

SILVER ZINC

Chemwatch Independent Material Safety Data Sheet

Issue Date: 15-Nov-2011

A317LP

CHEMWATCH 4770-25

Version No:2.0

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Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME

SILVER ZINC

PROPER SHIPPING NAME

AEROSOLS

PRODUCT USE

■ Used according to manufacturer's directions.

Application is by spray atomisation from a hand held aerosol pack.

SUPPLIER

Company: Dy- Mark Pty Ltd

Address:

89 Formation Street

Wacol

QLD, 4076

Australia

Telephone: +61 7 3271 2222

Emergency Tel: **0403 186 708**

Fax: +61 7 3271 2751

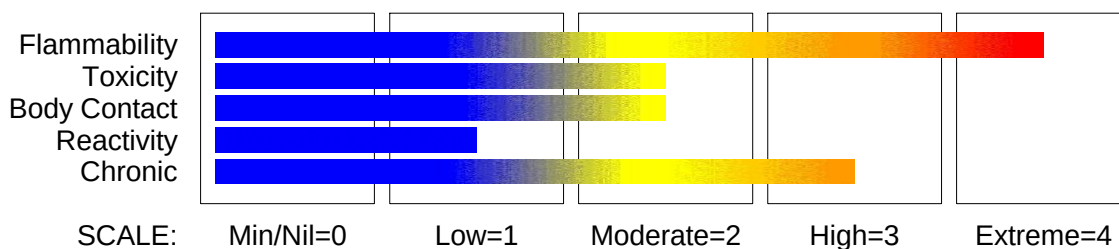
Email: info@dymark.com.au

Section 2 - HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE

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

CHEMWATCH HAZARD RATINGS



RISK

Risk Codes

R12

R44

R48/20

R52/53

R61(2)

R67

Risk Phrases

- Extremely flammable.

- Risk of explosion if heated under confinement.

- Harmful: danger of serious damage to health by prolonged exposure through inhalation.

- Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

- May cause harm to the unborn child.

- Vapours may cause drowsiness and dizziness.

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Section 2 - HAZARDS IDENTIFICATION

SAFETY

Safety Codes

Safety Codes	Safety Phrases
S01	• Keep locked up.
S16	• Keep away from sources of ignition. No smoking.
S23	• Do not breathe gas/fumes/vapour/spray.
S24	• Avoid contact with skin.
S25	• Avoid contact with eyes.
S36	• Wear suitable protective clothing.
S38	• In case of insufficient ventilation, wear suitable respiratory equipment.
S37	• Wear suitable gloves.
S39	• Wear eye/face protection.
S51	• Use only in well ventilated areas.
S09	• Keep container in a well ventilated place.
S53	• Avoid exposure - obtain special instructions before use.
S401	• To clean the floor and all objects contaminated by this material, use water and detergent.
S07	• Keep container tightly closed.
S35	• This material and its container must be disposed of in a safe way.
S13	• Keep away from food, drink and animal feeding stuffs.
S26	• In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.
S60	• This material and its container must be disposed of as hazardous waste.

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
toluene	108-88-3	10-30
acetone	67-64-1	10-30
resin, proprietary		10-30
filler		1-10
additives		1-10
dibutyl phthalate	84-74-2	<1
hydrocarbon propellant	68476-85-7.	10-30
dimethyl ether	115-10-6	10-30

Section 4 - FIRST AID MEASURES

SWALLOWED

- - 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.

EYE

- If aerosols come in contact with the eyes:
 - Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water.
 - Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
 - Transport to hospital or doctor without delay.
 - Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

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Section 4 - FIRST AID MEASURES

SKIN

- If solids or aerosol mists are deposited upon the skin:
 - Flush skin and hair with running water (and soap if available).
 - Remove any adhering solids with industrial skin cleansing cream.
 - DO NOT use solvents.
 - Seek medical attention in the event of irritation.

INHALED

- If aerosols, fumes or combustion products are inhaled:
 - Remove to fresh air.
 - Lay patient down. Keep warm and rested.
 - Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
 - If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
 - Transport to hospital, or doctor.

NOTES TO PHYSICIAN

- For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:
 - Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
 - Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ 50 mm Hg) should be intubated.
 - Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
 - A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
 - Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
 - Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology].
- Treat symptomatically.
for lower alkyl ethers:

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- A low-stimulus environment must be maintained.
- Monitor and treat, where necessary, for shock.
- Anticipate and treat, where necessary, for seizures.
- DO NOT use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension without signs of hypovolaemia may require vasopressors.
- Treat seizures with diazepam.

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Section 4 - FIRST AID MEASURES

- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.

- Ethers may produce anion gap acidosis. Hyperventilation and bicarbonate therapy might be indicated.

- Haemodialysis might be considered in patients with impaired renal function.

- Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994.

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

■ SMALL FIRE:

- Water spray, dry chemical or CO2

LARGE FIRE:

- Water spray or fog.

FIRE FIGHTING

■ FOR FIRES INVOLVING MANY GAS CYLINDERS:

- To stop the flow of gas, specifically trained personnel may inert the atmosphere to reduce oxygen levels thus allowing the capping of leaking container(s).

- Reduce the rate of flow and inject an inert gas, if possible, before completely stopping the flow to prevent flashback.

- DO NOT extinguish the fire until the supply is shut off otherwise an explosive re-ignition may occur.

- If the fire is extinguished and the flow of gas continues, used increased ventilation to prevent build-up, of explosive atmosphere.

- Use non-sparking tools to close container valves.

- Be CAUTIOUS of a Boiling Liquid Evaporating Vapour Explosion, BLEVE, if fire is impinging on surrounding containers.

- Direct 2500 litre/min (500 gpm) water stream onto containers above liquid level with the assistance remote monitors.

- 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.

When any large container (including road and rail tankers) is involved in a fire, consider evacuation by 100 metres in all directions.

FIRE/EXPLOSION HAZARD

■ - Liquid and vapour are highly flammable.

- Severe fire hazard when exposed to heat or flame.

- Vapour forms an explosive mixture with air.

- Severe explosion hazard, in the form of vapour, when exposed to flame or spark.

- Vapour may travel a considerable distance to source of ignition.

- Heating may cause expansion or decomposition with violent container rupture.

- Aerosol cans may explode on exposure to naked flames.

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Section 5 - FIRE FIGHTING MEASURES

- Rupturing containers may rocket and scatter burning materials.

