEs are not skin sensitisers, and are not harmful via skin or inhalation exposures. DBE is slightly toxic by inhalation with 1-and 4-hour LC50s in rats of > 10.7 and > 11 mg/L, respectively. In subchronic inhalation studies with all four DBEs, degeneration of the olfactory epithelium of the nose was observed. This change in the nasal tissues is related to enzymatic hydrolysis of DBE within the nasal cavity. However, risk to human nasal tissue due to DBE toxicity is likely to be reduced when compared to rats since DBEs are hydrolysed more slowly in humans. No information is available on the carcinogenic potential of DBEs. A range of studies with DMS, DMG, DMA and DBE did not produce genetic damage in animals or bacterial cell cultures. DBE was positive in one study with cultured mammalian cells, but the positive findings were not apparent when the assay was repeated. Testing in rats indicates DBEs are not developmental or reproductive toxicants.
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 sensitisers. 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

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	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. 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.
ISOPROPANOL	For isopropanol (IPA): **Acute toxicity:** Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat. Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred. **Repeat dose studies**: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney. **Reproductive toxicity**: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful. **Developmental toxicity:** The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity **Genotoxicity:** All genotoxicity assays reported for isopropanol have been negative **Carcinogenicity:** rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. 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.
DIMETHYL SUCCINATE	The family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CAS No. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixture DBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produce DMS, DMG, and DMA and three other fractions that are mixtures of these esters generally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbon straight-chain dicarboxylic acid dimethyl esters differing incrementally by one carbon atom. The four members of the category produce similar levels of acute and repeated-dose toxicity in experimental animals DBEs have very low acute oral toxicities with LD50 s in rats generally > 5,000 mg/kg (with two exceptions reported as >500 and <5,000 mg/kg b.wt. for DBE (the mixture) and DMS By skin absorption, DBEs have a low order of acute toxicity to rabbits with dermal LD50s of 3,000 mg/kg . Based upon the most recent GLP studies DBEs are not considered to produce primary dermal irritation as defined in EPA Guidelines . Earlier studies did show moderate irritation in one of six rabbits, but these results were not repeated in later studies. All four DBE materials are considered to produce eye irritation as defined by EPA Guidelines. Mild to moderate irritation involving the cornea was observed in rabbits with recovery by 7 days. DBEs are not skin sensitisers, and are not harmful via skin or inhalation exposures. DBE is slightly toxic by inhalation with 1-and 4-hour LC50s in rats of > 10.7 and > 11 mg/L, respectively. In subchronic inhalation studies with all four DBEs, degeneration of the olfactory epithelium of the nose was observed. This change in the nasal tissues is related to enzymatic hydrolysis of DBE within the nasal cavity. However, risk to human nasal tissue due to DBE toxicity is likely to be reduced when compared to rats since DBEs are hydrolysed more slowly in humans. No information is available on the carcinogenic potential of DBEs. A range of studies with DMS, DMG, DMA and DBE did not produce genetic damage in animals or bacterial cell cultures. DBE was positive in one study with cultured mammalian cells, but the positive findings were not apparent when the assay was repeated. Testing in rats indicates DBEs are not developmental or reproductive toxicants.
DIMETHYL ADIPATE	The family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CAS No. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixture DBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produce DMS, DMG, and DMA and three other fractions that are mixtures of these esters generally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbon straight-chain dicarboxylic acid dimethyl esters differing incrementally by one carbon atom. The four members of the category produce similar levels of acute and repeated-dose toxicity in experimental animals DBEs have very low acute oral toxicities with LD50 s in rats generally > 5,000 mg/kg (with two exceptions reported as >500 and <5,000 mg/kg b.wt. for DBE (the mixture) and DMS By skin absorption, DBEs have a low order of acute toxicity to rabbits with dermal LD50s of 3,000 mg/kg . Based upon the most recent GLP studies DBEs are not considered to produce primary dermal irritation as defined in EPA Guidelines . Earlier studies did show moderate irritation in one of six rabbits, but these results were not repeated in later studies. All four DBE materials are considered to produce eye irritation as defined by EPA Guidelines. Mild to moderate irritation involving the cornea was observed in rabbits with recovery by 7 days. DBEs are not skin sensitisers, and are not harmful via skin or inhalation exposures. DBE is slightly toxic by inhalation with 1-and 4-hour LC50s in rats of > 10.7 and > 11 mg/L, respectively. In subchronic inhalation studies with all four DBEs, degeneration of the olfactory epithelium of the nose was observed. This change in the nasal tissues is related to enzymatic hydrolysis of DBE within the nasal cavity. However, risk to human nasal tissue due to DBE toxicity is likely to be reduced when compared to rats since DBEs are hydrolysed more slowly in humans. No information is available on the carcinogenic potential of DBEs. A range of studies with DMS, DMG, DMA and DBE did not produce genetic damage in animals or bacterial cell cultures. DBE was positive in one study with cultured mammalian cells, but the positive findings were not apparent when the assay was repeated. Testing in rats indicates DBEs are not developmental or reproductive toxicants. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.

