

Servisol NA1002 PCB Aerosol Lacquer

Electus Distribution

Chemwatch: 4643-90

Version No: 4.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 4

Issue Date: 01/01/2013

Print Date: 11/04/2015

Initial Date: **Not Available**

L.GHS.AUS.EN

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

Product Identifier

Product name	Servisol NA1002 PCB Aerosol Lacquer
Synonyms	Circuit Board Lacquer
Proper shipping name	AEROSOLS
Other means of identification	Not Available

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 Use according to manufacturer's directions. Coating product.
---------------------------------	--

Details of the manufacturer/importer

Registered company name	Electus Distribution
Address	320 Victoria Road Rydalmere 2116 NSW Australia
Telephone	1300 738 555
Fax	1300 758 500
Website	Not Available
Email	Not Available

Emergency telephone number

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

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

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

Poisons Schedule	S5
GHS Classification [1]	Flammable Aerosol Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Reproductive Toxicity Category 2, STOT - SE (Narcosis) Category 3, STOT - RE Category 2

Continued...

Servisol NA1002 PCB Lacquer

Legend: 1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	
SIGNAL WORD	DANGER

Hazard statement(s)

H222	Extremely flammable aerosol
H302	Harmful if swallowed
H315	Causes skin irritation
H319	Causes serious eye irritation
H361	Suspected of damaging fertility or the unborn child
H336	May cause drowsiness or dizziness
H373	May cause damage to organs through prolonged or repeated exposure
AUH044	Risk of explosion if heated under confinement
AUH066	Repeated exposure may cause skin dryness and cracking

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P314	Get medical advice/attention if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration
------	--

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
108-88-3	10-30	toluene
67-64-1	10-30	acetone
Not Available	10-30	additives nonhazardous
68476-85-7.	25-35	hydrocarbon propellant

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> ■ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ■ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ■ Transport to hospital or doctor without delay. ■ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> ■ Flush skin and hair with running water (and soap if available). ■ Remove any adhering solids with industrial skin cleansing cream. ■ DO NOT use solvents. ■ Seek medical attention in the event of irritation.
Inhalation	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> ■ Remove to fresh air. ■ Lay patient down. Keep warm and rested. ■ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ■ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ■ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ■ Avoid giving milk or oils. ■ Avoid giving alcohol. ■ Not considered a normal route of entry. ■ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

For acute or short term repeated exposures to acetone:

- Symptoms of acetone exposure approximate ethanol intoxication.
- About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.
- There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]

Management:

Measurement of serum and urine acetone concentrations may be useful to monitor the severity of ingestion or inhalation.

Inhalation Management:

- Maintain a clear airway, give humidified oxygen and ventilate if necessary.
- If respiratory irritation occurs, assess respiratory function and, if necessary, perform chest X-rays to check for chemical pneumonitis.
- Consider the use of steroids to reduce the inflammatory response.
- Treat pulmonary oedema with PEEP or CPAP ventilation.

Dermal Management:

- Remove any remaining contaminated clothing, place in double sealed, clear bags, label and store in secure area away from patients and staff.
- Irrigate with copious amounts of water.
- An emollient may be required.

Eye Management:

- Irrigate thoroughly with running water or saline for 15 minutes.
- Stain with fluorescein and refer to an ophthalmologist if there is any uptake of the stain.

Oral Management:

- No **GASTRIC LAVAGE OR EMETIC**
- Encourage oral fluids.

Systemic Management:

- Monitor blood glucose and arterial pH.
- Ventilate if respiratory depression occurs.

Continued...

Servisol NA1002 PCB Lacquer

- If patient unconscious, monitor renal function.
- Symptomatic and supportive care.

The Chemical Incident Management Handbook:

Guy's and St. Thomas' Hospital Trust, 2000

BIOLOGICAL EXPOSURE INDEX

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Sampling Time	Index	Comments
Acetone in urine	End of shift	50 mg/L	NS

NS: Non-specific determinant; also observed after exposure to other material

Following acute or short term repeated exposures to toluene:

- Toluene is absorbed across the alveolar barrier, the blood/air mixture being 11.2/15.6 (at 37 degrees C.) The concentration of toluene, in expired breath, is of the order of 18 ppm following sustained exposure to 100 ppm. The tissue/blood proportion is 1/3 except in adipose where the proportion is 8/10.
- Metabolism by microsomal mono-oxygenation, results in the production of hippuric acid. This may be detected in the urine in amounts between 0.5 and 2.5 g/24 hr which represents, on average 0.8 gm/gm of creatinine. The biological half-life of hippuric acid is in the order of 1-2 hours.
- Primary threat to life from ingestion and/or inhalation is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (eg cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ <50 mm Hg or pCO₂ > 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial damage has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenaline) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- Lavage is indicated in patients who require decontamination; ensure use.