- Hazards may not be restricted to pressure effects.

- May emit acrid, poisonous or corrosive fumes.

- On combustion, may emit toxic fumes of carbon monoxide (CO).

Combustion products include: carbon 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.

FIRE INCOMPATIBILITY

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

HAZCHEM

2YE

Section 6 - ACCIDENTAL RELEASE MEASURES

MINOR SPILLS

- - Clean up all spills immediately.

- Avoid breathing vapours and contact with skin and eyes.

- Wear protective clothing, impervious gloves and safety glasses.

- Shut off all possible sources of ignition and increase ventilation.

- Wipe up.

- If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.

- Undamaged cans should be gathered and stowed safely.

MAJOR SPILLS

- - Clear area of personnel and move upwind.

- Alert Fire Brigade and tell them location and nature of hazard.

- May be violently or explosively reactive.

- Wear breathing apparatus plus protective gloves.

- Prevent, by any means available, spillage from entering drains or water courses

- No smoking, naked lights or ignition sources.

- Increase ventilation.

- Stop leak if safe to do so.

- Water spray or fog may be used to disperse / absorb vapour.

- Absorb or cover spill with sand, earth, inert materials or vermiculite.

- If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.

- Undamaged cans should be gathered and stowed safely.

- Collect residues and seal in labelled drums for disposal.

EMERGENCY RESPONSE PLANNING GUIDELINES (ERPG)

The maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour WITHOUT experiencing or developing

life-threatening health effects is:

toluene 1000ppm

irreversible or other serious effects or symptoms which could impair an individual's ability to take protective action is:

toluene 300ppm

other than mild, transient adverse effects without perceiving a clearly defined odour is:

toluene 50ppm

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Section 6 - ACCIDENTAL RELEASE MEASURES

American Industrial Hygiene Association (AIHA)

Ingredients considered according to the following cutoffs

Very Toxic (T+)	$\geq 0.1\%$	Toxic (T)	$\geq 3.0\%$
R50	$\geq 0.25\%$	Corrosive (C)	$\geq 5.0\%$
R51	$\geq 2.5\%$		
else	$\geq 10\%$		

where percentage is percentage of ingredient found in the mixture

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

- - Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- DO NOT enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights or ignition sources.
- Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- DO NOT incinerate or puncture aerosol cans.
- DO NOT spray directly on humans, exposed food or food utensils.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- Use good occupational work practice.
- Observe manufacturer's storing and handling recommendations.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

SUITABLE CONTAINER

- - Aerosol dispenser.
- Check that containers are clearly labelled.

STORAGE INCOMPATIBILITY

- Dimethyl ether:
 - is a peroxidisable gas
 - may be heat and shock sensitive
 - is able to form unstable peroxides on prolonged exposure to air
 - reacts violently with oxidisers, aluminium hydride, lithium aluminium hydride
 - is incompatible with strong acids, metal salts.
- Toluene:
 - reacts violently with strong oxidisers, bromine, bromine trifluoride, chlorine, hydrochloric acid/ sulfuric acid mixture, 1,3-dichloro-5,5-dimethyl-2,4-imidazolidindione, dinitrogen tetroxide, fluorine, concentrated nitric acid, nitrogen dioxide, silver chloride, sulfur dichloride, uranium fluoride, vinyl acetate
 - forms explosive mixtures with strong acids, strong oxidisers, silver perchlorate, tetranitromethane
 - is incompatible with bis-toluenediazo oxide
 - attacks some plastics, rubber and coatings
 - may generate electrostatic charges, due to low conductivity, on flow or agitation.
 - Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances.

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Section 7 - HANDLING AND STORAGE

STORAGE REQUIREMENTS

- - Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can.
- Store in original containers in approved flammable liquid storage area.
- DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
- No smoking, naked lights, heat or ignition sources.
- Keep containers securely sealed. Contents under pressure.
- Store away from incompatible materials.
- Store in a cool, dry, well ventilated area.
- Avoid storage at temperatures higher than 40 deg C.
- Store in an upright position.
- Protect containers against physical damage.
- Check regularly for spills and leaks.
- Observe manufacturer's storing and handling recommendations.

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m ³	STEL ppm	STEL mg/m ³	Notes
Australia Exposure Standards	toluene (Toluene)	50	191	150	574	Sk
Australia Exposure Standards	acetone (Acetone)	500	1185	1000	2375	
Australia Exposure Standards	dibutyl phthalate (Dibutyl phthalate)		5			
Australia Exposure Standards	hydrocarbon propellant (LPG (liquified petroleum gas))	1000	1800			
Australia Exposure Standards	dimethyl ether (Dimethyl ether)	400	760	500	950	

EMERGENCY EXPOSURE LIMITS

Material	Revised	IDLH
toluene	84	500
acetone	265	2, 500 [LEL]
acetone	159	1, 500
dibutyl phthalate 10078	4, 000	
hydrocarbon propellant	0	2, 000 [LEL]

NOTES

Values marked LEL indicate that the IDLH was based on 10% of the lower explosive limit for safety considerations even though the relevant toxicological data indicated that irreversible health effects or impairment of escape existed only at higher concentrations.

MATERIAL DATA

SILVER ZINC:

Not available

TOLUENE:

- 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

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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).

Exposure limits with "skin" notation indicate that vapour and liquid may be absorbed through intact skin. Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation. Contact with eyes and mucous membranes may also contribute to overall exposure and may also invalidate the exposure standard.

ACETONE:

■ 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).

Exposed individuals are reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class A or B.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV- TWA for example) is being reached, even when distracted by working activities
B	26- 550	As " A" for 50- 90% of persons being distracted
C	1- 26	As " A" for less than 50% of persons being distracted
D	0.18- 1	10- 50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As " D" for less than 10% of persons aware of being tested

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DIBUTYL PHTHALATE:

■ Dibutyl phosphate has caused respiratory tract irritation and headache in workers. The TLV-TWA is thought to be protective against these effects although it is possible that the limit is not sufficiently low to prevent sensory effects.

HYDROCARBON PROPELLANT:

■ For butane:

Odour Threshold Value: 2591 ppm (recognition)

Butane in common with other homologues in the straight chain saturated aliphatic hydrocarbon series is not characterised by its toxicity but by its narcosis-inducing effects at high concentrations. The TLV is based on analogy with pentane by comparing their lower explosive limits in air. It is concluded that this limit will protect workers against the significant risk of drowsiness and other narcotic effects.

