

Motor Active

Chemwatch: 4782-52

Version No: 3.1.1.1 Safety Data Sheet according to WHS and ADG requirements Chemwatch Hazard Alert Code: 2 Issue Date: 01/09/2015

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L.GHS.AUS.EN

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

Product Identifier	
Product name	Meguiar's G82, Perfect Clarity Glass Cleaner Trigger Spray
Synonyms	Not Available
Other means of identification	Not Available
Relevant identified uses of the substance or mixture and uses advised against	
Relevant identified uses	Use according to manufacturer's directions. Automotive; glass cleaner.

Details of the supplier of the safety data sheet

Registered company name	Motor Active	Meguiars
Address	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia	17991 Mitchell South Irvine CA 92714 United States
Telephone	+61 2 9737 9422 1800 350 622	+1 949 752 8000 +1 800 347 5700
Fax	+61 2 9737 9414	+1 949 752 5784
Website	www.motoractive.com.au	https://www.meguiars.com/
Email	andrew.spira@motoractive.com.au	Not Available

Emergency telephone number

Association / Organisation	MotorActive	Not Available
Emergency telephone numbers	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)	Not Available
Other emergency telephone numbers	13 11 26 (In Case of Emergency contact: Poison Information Hotline)	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

NON-HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

CHEMWATCH HAZARD RATINGS

	Min	Max	
Flammability	1	1	
Toxicity	1		0 = Minimum
Body Contact	2	1	1 = Low 2 = Moderate
Reactivity	1		3 = High
Chronic	0	1	4 = Extreme

Poisons Schedule	Not Applicable
Classification	Not Applicable
Label elements	
GHS label elements	Not Applicable
SIGNAL WORD	NOT APPLICABLE

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
111-76-2	1-5	ethylene glycol monobutyl ether
67-63-0	1-5	isopropanol
	balance	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	 If this product comes in contact with the eyes: Wash out immediately with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	 If skin contact occurs: Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation.
Inhalation	 If fumes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
Ingestion	 If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to ethylene glycol:

- Early treatment of ingestion is important. Ensure emesis is satisfactory.
- Test and correct for metabolic acidosis and hypocalcaemia.
- Apply sustained diuresis when possible with hypertonic mannitol.
- Evaluate renal status and begin haemodialysis if indicated. [I.L.O]
- P Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- + Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures. Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- Water spray or fog.
- ► Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

Special hazards arising from the substrate or mixture

	Fire Incompatibility	• Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result	
A	dvice for firefighters		
	Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear full body protective clothing with breathing apparatus. Prevent, by any means available, spillage from entering drains or water course. Use water delivered as a fine spray to control fire and cool adjacent area. Avoid spraying water onto liquid pools. 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. 	

	Fire/Explosion Hazard	 Combustible. Slight fire hazard when exposed to heat or flame. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO). May emit acrid smoke. Mists containing combustible materials may be explosive. Combustion products include; carbon dioxide (CO2) other pyrolysis products typical of burning organic materialMay emit poisonous fumes.May emit corrosive fumes.
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SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	 Remove all ignition sources. Clean up all spills immediately. Avoid breathing vapours and contact with skin and eyes. Control personal contact with the substance, by using protective equipment. Contain and absorb spill with sand, earth, inert material or vermiculite. Wipe up. Place in a suitable, labelled container for waste disposal.
Major Spills	 Moderate hazard. Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. No smoking, naked lights or ignition sources. Increase ventilation. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. Collect recoverable product into labelled containers for recycling. Absorb remaining product with sand, earth or vermiculite. Collect solid residues and seal in labelled drums for disposal. Wash area and prevent runoff into drains. If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

	5
Safe handling	 Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. Prevent concentration in hollows and sumps. DO NOT enter confined spaces until atmosphere has been checked. Avoid smoking, naked lights or ignition sources. Avoid contact with incompatible materials. When handling, DO NOT est, drink or smoke. Keep containers securely sealed when not in use. Avoid physical damage to containers. Always wash hands with scap and water after handling. Work clothes should be laundered separately. Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions. DO NOT allow clothing wet with material to stay in contact with skin
Other information	 Store in original containers. Keep containers securely sealed. No smoking, naked lights or ignition sources. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.
onditions for safe storag	ge, including any incompatibilities
Suitable container	 Metal can or drum Packaging as recommended by manufacturer.

Suitable container	 Metal can or drum Packaging as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Avoid reaction with oxidising agents

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

strong acids

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA		STEL		Peak		Notes
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	96.9 mg/m3 / 2	20 ppm	242 mg/m3	3 / 50 ppm	Not Ava	ailable	Sk
Australia Exposure Standards	isopropanol	Isopropyl alcohol	983 mg/m3 / 4	00 ppm	1230 mg/m	n3 / 500 ppm	Not Ava	ailable	Not Available
EMERGENCY LIMITS									
Ingredient	Material name		TEEL-1		TEEL-2		TEEL-3		
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)		20 ppm		20 ppm		700 ppm		
isopropanol	Isopropyl alcohol		400 ppm		400 ppm		12000 pp	m	
Ingredient	Original IDLH		Revised IDLH						
ethylene glycol monobutyl ether	700 ppm		700 [Unch] ppm						
isopropanol	12,000 ppm		2,000 [LEL] ppm						