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
o-Cresol in urine	0.5 mg/L	End of shift	B
Hippuric acid in urine	1.6 g/g creatinine	End of shift	B, NS
Toluene in blood	0.05 mg/L	Prior to last shift of workweek	

NS: Non-specific determinant; also observed after exposure to other material

B: Background levels occur in specimens collected from subjects NOT exposed

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

SMALL FIRE:

- Water spray, dry chemical or CO₂

LARGE FIRE:

- Water spray or fog.

Special hazards arising from the substrate or mixture

Fire Incompatibility

- Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting

- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- If safe, switch off electrical equipment until vapour fire hazard removed.
- Use water delivered as a fine spray to control fire and cool adjacent area.
- **DO NOT** approach containers suspected to be hot.
- Cool fire exposed containers with water spray from a protected location.
- If safe to do so, remove containers from path of fire.
- Equipment should be thoroughly decontaminated after use.

Fire/Explosion Hazard	<ul style="list-style-type: none"> ■ Liquid and vapour are highly flammable. ■ Severe fire hazard when exposed to heat or flame. ■ Vapour forms an explosive mixture with air. ■ Severe explosion hazard, in the form of vapour, when exposed to flame or spark. ■ Vapour may travel a considerable distance to source of ignition. ■ Heating may cause expansion or decomposition with violent container rupture. ■ Aerosol cans may explode on exposure to naked flames. ■ Rupturing containers may rocket and scatter burning materials. ■ Hazards may not be restricted to pressure effects. ■ May emit acrid, poisonous or corrosive fumes. ■ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include; carbon monoxide (CO) carbon dioxide (CO₂) other pyrolysis products typical of burning organic material Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.</p>
------------------------------	--

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	<ul style="list-style-type: none"> ■ Clean up all spills immediately. ■ Avoid breathing vapours and contact with skin and eyes. ■ Wear protective clothing, impervious gloves and safety glasses. ■ Shut off all possible sources of ignition and increase ventilation. ■ Wipe up. ■ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ■ Undamaged cans should be gathered and stowed safely.
Major Spills	<ul style="list-style-type: none"> ■ DO NOT exert excessive pressure on valve DO NOT attempt to operate damaged valve. ■ Clear area of personnel and move upwind. ■ Alert Fire Brigade and tell them location and nature of hazard. ■ May be violently or explosively reactive. ■ Wear breathing apparatus plus protective gloves. ■ Prevent, by any means available, spillage from entering drains or water courses ■ No smoking, naked lights or ignition sources. ■ Increase ventilation. ■ Stop leak if safe to do so. ■ Water spray or fog may be used to disperse / absorb vapour. ■ Absorb or cover spill with sand, earth, inert materials or vermiculite. ■ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. ■ Undamaged cans should be gathered and stowed safely. ■ Collect residues and seal in labelled drums for disposal. ■ Remove leaking cylinders to a safe place if possible. ■ Release pressure under safe, controlled conditions by opening the valve.
	Personal Protective Equipment advice is contained in Section 8 of the MSDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ■ Avoid all personal contact, including inhalation. ■ Wear protective clothing when risk of exposure occurs. ■ Use in a well-ventilated area. ■ Prevent concentration in hollows and sumps. ■ 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	<ul style="list-style-type: none"> ■ Store below 38 deg. C. ■ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can

Servisol NA1002 PCB Lacquer

- 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	<ul style="list-style-type: none"> ▪ Aerosol dispenser. ▪ Check that containers are clearly labelled.
Storage incompatibility	<ul style="list-style-type: none"> ▪ Avoid reaction with oxidising agents