Odour Safety Factor(OSF)

OSF=0.22 (n-BUTANE).

For propane

Odour Safety Factor(OSF)

OSF=0.16 (PROPANE).

DIMETHYL ETHER:

■ Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.

OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

for dimethyl ether:

The no-effect-level for dimethyl ether is somewhere between 2000 ppm (rabbits) and 50,000 ppm (humans) with possible cardiac sensitisation occurring around 200,000 ppm (dogs). The AIHA has adopted a safety factor of 100 in respect to the 50,000 ppm level in its recommendation for a workplace environmental exposure level (WEEL) which is thought to protect against both narcotic and sensitising effects. This level is consistent with the TLV-TWA of 400 ppm for diethyl ether and should be easily achievable using current technologies. The use of the traditionally allowable excursion of 1.25 to the level of 6.25 ppm is felt to be more than adequate as an upper safe limit of exposure.

Human data:

50,000 ppm (12 mins): Feelings of mild intoxication.

75,000 ppm (12 mins): As above plus slight lack of attenuation.

82,000 ppm (12 mins): Some incoordination, slight blurring of vision

(30 mins): As above plus analgesia of the face and rushing of blood to the face.

100,000 ppm (10-20 mins): Narcotic symptoms; (64 mins): Sickness (assumed to be nausea)

144,000 ppm (36 mins): Unconsciousness.

May act as a simple asphyxiants; these are gases which, when present in high concentrations, reduce the oxygen content in air below that required to support breathing, consciousness and life; loss of consciousness, with death by suffocation may rapidly occur in an oxygen deficient atmosphere.

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CARE: Most simple asphyxiants are odourless or possess low odour and there is no warning on entry into an oxygen deficient atmosphere. If there is any doubt, oxygen content can be checked simply and quickly. It may not be appropriate to only recommend an exposure standard for simple asphyxiants rather it is essential that sufficient oxygen be maintained. Air normally has 21 percent oxygen by volume, with 18 percent regarded as minimum under normal atmospheric pressure to maintain consciousness / life. At pressures significantly higher or lower than normal atmospheric pressure, expert guidance should be sought.

PERSONAL PROTECTION

EYE

- - 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 lens 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].

HANDS/FEET

- - 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.

OTHER

- No special equipment needed when handling small quantities.
 - OTHERWISE:
 - Overalls.
 - Skin cleansing cream.
 - Eyewash unit.
 - Do not spray on hot surfaces.
 - The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton.
 - Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost.
- BRETHERRICK: Handbook of Reactive Chemical Hazards.

RESPIRATOR

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

The local concentration of material, quantity and conditions of use determine the type of personal protective equipment required. For further information consult site specific CHEMWATCH data (if available), or your Occupational Health and Safety Advisor.

ENGINEERING CONTROLS

- Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent

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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.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

■ Supplied as an aerosol pack. Contents under PRESSURE. Contains highly flammable ether propellant. Flammable aerosol; does not mix with water.

PHYSICAL PROPERTIES

Liquid.

Gas.

Does not mix with water.

State	Liquid	Molecular Weight	Not Available
Melting Range (°C)	Not Available	Viscosity	Not Available
Boiling Range (°C)	Not Available	Solubility in water (g/L)	Immiscible
Flash Point (°C)	Not Available	pH (1% solution)	Not Applicable
Decomposition Temp (°C)	Not Available	pH (as supplied)	Not Applicable
Autoignition Temp (°C)	Not Available	Vapour Pressure (kPa)	Not Available
Upper Explosive Limit (%)	Not Available	Specific Gravity (water=1)	Not Available
Lower Explosive Limit (%)	Not Available	Relative Vapour Density (air=1)	Not Available
Volatile Component (%vol)	>50	Evaporation Rate	Not Available
toluene			
log Kow (Sangster 1997):		2.73	
acetone			
log Kow (Prager 1995):		- 0.24	
log Kow (Sangster 1997):		- 0.24	
dibutyl phthalate			
log Kow (Sangster 1997):		4.72	
dimethyl ether			
log Kow (Sangster 1997):		0.1	

Section 10 - STABILITY AND REACTIVITY

CONDITIONS CONTRIBUTING TO INSTABILITY

- - Elevated temperatures.
- Presence of open flame.
- Product is considered stable.
- Hazardous polymerisation will not occur.

For incompatible materials - refer to Section 7 - Handling and Storage.

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POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

■ 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.

Swallowing of the liquid may cause aspiration into the lungs with the risk of chemical pneumonitis; serious consequences may result. (ICSC13733).

Not normally a hazard due to physical form of product.

Considered an unlikely route of entry in commercial/industrial environments.

EYE

■ This material can cause eye irritation and damage in some persons.

Instillation of isoparaffins into rabbit eyes produces only slight irritation.

Not considered to be a risk because of the extreme volatility of the gas.

Eye contact with alkyl ethers (vapour or liquid) may produce irritation, redness and tears.

SKIN

■ 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.

There is some evidence to suggest that this material can cause inflammation of the skin on contact in some persons.

Skin exposure to isoparaffins may produce slight to moderate irritation in animals and humans. Rare sensitisation reactions in humans have occurred.

Spray mist may produce discomfort.

Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression.

Open cuts, abraded or irritated skin should not be exposed to this material.

Entry into the blood-stream, through, for example, cuts, abrasions 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.

INHALED

■ Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by sleepiness, reduced alertness, loss of reflexes, lack of co-ordination, and vertigo.

Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.

There is some evidence to suggest that the material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage.

Inhalation of toxic gases may cause:

- Central Nervous System effects including depression, headache, confusion, dizziness, stupor, coma and seizures;

- respiratory: acute lung swellings, shortness of breath, wheezing, rapid breathing, other symptoms and respiratory arrest;

- heart: collapse, irregular heartbeats and cardiac arrest;

- gastrointestinal: irritation, ulcers, nausea and vomiting (may be bloody), and abdominal pain.

Inhalation of high concentrations of gas/vapour causes lung irritation with coughing and nausea, central nervous depression with headache and dizziness, slowing of reflexes, fatigue and inco-ordination.

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

CHRONIC HEALTH EFFECTS

■ Harmful: danger of serious damage to health by prolonged exposure through inhalation.

This material can cause serious damage if one is exposed to it for long periods. It can be assumed that it contains a substance which can produce severe defects. This has been demonstrated via both short- and long-

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term experimentation.