MATERIAL DATA

For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance. Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL)

Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)

Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

Exposure 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 operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant. Air Speed: Type of Contaminant 0.25-0.5 m/s (50-100 solvent, vapours, degreasing etc., evaporating from tank (in still air). f/min) aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating 0.5-1 m/s (100-200 acid fumes, pickling (released at low velocity into zone of active generation) f/min.) Appropriate engineering direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into 1-2.5 m/s (200-500 controls zone of rapid air motion) f/min.) grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid 2.5-10 m/s (500-2000 air motion) f/min.) Within each range the appropriate value depends on: I ower 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



 Safety glasses with side shields. Chemical goggles

Eye and face protection

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	 Wear chemical protective gloves, e.g. PVC. Wear safety footwear or safety gumboots, e.g. Rubber The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: frequency and duration of contact, chemical resistance of glove material, glove thickness and dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). When only brief contact is expected, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. Contaminated gloves should be replaced.
Body protection	See Other protection below
Other protection	 Overalls. P.V.C. apron. Barrier cream. Skin cleansing cream. Eye wash unit.
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:

Meguiar's G82, Perfect Clarity Glass Cleaner Trigger Spray

Material	CPI
NITRILE	А
PE/EVAL/PE	А
NEOPRENE	В
PVC	В
NAT+NEOPR+NITRILE	С
NATURAL RUBBER	С

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	A-AUS / Class 1	-	A-PAPR-AUS / Class 1
up to 25 x ES	Air-line*	A-2	A-PAPR-2
up to 50 x ES	-	A-3	-
50+ x ES	-	Air-line**	-

^ - Full-face

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

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Off-white liquid with a sweet odour; miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.99
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	4.5-5.5	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available

Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>93	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	VOC = 4.00%
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	 Unstable in the presence of incompatible materials. 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

Inhaledfollowing inhala the damage. The damage resultir involving the redIngestionAccidental ingestSkin ContactLimited evidence following direct to being present to result in a form progress to blist the skin (spong Open cuts, abreEyeLimited evidence following direct to being present to result in a form progress to blist the skin (spong Open cuts, abre and/or other trans Limited evidence produce signific eye contact may and/or other trans Limited evidence of on sequence of On the basis, prevailable inform Long term or red Repeated inhala exposure levels animals.ChronicRepeated inhala exposure levels animals.There are incor are persons when Continued volume	tion. In contrast to most organs, the lung is able to respond to a		
Skin Contact Limited evidence following direct being present to result in a form progress to blist the skin (spong Open cuts, abra Eye Limited evidence produce signific eye contact may and/or other trai Limited evidence roduce signific eye contact may and/or other trai Limited evidence There is some e been observed i consequences of consequence of . Chronic . Chronic . There are incor are persons who Continued volur	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.		
Skin Contact following direct is being present to result in a form progress to bilis the skin (spong Open cuts, abra Eye Limited evidence signific eve contact may and/or other transand/or	stion of the material may be damaging to the health of the individu	Jal.	
Eye produce signific eye contact may and/or other trai Limited evidence There is some e been observed is consequences of consequences of . Chronic . Chronic . Repeated inhala exposure levels animals. There are incor are persons who Continued volur	Limited 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. Open cuts, abraded or irritated skin should not be exposed to this material		
Chronic Chronic There is some a been observed is consequences of toxic effects, or consequence of . On the basis, pravailable inform Long term or replayed to the exposure levels animals. There are incorr are persons who continued volur	Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.		
been shown to b	evidence that human exposure to the material may result in develop in the absence of marked maternal toxicity, or at around the same of the other toxic effects. ematerial may cause concerns for human fertility, on the basis that evidence of impaired fertility occurring at around the same dose le f other toxic effects. rimarily, of animal experiments, concern has been expressed that ation, however, there presently exists inadequate data for making peated ingestion exposure of isopropanol may produce incoordin ation exposure to isopropanol may produce narcosis, incoordinatic that produce toxic effects in the adult animals. Isopropanol does inclusive reports of human sensitisation from skin contact with isop o do not consume alcohol; alcoholics have survived as much as 5 intary drinking of a 2.5% aqueous solution through two successive orcial isopropanol does not contain "isopropyl oil". An excess inci-	ation, lethargy and reduced weight gain. on and liver degeneration. Animal data show developmental effects only at not cause genetic damage in bacterial or mammalian cell cultures or in propanol. Chronic alcoholics are more tolerant of systemic isopropanol than 500 ml. of 70% isopropanol.	
Meguiar's G82, Perfect TOXICITY		IRRITATION	
Clarity Glass Cleaner Trigger Spray		Not Available	