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION**Control parameters****OCCUPATIONAL EXPOSURE LIMITS (OEL)****INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	toluene	Toluene	191 mg/m3 / 50 ppm	574 mg/m3 / 150 ppm	Not Available	Sk
Australia Exposure Standards	acetone	Acetone	1185 mg/m3 / 500 ppm	2375 mg/m3 / 1000 ppm	Not Available	Not Available
Australia Exposure Standards	hydrocarbon propellant	LPG (liquified petroleum gas)	1800 mg/m3 / 1000 ppm	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
toluene	Toluene	Not Available	Not Available	Not Available
acetone	Acetone	Not Available	Not Available	Not Available
hydrocarbon propellant	Liquified petroleum gas; (L.P.G.)	3,000 ppm	3200 ppm	19000 ppm

Ingredient	Original IDLH	Revised IDLH
toluene	2,000 ppm	500 ppm
acetone	20,000 ppm	2,500 [LEL] ppm
additives nonhazardous	Not Available	Not Available
hydrocarbon propellant	19,000 [LEL] ppm	2,000 [LEL] ppm

MATERIAL DATA

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)

Saturation vapour concentration: 237000 ppm @ 20 C

NOTE: Detector tubes measuring in excess of 40 ppm, are available.

Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.

Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.

A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.

Odour Safety Factor(OSF)

OSF=38 (ACETONE)

Continued...

For toluene:

Odour Threshold Value: 0.16-6.7 (detection), 1.9-69 (recognition)

NOTE: Detector tubes measuring in excess of 5 ppm, are available.

High concentrations of toluene in the air produce depression of the central nervous system (CNS) in humans. Intentional toluene exposure (glue-sniffing) at maternally-intoxicating concentration has also produced birth defects. Foetotoxicity appears at levels associated with CNS narcosis and probably occurs only in those with chronic toluene-induced kidney failure. Exposure at or below the recommended TLV-TWA is thought to prevent transient headache and irritation, to provide a measure of safety for possible disturbances to human reproduction, the prevention of reductions in cognitive responses reported amongst humans inhaling greater than 40 ppm, and the significant risks of hepatotoxic, behavioural and nervous system effects (including impaired reaction time and incoordination). Although toluene/ethanol interactions are well recognised, the degree of protection afforded by the TLV-TWA among drinkers is not known.

Odour Safety Factor(OSF)

OSF=17 (TOLUENE)

NOTE K: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.1%/w/w 1,3-butadiene (EINECS No 203-450-8). - European Union (EU) List of Dangerous Substances (Annex VI).

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 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]

Servisol NA1002 PCB Lacquer

Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ■ 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	<p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ■ 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. <p>BREThERICK: Handbook of Reactive Chemical Hazards.</p>
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:
Servisol PCB Lacquer

Material	CPI
PE/EVAL/PE	A
BUTYL/NEOPRENE	B
PVA	B
SARANEX-23 2-PLY	B
TEFLON	B
BUTYL	C
CPE	C
HYPALON	C
NATURAL RUBBER	C
NEOPRENE	C
NITRILE	C
NITRILE+PVC	C
PVC	C
SARANEX-23	C
VITON	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AX-AUS / Class 1	-	AX-PAPR-AUS / Class 1
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	AX-3	-
100+ x ES	-	Air-line**	-

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Continued...

Appearance	Supplied as an aerosol pack. Contents under PRESSURE . Contains highly flammable hydrocarbon propellant.		
Physical state	Liquid	Relative density (Water = 1)	<1
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	-81	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY 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)	Not Available
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)	>1	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ■ Elevated temperatures. ■ Presence of open flame. ■ Product is considered stable. ■ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Common, generalised symptoms associated with toxic gas inhalation include:</p> <ul style="list-style-type: none"> ■ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures; ■ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest;
----------------	--

- cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest;
- gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain.

Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination

Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.

WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.

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

Systemic effects of acetone inhalation exposure include central nervous system depression, light-headedness, incoherent speech, ataxia, stupor, hypotension, tachycardia, metabolic acidosis, hyperglycaemia and ketosis. Rarely, convulsions and tubular necrosis may be evident. Other symptoms of exposure may include restlessness, headache, vomiting, low blood-pressure and rapid and irregular pulse, eye and throat irritation, weakness of the legs and dizziness. Inhalation of high concentrations may produce dryness of the mouth and throat, nausea, uncoordinated movement, loss of coordinated speech, drowsiness and, in severe cases, coma. Inhalation of acetone vapours over long periods causes irritation of the respiratory tract, coughing and headache. Rats exposed to 52200 ppm vapour for 1 hour showed clear signs of narcosis; fatalities occurred at 126600 ppm.