Ample evidence exists, from results in experimentation, that developmental disorders are directly caused by human exposure to the material.

There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment.

TOXICITY AND IRRITATION

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

HYDROCARBON PROPELLANT:

SILVER ZINC:

■ No significant acute toxicological data identified in literature search.

TOLUENE:

TOXICITY

Oral (human) LDLo: 50 mg/kg

Oral (rat) LD50: 636 mg/kg

Inhalation (human) TClO: 100 ppm

Inhalation (man) TClO: 200 ppm

Inhalation (rat) LC50: >26700 ppm/1h

Dermal (rabbit) LD50: 12124 mg/kg

■ The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

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, 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 sensitizer 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,

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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.

ACETONE:

TOXICITY

Oral (man) TDLo: 2857 mg/kg

Oral (rat) LD50: 5800 mg/kg

Inhalation (human) TCLo: 500 ppm

Inhalation (man) TCLo: 12000 ppm/4 hr

Inhalation (man) TCLo: 10 mg/m³/6 hr

Inhalation (rat) LC50: 50100 mg/m³/8 hr

Dermal (rabbit) LD50: 20000 mg/kg

■ The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
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

IRRITATION

Eye (human): 500 ppm - Irritant

Eye (rabbit): 3.95 mg - SEVERE

Eye (rabbit): 20mg/24hr - Moderate

Skin (rabbit):395mg (open) - Mild

Skin (rabbit): 500 mg/24hr - Mild

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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.

DIBUTYL PHTHALATE:

TOXICITY

Oral (human) TDLo: 140 mg/kg

Oral (rat) LD50: 8000 mg/kg

Inhalation (rat) LD50: 4250 mg/m³

■ For dibutyl phthalate (DBP):

IRRITATION

Nil Reported

In studies on rats, DBP is absorbed through the skin, although in in vitro studies human skin has been found to be less permeable than rat skin to this compound. Studies in laboratory animals indicate that DBP is rapidly absorbed from the gastrointestinal tract, distributed primarily to the liver and kidneys of rats and excreted in urine as metabolites following oral or intravenous administration. Following inhalation, it was consistently detected at low concentrations in the brain. Available data indicate that in rats, following ingestion, DBP is metabolised by nonspecific esterases mainly in the small intestine to yield mono- n-butyl phthalate (MBP) with limited subsequent biochemical oxidation of the alkyl side chain of MBP. MBP is stable and resistant to hydrolysis of the second ester group. Accumulation has not been observed in any organ. The profile of effects following exposure to DBP is similar to that of other phthalate esters, which, in susceptible species, can induce hepatomegaly, increased numbers of hepatic peroxisomes, foetotoxicity, teratogenicity and testicular damage.

Acute toxicity: The acute toxicity of DBP in rats and mice is low. Signs of acute toxicity in laboratory animals include depression of activity, laboured breathing and lack of coordination. In a case of accidental poisoning of a worker who ingested approximately 10 grams of DBP, recovery was gradual within two weeks and complete after 1 month.

On the basis of limited available data in animal species, DBP appears to have little potential to irritate skin or eyes or to induce sensitization. In humans, a few cases of sensitization after exposure to DBP have been reported, although this was not confirmed in controlled studies of larger numbers of individuals reported only in secondary accounts

Repeat dose toxicity: In short-term repeated-dose toxicity studies, effects at lowest levels in rats after oral administration for 5 to 21 days included peroxisome proliferation and hepatomegaly at doses of 420 mg/kg body weight per day or more. In longer-term studies, the effects in rats observed following ingestion of DBP for periods up to 7 months included reduced rate of weight gain at doses of 250 mg/kg body weight per day or more. Increase in relative liver weight has been observed at doses of 120 mg/kg body weight or more.

Peroxisomal proliferation with increased peroxisomal enzyme activity has been observed at doses of 279 mg/kg body weight per day or more. Necrotic hepatic changes in Wistar rats have been reported at doses of 250 mg/kg body weight per day or more but not in F-344 or Sprague-Dawley rats exposed to up to 2500 mg/kg body weight per day. Alteration in testicular enzymes and degeneration of testicular germinal cells of rats have been observed at doses of 250 and 571 mg/kg body weight per day. There are considerable species differences in effects on the testes following exposure to DBP, minimal effects being observed in mice and hamsters at doses as high as 2000 mg/kg body weight per day. In mice, effects on body and organ weights and histological alterations in the liver indicative of metabolic stress have been reported in a recent subchronic bioassay, for which the no-observed-effect-level (NOEL) was 353 mg/kg body weight per day.

Developmental toxicity: . In a continuous breeding protocol, which included cross-over mating and offspring assessment phases, rats were exposed to 0, 1000, 5000 or 10 000 mg DBP/kg in the diet (equivalent to 0, 66, 320 and 651 mg/kg body weight per day). In the first generation the reduction in pup weight in the mid-dose group, in the absence of any adverse effect on maternal weight, could be regarded as a developmental toxicity effect. There was also a significant reduction of live litter numbers at all three dose levels. The effects

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in the second generation were more severe, with reduced pup weight in all groups including the low-dose group, structural defects (such as prepuccial/ penile malformations, seminiferous tubule degeneration, and absence or underdevelopment of the epididymides) in the mid- and high-dose groups, and severe effects on spermatogenesis in the high-dose group that were not seen in the parent animals. These results suggest that the adverse effects of DBP are more marked in animals exposed during development and maturation than in animals exposed as adults only. No clear NOEL was established in this study. The lowest-observed- adverse-effect-level (LOAEL) was considered to be 66 mg/kg body weight per day. The available studies show that DBP generally induces foetotoxic effects in the absence of maternal toxicity. Available data also indicate that DBP is teratogenic at high doses and that susceptibility to teratogenesis varies with developmental stage and period of administration. In mice, DBP caused dose-dependent increases in the number of resorptions and dead fetuses at oral doses of 400 mg/kg body weight per day or more. Dose-dependent decreases in fetal weights and number of viable litters were also observed in mice at these doses. Adequate carcinogenesis bioassays for DBP have not been conducted. The weight of the available evidence indicates that DBP is not genotoxic.

The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited organelles in the cytoplasm that are found in the cells of animals, plants, fungi, and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Animal studies have shown that peroxisome proliferators clearly cause cancer, especially of the liver.