	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	* [Union Carbide]
ethylene glycol monobutyl ether	Inhalation (rat) LC50: 450 ppm/4hr ^[2]	Eye (rabbit): 100 mg SEVERE
	Oral (rat) LD50: 250 mg/kg ^[2]	Eye (rabbit): 100 mg/24h-moderate
		Skin (rabbit): 500 mg, open; mild
	ΤΟΧΙΟΙΤΥ	IRRITATION
	Dermal (rabbit) LD50: 12792 mg/kg ^[1]	Eye (rabbit): 10 mg - moderate
isopropanol	Inhalation (rat) LC50: 72.6 mg/L/4hr ^[2]	Eye (rabbit): 100 mg - SEVERE
	Oral (rat) LD50: 5000 mg/kg ^[2]	Eye (rabbit): 100mg/24hr-moderate
		Skin (rabbit): 500 mg - mild
Legend:	1. Value obtained from Europe ECHA Registered Substances extracted from RTECS - Register of Toxic Effect of chemical S	 Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data Substances
	conjunctivitis. For ethylene glycol monoalkyl ethers and their acetates (EGM. Typical members of this category are ethylene glycol propylene their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme / transient metabolites). Further, rapid conversion of the aldehyd	pronounced inflammation. Repeated or prolonged exposure to irritants may produce AEs): e ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are es by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary
	metabolites of mono substituted glycol ethers. Acute Toxicity: Oral LD50 values in rats for all category mem-	bers range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing

Acute Toxicity: Cral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGPE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemolysis or haemolytion as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity form EGPE and EGBE *in vitro* than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are

not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the

ETHYLENE GLYCOL MONOBUTYL ETHER

> Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE). Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species. At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol. Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infraction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration

-dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.
 1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

testes)

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glycal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glycoxylate; glycoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO2, ethylene glycol is eliminated in the urine as

both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12-24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown. **Gastrointestinal Effects**. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tendemess and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia. Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available in vivo and in vitro laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS 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 dearees 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.

ISOPROPANOL

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 male rats are considered of no significance in terms of human cancer risk assessment

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.

Meguiar's G82, Perfect Clarity Glass Cleaner Trigger Spray

ETHYLENE GLYCOL MONOBUTYL ETHER & ISOPROPANOL	The material may cause skin irritation after prolonged or repe- characterised by skin redness (erythema) and swelling epider intracellular oedema of the epidermis.	1	
Acute Toxicity	0	Carcinogenicity	0
Skin Irritation/Corrosion	0	Reproductivity	0
Serious Eye Damage/Irritation	0	STOT - Single Exposure	0
Respiratory or Skin sensitisation	0	STOT - Repeated Exposure	0
Mutagenicity	0	Aspiration Hazard	0
		Legend: X	- Data available but does not fill the criteria for classification

✓ – Data required to make classification available 🚫 – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

loxicity					
Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
ethylene glycol monobutyl ether	EC50	384	Crustacea	51.539mg/L	3
ethylene glycol monobutyl ether	LC50	96	Fish	222.042mg/L	3
ethylene glycol monobutyl ether	EC50	48	Crustacea	164mg/L	2
ethylene glycol monobutyl ether	NOEC	168	Crustacea	56mg/L	2
ethylene glycol monobutyl ether	EC50	96	Algae or other aquatic plants	720mg/L	2
isopropanol	EC50	384	Crustacea	42.389mg/L	3
isopropanol	EC50	96	Algae or other aquatic plants	993.232mg/L	3
isopropanol	LC50	96	Fish	183.844mg/L	3
isopropanol	NOEC	5760	Fish	0.02mg/L	4
isopropanol	EC50	48	Crustacea	12500mg/L	5
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
isopropanol	LOW (LogKOW = 0.05)

Mobility in soil

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (KOC = 1)
isopropanol	HIGH (KOC = 1.06)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

	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 areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: Reduction	
Product / Packaging	► Reuse	
disposal	▶ 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.	
	The analysis of a material may shange in dee, and respense of reader may not analysis of 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.
 Recycle wherever possible or consult manufacturer for recycling options.
 Consult State Land Waste Authority for disposal.
 Bury or incinerate residue at an approved site.
 Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards		Australia Inventory of Chemical Substances (AICS)		
Australia Hazardous Substances Information System - Consolidated Lists		International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs		
ISOPROPANOL(67-63-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS				
Australia Exposure Standards		Australia Inventory of Chemical Substances (AICS)		
Australia Hazardous Substances Information System - Consolidated Lists		International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs		
National Inventory	Status			
Australia - AICS	Y			

Australia - AICS	Y	
Canada - DSL	Y	
Canada - NDSL	N (isopropanol; ethylene glycol monobutyl ether)	
China - IECSC	Y	
Europe - EINEC / ELINCS / NLP	Y	
Japan - ENCS	Y	
Korea - KECI	Y	
New Zealand - NZIoC	Y	
Philippines - PICCS	Y	
USA - TSCA	Y	
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)	

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 Chernwatch Classification committee using available literature references.

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average PC – STEL: Permissible Concentration-Short Term Exposure Limit IARC: International Agency for Research on Cancer ACGIH: American Conference of Governmental Industrial Hygienists STEL: Short Term Exposure Limit TEEL: Temporary Emergency Exposure Limit。 IDLH: Immediately Dangerous to Life or Health Concentrations OSF: Odour Safety Factor NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index

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