Hydrocarbons may sensitise the heart to adrenalin and other circulatory catecholamines; as a result cardiac arrhythmias and ventricular fibrillation may occur. Abrupt collapse may produce traumatic injury. Central nervous system (CNS) depression may be evident early. Symptoms of moderate poisoning may include giddiness, headache, dizziness and nausea. Serious poisonings may result in respiratory depression and may be fatal.

The paraffin gases C1-4 are practically non-toxic below their lower flammability limits (18000-50000 ppm). Above this level, incidental effects include CNS depression and irritation but these are reversible upon cessation of the exposure. The C3 and iso-C5 hydrocarbons show increasing narcotic properties; branching of the chain also enhances the effect. The C4 hydrocarbons appear to be more highly neurotoxic than the C3 and C5 members. Several fatalities due to voluntary inhalation of butane have been reported, possibly due to central, respiratory and circulatory effects resulting from anaesthesia, laryngeal oedema, chemical pneumonia or the combined effects of cardiac toxicity and increased sympathomimetic effects.

Inhalation of petroleum gases may produce narcosis, due in part to olefinic impurities. Displacement of oxygen in the air may cause cyanosis. If present in sufficient quantity these gases may reduce the oxygen level to below 18% producing asphyxiation. Symptoms include rapid respiration, mental dullness, lack of coordination, poor judgement, nausea and vomiting. The onset of cyanosis may lead to unconsciousness and death.

Ingestion

Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage 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

Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis

Skin Contact

The material may accentuate any pre-existing dermatitis condition

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.

The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either

	<ul style="list-style-type: none"> ■ 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. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
<p style="text-align: center;">Eye</p>	<p>Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p> <p>The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.</p> <p>Evidence exists, or practical experience predicts, that the material may cause severe 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. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants 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.</p> <p>The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration</p>
<p style="text-align: center;">Chronic</p>	<p>Harmful: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Principal route of occupational exposure to the gas is by inhalation.</p> <p>Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.</p> <p>Chronic toluene habituation occurs following intentional abuse (glue sniffing) or from occupational exposure. Ataxia, incoordination and tremors of the hands and feet (as a consequence of diffuse cerebral atrophy), headache, abnormal speech, transient memory loss, convulsions, coma, drowsiness, reduced colour perception, frank blindness, nystagmus (rapid, involuntary eye-movements), hearing loss leading to deafness and mild dementia have all been associated with chronic abuse. Peripheral nerve damage, encephalopathy, giant axonopathy electrolyte disturbances in the cerebrospinal fluid and abnormal computer tomographic (CT scans) are common amongst toluene addicts. Although toluene abuse has been linked with kidney disease, this does not commonly appear in cases of occupational toluene exposures. Cardiac and haematological toxicity are however associated with chronic toluene exposures. Cardiac arrhythmia, multifocal and premature ventricular contractions and supraventricular tachycardia are present in 20% of patients who abused toluene-containing paints. Previous suggestions that chronic toluene inhalation produced human peripheral neuropathy have been discounted. However central nervous system (CNS) depression is well documented where blood toluene exceeds 2.2 mg%. Toluene abusers can achieve transient circulating concentrations of 6.5 %. Amongst workers exposed for a median time of 29 years, to toluene, no subacute effects on neurasthenic complaints and psychometric test results could be established.</p> <p>The prenatal toxicity of very high toluene concentrations has been documented for several animal species and man. Malformations indicative of specific teratogenicity have not generally been found. Neonatal toxicity, described in the literature, takes the form of embryo death or delayed foetal growth and delayed skeletal system development. Permanent damage of children has been seen only when mothers have suffered from chronic intoxication as a result of "sniffing".</p>

<p>Servisol PCB Lacquer</p>	<p style="text-align: center;">TOXICITY</p> <p style="text-align: center;">Not Available</p>	<p style="text-align: center;">IRRITATION</p> <p style="text-align: center;">Not Available</p>
------------------------------------	---	---

toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation (rat) LC50: >26700 ppm/1hd ^[2]	Eye (rabbit):0.87 mg - mild
	Inhalation (rat) LC50: 49 mg/L/4H ^[2]	Eye (rabbit):100 mg/30sec - mild
acetone	Oral (rat) LD50: 636 mg/kg ^[2]	Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 20000 mg/kg ^[2]	Eye (human): 500 ppm - irritant
hydrocarbon propellant	Inhalation (rat) LC50: 50.1 mg/L/8 hr ^[2]	Eye (rabbit): 20mg/24hr -moderate
	Oral (rat) LD50: 5800 mg/kgE ^[2]	Eye (rabbit): 3.95 mg - SEVERE
		Skin (rabbit): 500 mg/24hr - mild
		Skin (rabbit):395mg (open) - mild
hydrocarbon propellant	TOXICITY	IRRITATION
	Inhalation (mouse) LC50: >15.6<17.9 mm/2 h ^[1]	Not Available
	Inhalation (mouse) LC50: 410000 ppm2 h ^[1]	
	Inhalation (rat) LC50: >570000<17.9 ppm15 min ^[1]	
	Inhalation (rat) LC50: >800000 ppm15 min ^[1]	
	Inhalation (rat) LC50: 1354.944 mg/L15 min ^[1]	
	Inhalation (rat) LC50: 1355 mg/l15 min ^[1]	
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's msds. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

Servisol PCB Lacquer	<p>For toluene:</p> <p>Acute Toxicity</p> <p>Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.</p> <p>Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.</p> <p>Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.</p> <p>Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.</p> <p>Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death</p> <p>Toluene can also strip the skin of lipids causing dermatitis</p> <p>Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days</p> <p>Subchronic/Chronic Effects:</p> <p>Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.</p> <p>Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin. Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L</p> <p>Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the</p>
----------------------	---

Servisol NA1002 PCB Lacquer

lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .

Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.

Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy

Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m³. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.

Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .

Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .

Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.

for acetone:

The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m³ and in rats at 26,100 mg/m³. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m³ for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m³, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.

The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m³ have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m³ were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m³ or greater.

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.

For toluene:

Acute Toxicity

Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.

Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.

Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.

Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.

Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils,

TOLUENE

Continued...

convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death
Toluene can also strip the skin of lipids causing dermatitis

Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days

Subchronic/Chronic Effects:

Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.

Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin. Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L

Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) . **Developmental/Reproductive Toxicity**

Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.

Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy

Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m³. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.

Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .

Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .

Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.

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.

for acetone:

The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m³ and in rats at 26,100 mg/m³. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m³ for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m³, respectively. Lifetime dermal

ACETONE

	<p>carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.</p> <p>The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m³ have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m³ were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m³ or greater.</p>
<p>HYDROCARBON PROPELLANT</p>	<p>No significant acute toxicological data identified in literature search.</p> <p>for Petroleum Hydrocarbon Gases:</p> <p>In many cases, there is more than one potentially toxic constituent in a refinery gas. In those cases, the constituent that is most toxic for a particular endpoint in an individual refinery stream is used to characterize the endpoint hazard for that stream. The hazard potential for each mammalian endpoint for each of the petroleum hydrocarbon gases is dependent upon each petroleum hydrocarbon gas constituent endpoint toxicity values (LC50, LOAEL, etc.) and the relative concentration of the constituent present in that gas. It should also be noted that for an individual petroleum hydrocarbon gas, the constituent characterizing toxicity may be different for different mammalian endpoints, again, being dependent upon the concentration of the different constituents in each, distinct petroleum hydrocarbon gas.</p> <p>All Hydrocarbon Gases Category members contain primarily hydrocarbons (i.e., alkanes and alkenes) and occasionally asphyxiant gases like hydrogen. The inorganic components of the petroleum hydrocarbon gases are less toxic than the C1 - C4 and C5 - C6 hydrocarbon components to both mammalian and aquatic organisms. Unlike other petroleum product categories (e.g. gasoline, diesel fuel, lubricating oils, etc.), the inorganic and hydrocarbon constituents of hydrocarbon gases can be evaluated for hazard individually to then predict the screening level hazard of the Category members</p> <p>Acute toxicity: No acute toxicity LC50 values have been derived for the C1 -C4 and C5- C6 hydrocarbon (HC) fractions because no mortality was observed at the highest exposure levels tested (~ 5 mg/l) for these petroleum hydrocarbon gas constituents. The order of acute toxicity of petroleum hydrocarbon gas constituents from most to least toxic is: C5-C6 HCs (LC50 > 1063 ppm) > C1-C4 HCs (LC50 > 10,000 ppm) > benzene (LC50 = 13,700 ppm) > butadiene (LC50 = 129,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Repeat dose toxicity: With the exception of the asphyxiant gases, repeated dose toxicity has been observed in individual selected petroleum hydrocarbon gas constituents. Based upon LOAEL values, the order of order of repeated-dose toxicity of these constituents from most toxic to the least toxic is:</p> <p>Benzene (LOAEL .>=10 ppm) >C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) > C5-C6 HCs (LOAEL = 6,625 ppm) > butadiene (LOAEL = 8,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Genotoxicity:</p> <p>In vitro: The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vitro</i> genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian <i>in vitro</i> test systems.</p> <p>In vivo: The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vivo</i> genotoxicity. The exceptions are benzene and 1,3-butadiene, which are genotoxic in <i>in vivo</i> test systems</p> <p>Developmental toxicity: Developmental effects were induced by two of the petroleum hydrocarbon gas constituents, benzene and the C5 -C6 hydrocarbon fraction. No developmental toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for developmental toxicity. Based on LOAEL and NOAEL values, the order of acute toxicity of these constituents from most to least toxic is:</p> <p>Benzene (LOAEL = 20 ppm) > butadiene (NOAEL .>=1,000 ppm) > C5-C6 HCs (LOAEL = 3,463 ppm) > C1-C4 HCs (NOAEL >=5,000 ppm; assumed to be 100% 2-butene) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Reproductive toxicity: Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the the C1-C4 hydrocarbon fraction). No reproductive toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for reproductive toxicity. Based on LOAEL and NOAEL values, the order of reproductive toxicity of these constituents from most to least toxic is:</p> <p>Benzene (LOAEL = 300 ppm) > butadiene (NOAEL .>=6,000 ppm) > C5-C6 HCs (NOAEL .>=6,521 ppm) > C1-C4 HCs (LOAEL = 9,000 ppm; assumed to be 100% isobutane) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen)</p>