Available data indicate that phthalate esters are minimally toxic by swallowing, inhalation and skin contact. Repeated exposure may result in weight gain, liver enlargement and induction of liver enzymes. They may also cause shrinking of the testicles and other structural malformations. They may reduce male and female fertility and number of live births, according to animal testing.

HYDROCARBON PROPELLANT:

- inhalation of the gas.

DIMETHYL ETHER:

TOXICITY

Inhalation (rat) LC50: 308000 mg/m³

IRRITATION

Nil Reported

CARCINOGEN

Toluene	International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group	3
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REPROTOXIN

toluene	ILO Chemicals in the electronics industry that have toxic effects on reproduction	Reduced fertility or sterility
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SKIN

toluene	Australia Exposure Standards - Skin	Notes	Sk
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Section 12 - ECOLOGICAL INFORMATION

ACETONE:

DIBUTYL PHTHALATE:

HYDROCARBON PROPELLANT:

DIMETHYL ETHER:

TOLUENE:

- DO NOT discharge into sewer or waterways.

TOLUENE:

- For Toluene:

log Kow : 2.1-3;

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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 : 5.94;
E-03BOD 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.

Atmospheric Fate: The majority of toluene evaporates to the atmosphere from the water and soil. 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 oxidized 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.

Terrestrial Fate: Toluene is moderately retarded by adsorption to soils rich in organic material, therefore, transport to ground water is dependent on 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 volatilized, biodegraded, or unaccounted for. In saturated deep soils with no soil-air phase, about 48% may be transported with flowing groundwater. In surface soil, volatilization to air is an important fate process for toluene. In the environment, biodegradation of toluene to carbon dioxide occurs with a typical half life of 1-7 days.

Aquatic Fate: An important fate process for toluene is volatilization, the rate of which depends on the amount of turbulence in the surface water. The volatilization 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.

Ecotoxicity: Bioaccumulation in the food chain is predicted to be low. Toluene has moderate acute toxicity to aquatic organisms. Toluene is, on the average, slightly toxic to fathead minnow, guppies and goldfish and not acutely toxic to bluegill or channel catfish and crab. Toluene, on the average, is slightly toxic to crustaceans specifically, shrimp species including grass shrimp and daggerblade grass shrimp. Toluene has a negative effect on green algae during their growth phase.

ACETONE:

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

Aquatic Fate: Hydrolysis of ketones in water is thermodynamically favourable only for low molecular weight ketones. Reactions with water are reversible with no permanent change in the structure of the ketone substrate. Ketones are stable to water under ambient environmental conditions. When pH levels are greater than 10, condensation reactions can occur which produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavourable. Based on its reactions in air, it seems likely that ketones undergo photolysis in water.

Terrestrial Fate: It is probable that ketones will be biodegraded by micro-organisms in soil and water.

Ecotoxicity: Ketones are unlikely to bioconcentrate or biomagnify.

For Acetone: l

og 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: 2.2BCF: 0.69.

Environmental Fate: The relatively long half-life allows acetone to be transported long distances from its emission source.

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Atmospheric Fate: Acetone preferentially locates in the air compartment when released to the environment. 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. Air Quality Standards: none available.

Terrestrial Fate: Very little acetone is expected to reside in soil, biota, or suspended solids and has low propensity for soil absorption and a high preference for moving through the soil and into the ground water. Acetone released to soil volatilizes although some may leach into the ground where it rapidly biodegrades.

Soil Guidelines: none available.

Aquatic Fate: A substantial amount of acetone can also be found in water. Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours Drinking Water Standard: none available.

Ecotoxicity: Acetone does not concentrate in the food chain, is minimally toxic to aquatic life and is considered to be readily biodegradable. Testing shows that acetone exhibits a low order of toxicity for brook trout, fathead minnow, Japanese quail, ring-neck pheasant and water fleas. Low toxicity for aquatic invertebrates. For aquatic plants, NOEC: 5400-7500 mg/L. Acetone vapours were shown to be relatively toxic to flour beetle and flour moths and their eggs. 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. Mild to moderate toxicity occurred in bacteria exposed to acetone for 6-4 days however, overall data indicates a low degree of toxicity for acetone. The only exception to these findings was the results obtained with the flagellated protozoa (*Entosiphon sulcatum*).

DIBUTYL PHTHALATE:

■ Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For Transitional Phthalate Esters:

Aquatic Fate: Phthalates have no associated acute or chronic aquatic toxicity. This cut-off in acute toxicity is due to the concentration causing acute toxicity being higher than the water solubility of the phthalate ester. The same situation exists for those phthalates which are more non-polar (higher carbon number) than dihexyl phthalate. The lower molecular weight transitional phthalate esters are more water soluble than the remaining higher molecular weight transitional phthalates (dihexyl and higher) and causes acute and chronic aquatic toxicity below 1 mg/L. There is an apparent cut-off in acute toxicity at dihexyl phthalate and higher. No effects at the limit of water solubility for dihexyl phthalate and higher are predicted.

Terrestrial Fate: Compounds with higher molecular weights and decreased solubility are strongly adsorbed to soil and to suspended particulate matter in water and are not very accessible to biochemical processes leading to degradation.

Ecotoxicity: Diheptyl phthalate (DHP) and di-iso-heptyl phthalate (DIHP) are not acutely toxic to fish, invertebrates, or algae. Phthalates with short alkyl chains phthalates are more toxic; however, are not persistent. Compounds with higher molecular weights and low solubility are strongly adsorbed to soil and to suspended particulate matter in water; therefore, they are not very accessible to biochemical processes leading to degradation.

for dibutyl phthalate (DBP):

log Kow : 4.72

Koc : 160-6400

Half-life (hr) air : 18

Half-life (hr) H₂O surface water : 1824

Henry's atm m³ /mol: 4.60E-07

BOD 5 if unstated: 0.43

ThOD : 2.24

BCF : 5000

log BCF : 1.07-1.5

Environmental Fate:

DBP is relatively non-persistent in air and surface waters, and has a half-life in these compartments of only a few days. Complete biodegradation of DBP is rapid under aerobic conditions but much slower under anaerobic conditions. For soil, similar half-lives to air and water have been predicted; however, studies suggest that DBP may be more persistent in soil. DBP would be expected to bioaccumulate as a result of its high octanol-water partition coefficient. However, it is quite readily metabolised in fish and, consequently, bioconcentration factors tend to be lower than predicted. The highest bioconcentration factor, observed was

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590 for the fathead minnow. Biomagnification is unlikely in terrestrial animals, based upon limited data on birds and the rapid metabolism and excretion observed in laboratory mammals.