Continued...

ETHANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
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**	⊘

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

REPROTOXIN	carbon dioxide	ILO Chemicals in the electronics industry that have toxic effects on reproduction	
SKIN	dimethyl glutarate	Australia Exposure Standards - Skin	Sk
	dimethyl adipate	Australia Exposure Standards - Skin	Sk

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
dimethyl glutarate	LOW	LOW
propylene glycol monomethyl ether - alpha isomer	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
dimethyl succinate	LOW	LOW
dimethyl adipate	LOW	LOW
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
carbon dioxide	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
dimethyl glutarate	LOW (LogKOW = 0.62)
propylene glycol monomethyl ether - alpha isomer	LOW (BCF = 2)
isopropanol	LOW (LogKOW = 0.05)
dimethyl succinate	LOW (LogKOW = 0.35)
dimethyl adipate	LOW (LogKOW = 1.03)
ethanol	LOW (LogKOW = -0.31)
carbon dioxide	LOW (LogKOW = 0.83)

Mobility in soil

Ingredient	Mobility
dimethyl glutarate	LOW (KOC = 10)
propylene glycol monomethyl ether - alpha isomer	HIGH (KOC = 1)
isopropanol	HIGH (KOC = 1.06)
dimethyl succinate	LOW (KOC = 10)
dimethyl adipate	LOW (KOC = 10.9)
ethanol	HIGH (KOC = 1)
carbon dioxide	HIGH (KOC = 1.498)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some

Continued...

disposal	areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **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	NO
HAZCHEM	2YE

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

Continued...

Special precautions for user	EMS Number	F-D , S-U
	Special provisions	63 190 277 327 344 959
	Limited Quantities	See SP277

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	dimethyl glutarate	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	propylene glycol monomethyl ether - alpha isomer	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	dimethyl succinate	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	dimethyl adipate	X

SECTION 15 REGULATORY INFORMATION

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

dimethyl glutarate(1119-40-0) is found on the following regulatory lists	"Australia Exposure Standards","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
propylene glycol monomethyl ether - alpha isomer(107-98-2) is found on the following regulatory lists	"Australia Exposure Standards","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
isopropanol(67-63-0) is found on the following regulatory lists	"Australia Exposure Standards","Australia Inventory of Chemical Substances (AICS)","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Australia Hazardous Substances Information System - Consolidated Lists"
dimethyl succinate(106-65-0) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
dimethyl adipate(627-93-0) is found on the following regulatory lists	"Australia Exposure Standards","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
ethanol(64-17-5) is found on the following regulatory lists	"Australia Exposure Standards","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
carbon dioxide(124-38-9) is found on the following regulatory lists	"Australia Exposure Standards","Australia Inventory of Chemical Substances (AICS)","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Australia Hazardous Substances Information System - Consolidated Lists"

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.

end of SDS