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

Legend: ✓ – Data required to make classification available
 ✗ – Data available but does not fill the criteria for classification
 ⊘ – Data Not Available to make classification

REPROTOXIN	toluene	ILO Chemicals in the electronics industry that have toxic effects on reproduction
SKIN	toluene	Australia Exposure Standards - Skin

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
toluene	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
acetone	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
additives nonhazardous	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
hydrocarbon propellant	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (*Palaemonetes pugio*) and brown shrimp (*Penaeus aztecus*) was dimethylnaphthalenes > methylnaphthalenes > naphthalenes.

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

For ketones:

Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds

Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrate. The higher molecular weight ketones do not form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions

Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH⁻) forming a carbanion intermediate that may react with other organic substrates (e.g., ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable. Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.

For toluene:

log Kow : 2.1-3

log Koc : 1.12-2.85

Koc : 37-260

log Kom : 1.39-2.89

Half-life (hr) air : 2.4-104

Half-life (hr) H₂O surface water : 5.55-528

Half-life (hr) H₂O ground : 168-2628

Half-life (hr) soil : <48-240

Henry's Pa m³/mol: 518-694

Henry's atm m³/mol: 5.94E-03

BOD 5 : 0.86-2.12, 5%

COD : 0.7-2.52, 21-27%

ThOD : 3.13

BCF : 1.67-380

log BCF : 0.22-3.28

Environmental fate:

Transport: The majority of toluene evaporates to the atmosphere from the water and soil. It is moderately retarded by adsorption to soils rich in organic material (Koc = 259), therefore, transport to ground water is dependent on the soil composition. In unsaturated topsoil containing organic material, it has been estimated that 97% of the toluene is adsorbed to the soil and only about 2% is in the soil-water phase and transported with flowing groundwater.

There is little retardation in sandy soils and 2-13% of the toluene was estimated to migrate with flowing water; the remainder was volatilised, biodegraded, or unaccounted for. In saturated deep soils with no soil-air phase, about 48% may be transported with flowing groundwater.

Transformation/Persistence:

Air - The main degradation pathway for toluene in the atmosphere is reaction with photochemically produced hydroxyl radicals. The estimated atmospheric half life for toluene is about 13 hours. Toluene is also oxidised by reactions with atmospheric nitrogen dioxide, oxygen, and ozone, but these are minor degradation pathways. Photolysis is not considered a significant degradative pathway for toluene

Soil - In surface soil, volatilisation to air is an important fate process for toluene. Biodegradation of toluene has been demonstrated in the laboratory to occur with a half life of about 1 hour. In the environment, biodegradation of toluene to carbon dioxide occurs with a typical half life of 1-7 days.