Ecotoxicity:

Fish - *Salmo gairdneri* LC50 (96h): 1.6 mg/L.

Daphnia Magna EC50 (48 h): 3.4 mg/L.

Algae - *Selenastrum capricornutum* EC50 (96h): 0.75 mg/L.

Phytotoxicity: Vapours of DBP at low concentrations can cause serious damage to plants.

Information on the ecotoxicity of DBP includes acute and chronic data for a number of species from various trophic levels in the aquatic environment. For freshwater algae the lowest reported 96-h EC50 was 750 ug DBP/litre. The lowest reported values in acute toxicity tests on aquatic invertebrates were a 96-h LC50 of 750 ug/litre (mysid shrimp) and a 48-h EC50 of 760 ug/litre (midge larvae). In chronic studies, the most sensitive invertebrate species was *Daphnia magna*, with a 21-day NOEC (parent survival) of 500 ug/litre. In a non-standard test with the scud (*Gammarus pulex*) a 10-day LOEC of 500 ug/litre and a NOEC of 100 ug/litre, both based on reduced locomotor activity, were reported.

In acute toxicity tests with fish the lowest reported 96-h LC50 for a freshwater species was 350 ug/litre (yellow perch) and for a marine species 600 ug/litre (sheepshead minnow). The most sensitive chronic study was based on the rainbow trout with a 99-day NOEC (growth) of 100 ug/litre and a 99-day LOEC of 190 ug/litre (growth reduced by about 27%).

The acute toxicity of DBP to birds is low. At average concentrations the risk of DBP to aquatic organisms is low, however the risk is increased in highly polluted rivers.

There is inadequate data to assess the risk of DBP to sediment-dwelling organisms. At current levels of exposure, it can be concluded that the risk to fish-eating birds and mammals is low.

for phthalate esters:

Environmental fate;

Under aerobic and anaerobic conditions, studies reveal that many phthalate esters are degraded by a wide range of bacteria and actinomycetes. Standardized aerobic biodegradation tests with sewage sludge inocula show that within 28 days approximately 50% ultimate degradation occurs. Biodegradation is, therefore, expected to be the dominant pathway in surface soils and sediments. In the atmosphere, photodegradation via free radical attack is the anticipated dominant pathway. The half-life of many phthalate esters is ca. 1 day in the air, from < 1 day to 2 weeks in surface and marine waters, and from < 1 week to several months in soils.

Phthalates are high molecular weight chemicals, and are not expected to partition significantly to air. However for the minor amount that may partition to air, modelled predictions indicate that they would be rapidly oxidised: with a predicted atmospheric oxidation half-life of around 0.52 days. They are expected to react appreciably with other photo oxidative species in the atmosphere, such as O₃. Therefore, it is expected that reactions with hydroxyl radicals will be the most important fate process in the atmosphere for phthalates.

Bioaccumulation of phthalate esters in the aquatic and terrestrial food chain is limited by biotransformation. Most phthalates have experimental bioaccumulation factor (BCFs) and bioconcentration factor (BAFs) below 5000 L/kg, as they are readily metabolised by fish

A study of 18 commercial phthalate esters with alkyl chains ranging from one to 13 carbons found an eight order of magnitude increase in octanol-water coefficients (K_{ow}) and a four order of magnitude decrease in vapor pressure with increasing length. This increase in K_{ow} and decrease in vapor pressure results in increased partitioning of the phthalate esters to suspended solids, soils, sediments, and aerosols

The phthalate esters are distributed throughout the environment ubiquitously. They are found complexed with fulvic acid components of the humic substances in soil and marine and estuarine waters. Fulvic acid appears to act as a solubiliser for the otherwise insoluble ester and serves to mediate its transport and mobilisation in water or immobilisation in soil. Phthalate esters have been found in open ocean environments, in deep sea jelly fish, Atlantic herring and in mackerel. Phthalic ester plasticisers are clearly recognised as general contaminants of almost every soil and water ecosystem. In general they have low acute toxicity but the weight of evidence supporting their carcinogenicity is substantial. Other subtle chronic effects have also been reported. As little as 4 ug/ml in culture medium is lethal to chick embryo heart cells. This concentration is similar to that reached in human blood stored in vinyl plastic bags for as little as one day. As phthalates are present in drinking water and food, concerns have been raised about their long term effects on humans.

Ecotoxicity:

Some phthalates (notably di-2-ethylhexyl phthalate and dibutyl phthalate) may be detrimental to the reproduction of the water flea (*Daphnia magna*), zebra fish and guppies

While phthalates may have very low true water solubilities, they possess the ability to form suspensions

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which may cause adverse effects through physical contact with Daphnia at very low concentrations. Available toxicity and water solubility information suggest that the high molecular weight phthalates, form these suspensions and are able to elicit chronic toxic effects at concentrations of approximately 0.05 mg/L. Therefore, these substances are considered to have the potential to harm aquatic organisms at relatively low concentrations.

HYDROCARBON PROPELLANT:

■ for Petroleum Hydrocarbon Gases:

Environmental fate:

The environmental fate characteristics of petroleum hydrocarbon gases are governed by these physical-chemical attributes. All components of these gases will partition to the air where interaction with hydroxyl radicals is an important fate process. Hydrocarbons having molecular weights represented in these streams are inherently biodegradable, but their tendency to partition to the atmosphere would prevent their biotic degradation in water and soils. However, if higher molecular weight fractions of these streams enter the aquatic or terrestrial environment, biodegradation may be an important fate mechanism.

The majority of components making up hydrocarbon gases typically have low melting and boiling points. They also have high vapor pressures and low octanol/water partition coefficients. The aqueous solubilities of these substances vary, and range from approximately 22 parts per million to several hundred parts per million. The environmental fate characteristics of refinery gases are governed by these physical-chemical attributes. Components of the hydrocarbon gas streams will partition to the air, and photodegradation reactions will be an important fate process for many of the hydrocarbon components. The hydrocarbons in these mixtures are inherently biodegradable, but due to their tendency to partition to the atmosphere, biodegradation is not anticipated to be an important fate mechanisms. However, if released to water or soil, some of the higher molecular weight fractions may become available for microbial attack. The inorganic gases are chemically stable and may be lost to the atmosphere or simply become involved in the environmental recycling of their atoms. Some show substantial water solubility, but their volatility eventually causes these gases to enter the atmosphere.