Servisol NA1002 PCB Lacquer

Water - An important fate process for toluene is volatilization, the rate of which depends on the amount of turbulence in the surface water. The volatilisation of toluene from static water has a half life of 1-16 days, whereas from turbulent water the half life is 5-6 hours. Degradation of toluene in surface water occurs primarily by biodegradation with a half life of less than one day under favorable conditions (presence of microorganisms, microbial adaptation, and optimum temperature). Biodegradation also occurs in shallow groundwater and in salt water at a reduced rate). No data are available on anaerobic degradation of toluene in deep ground water conditions where aerobic degradation would be minimal.

Biota - Bioaccumulation in most organisms is limited by the metabolism of toluene into more polar compounds that have greater water solubility and a lower affinity for lipids. Bioaccumulation in the food chain is predicted to be low.

Ecotoxicity:

Toluene has moderate acute toxicity to aquatic organisms; several toxicity values are in the range of greater than 1 mg/L and 100 mg/L.

Fish LC50 (96 h): fathead minnow (*Pimephales promelas*) 12.6-72 mg/l; *Lepomis macrochirus* 13-24 mg/l;

guppy (*Poecilia reticulata*) 28.2-59.3 mg/l; channel catfish (*Ictalurus punctatus*) 240 mg/l; goldfish (*Carassius auratus*): 22.8-57.68 mg/l

Crustaceans LC50 (96 h): grass shrimp (*Palaemonetes pugio*) 9.5 ppm, crab larvae stage 4 (*Cancer magister*) 28 ppm; shrimp (*Crangon franciscorum*) 4.3 ppm; daggerblade grass shrimp (*Palaemonetes pugio*) 9.5 mg/l

Algae EC50 (24 h): green algae (*Chlorella vulgaris*) 245 mg/l (growth); (72 h) green algae (*Selenastrum capricornutum*) 12.5 mg/l (growth)

DO NOT discharge into sewer or waterways.

for acetone:

log Kow: -0.24

Half-life (hr) air: 312-1896

Half-life (hr) H₂O surface water: 20

Henry's atm m³/mol: 3.67E-05

BOD 5: 0.31-1.76, 46-55%

COD: 1.12-2.07

ThOD: 2.2

BCF: 0.69

Environmental fate:

Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water.

In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.

Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.

Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.

Acetone does not concentrate in the food chain.

Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period.

Drinking Water Standard: none available.

Soil Guidelines: none available.

Air Quality Standards: none available.

Ecotoxicity:

Testing shows that acetone exhibits a low order of toxicity

Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l Bird

LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l

Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l

Aquatic invertebrate 2100 - 16700 mg/l

Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephesia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m³. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
toluene	LOW (BCF = 90)
acetone	LOW (BCF = 69)

Mobility in soil

Ingredient	Mobility
------------	----------

toluene	LOW (KOC = 268)
acetone	HIGH (KOC = 1.981)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▪ DO NOT allow wash water from cleaning or process equipment to enter drains. ▪ It may be necessary to collect all wash water for treatment before disposal. ▪ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▪ Where in doubt contact the responsible authority. ▪ Consult State Land Waste Management Authority for disposal. ▪ Discharge contents of damaged aerosol cans at an approved site. ▪ Allow small quantities to evaporate. ▪ DO NOT incinerate or puncture aerosol cans. ▪ Bury residues and emptied aerosol cans at an approved site.
------------------------------	---

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	Not Applicable	
Transport hazard class(es)	IMDG Class	2.1
	IMDG Subrisk	Not Applicable
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	toluene	Y

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

toluene(108-88-3) 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"
acetone(67-64-1) is found on the following regulatory lists	"Australia Exposure Standards", "Australia Inventory of Chemical Substances (AICS)", "Australia Hazardous Substances Information System - Consolidated Lists"
hydrocarbon propellant(68476-85-7.) is found on the following regulatory lists	"Australia Exposure Standards", "Australia Inventory of Chemical Substances (AICS)", "Australia Hazardous Substances Information System - Consolidated Lists"

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
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****Ingredients with multiple cas numbers**

Name	CAS No
------	--------

hydrocarbon propellant	68476-85-7., 68476-86-8.
------------------------	--------------------------

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

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.

This document is copyright. Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH. TEL (+61 3) 9572 4700.