Substances in Refinery Gases that volatilise to air may undergo a gas-phase oxidation reaction with photochemically produced hydroxyl radicals (OH⁻). Atmospheric oxidation as a result of hydroxyl radical attack is not direct photochemical degradation, but rather indirect degradation. Indirect photodegradation of the hydrocarbon components in Refinery Gases can be an important fate process for these constituents. In general, half lives decrease with increasing carbon chain length. Half lives for this fraction of Refinery Gases ranged from 960 days (methane) to 0.16 days (butadiene). The constituents of the C5- C6 hydrocarbon fraction have photodegradation half-lives of approximately two days.

The hydrocarbon and non-hydrocarbon constituents in Refinery Gases do not contain the functional groups or chemical linkages known to undergo hydrolysis reactions. Therefore hydrolysis will not play an important role in the environmental fate for the components in Refinery Gas streams.

Biodegradation of the hydrocarbon components in refinery gases may occur in soil and water. Gaseous hydrocarbons are widespread in nature and numerous types of microbes have evolved which are capable of oxidizing these substances as their sole energy source. Although volatilization is the predominant behavior for these gases, sufficient aqueous solubility and bioavailability is exhibited by these compounds. The use of gaseous carbon sources for cell growth is common among autotrophic organisms. Higher chain length hydrocarbons typical of naphtha streams also are known to inherently biodegrade in the environment.

Ecotoxicity:

Acute LC/EC50 values for the hydrocarbon components of these gas streams ranged roughly from 1 to 100 mg/L. Although the LC/EC50 data for the individual gases illustrate the potential toxicity to aquatic organisms, aqueous concentrations from releases of these gases would likely not persist in the aquatic environment for a sufficient duration to elicit toxicity. Based on a simple conceptual exposure model analysis, emissions of petroleum hydrocarbon gases to the atmosphere would not likely result in acutely toxic concentrations in adjacent water bodies because such emissions will tend to remain in the atmosphere.

Several of the constituents in refinery gases were shown to be highly hazardous to aquatic organisms in laboratory toxicity tests where exposure concentrations can be maintained over time. Hydrogen sulfide was shown to be the most toxic constituent to fish (LC50 ranged 0.007 to 0.2 mg/L) and invertebrates (EC50 ranged 0.022 to 1.07 mg/L), although several LC/EC50 values for ammonia also were below 1 mg/l for these organisms (0.083 to 4.6 mg/L and 0.53 to 22.8 mg/L, respectively).

For isobutane:

Refrigerant Gas: Saturated Hydrocarbons have zero ozone depletion potential (ODP) and will photodegrade under atmospheric conditions. [Calor Gas]

Environmental Fate

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Terrestrial fate: An estimated Koc value of 35 suggests that isobutane will have very high mobility in soil. Its very high Henry's Law constant, 4.08 atm-cu m/mole, (calculated from its vapor pressure and water solubility, high vapor pressure, 2611 mm Hg at 25 deg C, and low adsorptivity to soil indicate that volatilisation will be an important fate process from both moist and dry soil surfaces. Isobutane is biodegradable, especially under acclimated conditions, and may biodegrade in soil.

Aquatic fate: The estimated Koc value suggests that isobutane would not adsorb to sediment and particulate matter in the water column. Additional evidence that isobutane is not removed to sediment has been obtained from microcosm experiments. Isobutane will readily volatilise from water based on its estimated Henry's Law constant of 4.08 atm-cu m/mole. Estimated half-lives for a model river and model lake are 2.2 hr and 3.0 days, respectively. An estimated BCF value of 74 based on the log Kow suggests that isobutane will not bioconcentrate in aquatic organisms.

Results indicate that gas exchange is the dominant removal mechanism for isobutane gases from the water column following a hypothetical input. The volatilisation half-lives for isobutane from the water columns in natural estuaries are estimated to be 4.4 and 6.8 days at 20 and 10 deg C, respectively.

Isobutane also biodegrades in the microcosm at a rate that is slower than for n-butane and falls between propane and ethane in susceptibility. Biodegradation of isobutane initially occurs with a half-lives of 16-26 days at 20 deg C and 33-139 days at 10 deg C, significantly slower than the loss predicted by gas exchange from typical natural estuaries. However, after a lag of 2-4 weeks, the biodegradation rate increases markedly so that in the case of chronic inputs, biodegradation can become the dominant removal mechanism.

Atmospheric fate: Isobutane is a gas at ordinary temperatures. It is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is 6.9 days, assuming a hydroxyl radical concn of 5×10^5 radicals per cubic cm. When isobutane was exposed to sunlight for 6 hr in a tedlar bag filled with Los Angeles air, 6% of the isobutane degraded. The air contained 4529 ppb-C hydrocarbons and 870 ppb of NOX. The tropospheric loss of volatile hydrocarbons such as isobutane by wet and dry deposition are believed to be of minor importance. Indeed, isobutane assimilated into precipitation may evaporate during transport as well as being reemitted into the atmosphere after deposition. Isobutane is a contributor to the production of PAN (peroxyacyl nitrates) under photochemical smog conditions.

For Propane: Koc 460. log

Kow 2.36.

Henry's Law constant of 7.07×10^{-1} atm-cu m/mole, derived from its vapour pressure, 7150 mm Hg, and water solubility, 62.4 mg/L. Estimated BCF: 13.1.

Terrestrial Fate: Propane is expected to have moderate mobility in soil. Volatilization from moist soil surfaces is expected to be an important fate process. Volatilization from dry soil surfaces is based vapor pressure. Biodegradation may be an important fate process in soil and sediment.

Aquatic Fate: Propane is expected to adsorb to suspended solids and sediment. Volatilization from water surfaces is expected and half-lives for a model river and model lake are estimated to be 41 minutes and 2.6 days, respectively. Biodegradation may not be an important fate process in water.

Ecotoxicity: The potential for bioconcentration in aquatic organisms is low.

Atmospheric Fate: Propane is expected to exist solely as a gas in the ambient atmosphere. Gas-phase propane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 14 days and is not expected to be susceptible to direct photolysis by sunlight.

DIMETHYL ETHER:

■ Most ethers are very resistant to hydrolysis, and the rate of cleavage of the carbon-oxygen bond by abiotic processes is expected to be insignificant.

Direct photolysis will not be an important removal process since aliphatic ethers do not absorb light at wavelengths >290 nm.

log Kow: 0.1-0.12

Koc: 14

Half-life (hr) air: 528

Half-life (hr) H₂O surface water: 2.6-30

Henry's atm m³ /mol: 9.78E-04

BCF: 1.7

Bioaccumulation: not sig

processes Abiotic: RxnOH*

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Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
toluene	LOW	MED	LOW	MED
acetone	LOW	HIGH	LOW	HIGH
dibutyl phthalate	LOW	MED	LOW	MED
hydrocarbon propellant	No Data Available	No Data Available		
dimethyl ether	LOW	No Data Available	LOW	HIGH

Section 13 - DISPOSAL CONSIDERATIONS

- - 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 - TRANSPORTATION INFORMATION

Labels Required: FLAMMABLE GAS

HAZCHEM:

2YE (ADG7)

ADG7:

Class or Division	2.1	Subsidiary Risk:	None
UN No.:	1950	Packing Group:	None
Special Provision:	63, 190, 277, 327	Limited Quantity:	See SP 277
Portable Tanks & Bulk Containers - Instruction:	None	Portable Tanks & Bulk Containers - Special Provision:	None
Packagings & IBCs - Packing Instruction:	PP17, PP87, L2	Packagings & IBCs - Special Packing Provision:	P003, LP02

Name and Description: AEROSOLS

Land Transport UNDG:

Class or division	2.1	Subsidiary risk:	None
UN No.:	1950	UN packing group:	None
Shipping Name:AEROSOLS			

Air Transport IATA:

ICAO/IATA Class:	2.1	ICAO/IATA Subrisk:	None
UN/ID Number:	1950	Packing Group:	-
Special provisions:	A145		
Cargo Only			
Packing Instructions:	203	Maximum Qty/Pack:	150 kg
Passenger and Cargo		Passenger and Cargo	

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Packing Instructions:	203	Maximum Qty/Pack:	75 kg
Passenger and Cargo		Passenger and Cargo	
Limited Quantity		Limited Quantity	
Packing Instructions:	Y203	Maximum Qty/Pack:	30 kg G

Shipping Name: AEROSOLS, FLAMMABLE

Maritime Transport IMDG:

IMDG Class:	2	IMDG Subrisk:	SP63
UN Number:	1950	Packing Group:	None
EMS Number:	F- D, S- U	Special provisions:	63 190 277 327 344 959
Limited Quantities:	See SP277		
Shipping Name:	AEROSOLS		

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE None

REGULATIONS

Regulations for ingredients

toluene (CAS: 108-88-3) is found on the following regulatory lists;

toluene (CAS: 108-88-3) is found on the following regulatory lists;

"Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (AQUA/1 to 6 - non-pesticide anthropogenic organics)", "Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (Domestic water supply - organic compounds)", "Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (Aquatic habitat)", "Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (Domestic water supply quality)", "Australia Customs (Prohibited Exports) Regulations 1958 - Schedule 9 Precursor substances - Part 2", "Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Illicit Drug Reagents/Essential Chemicals - Category III", "Australia Inventory of Chemical Substances (AICS)", "Australia National Pollutant Inventory", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6", "IMO IBC Code Chapter 17: Summary of minimum requirements", "IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk", "IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "International Fragrance Association (IFRA) Standards Prohibited", "WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water"

acetone (CAS: 67-64-1) is found on the following regulatory lists;

acetone (CAS: 67-64-1) is found on the following regulatory lists;

"Australia Customs (Prohibited Exports) Regulations 1958 - Schedule 9 Precursor substances - Part 2", "Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Illicit Drug Reagents/Essential Chemicals - Category III", "Australia Inventory of Chemical Substances (AICS)", "Australia National Pollutant Inventory", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5", "FEMA Generally Recognized as Safe (GRAS) Flavoring Substances 23 - Examples of FEMA GRAS Substances with Non-Flavor Functions", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "IMO IBC Code Chapter 18: List of products to which the Code does not apply", "IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances", "International Fragrance Association (IFRA) Survey: Transparency List"

DURCUPAN COMPONENT D: PLASTICIZER (CAS: 84-74-2) is found on the following regulatory lists;

DURCUPAN COMPONENT D: PLASTICIZER (CAS: 84-74-2) is found on the following regulatory lists;

"Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (AQUA/1 to 6 - non-pesticide anthropogenic organics)", "Australia Exposure Standards", "Australia Hazardous Substances", "Australia Inventory of Chemical Substances (AICS)", "Australia National Pollutant Inventory", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "IMO IBC Code Chapter 17: Summary of minimum requirements", "IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk", "International Chemical Secretariat (ChemSec) SIN List (*Substitute It Now!)", "OSPAR List of Substances of Possible Concern"

hydrocarbon propellant (CAS: 68476-85-7,68476-86-8) is found on the following regulatory lists;

hydrocarbon propellant (CAS: 68476-85-7,68476-86-8) is found on the following regulatory lists;

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)"

dimethyl ether (CAS: 115-10-6,157621-61-9) is found on the following regulatory lists;

dimethyl ether (CAS: 115-10-6,157621-61-9) is found on the following regulatory lists;

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia Inventory of Chemical Substances (AICS)", "International Council of Chemical Associations (ICCA) - High Production Volume List"

No data for Silver Zinc (CW: 4770-25)

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Section 16 - OTHER INFORMATION

INGREDIENTS WITH MULTIPLE CAS NUMBERS

Ingredient Name	CAS
hydrocarbon propellant	68476- 85- 7, 68476- 86- 8
dimethyl ether	115- 10- 6, 157621- 61- 9

REPRODUCTIVE HEALTH GUIDELINES

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
toluene	9.6 mg/m3	10	D	NA	-
dibutyl phthalate	0.72 mg/m3	1000	D	NA	-

■ These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.

CR = Cancer Risk/10000; UF = Uncertainty factor:

TLV believed to be adequate to protect reproductive health:

LOD: Limit of detection

Toxic endpoints have also been identified as:

D = Developmental; R = Reproductive; TC = Transplacental carcinogen

Jankovic J., Drake F.: A Screening Method for Occupational Reproductive

American Industrial Hygiene Association Journal 57: 641-649 (1996).

■ Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

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

www.chemwatch.net/references.

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

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This is the end of the MSDS.