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

PRODUCT IDENTIFIER

Product Name	Bright Silver Zinc (Hot Dip in a Can)
Synonyms	A1040D
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	Use according to manufacturer's directions. Application is by spray atomization from a handheld aerosol pack.

DETAILS OF THE SUPPLIER OF THE SAFETY DATA SHEET

Registered company name	Silver Zinc Supplies
Address	2/10 Maiella Street Stapylton Queensland 4207 Australia
Telephone	(07) 3287 4567
Website	silverzinc.com.au
Email	orders@silverzinc.com.au

EMERGENCY CONTACT NUMBER

Association / Organization	Silver Zinc Supplies
Emergency Contact	04000 18006
Other Emergency Contact	Not Available

SECTION 2 HAZARDS IDENTIFICATION

CLASSICATION OF THE SUBSTANCE OR MIXTURE

HAZARDOUS CHEMICAL. DANGEROUS GOODS. ACCORDING TO THE WHS REGULATIONS AND THE ADG CODE.

CHEMWATCH HAZARD RATINGS

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

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

Poisons Schedule	Not Applicable
Classification[1]	Flammable Aerosols Category 1, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Specific target organ toxicity - single exposure Category 3 (narcotic effects)
Legend	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

LABEL ELEMENTS

Hazard Pictogram(s)	
Signal Word	**Danger**

HAZARD STATEMENT (S)

H222	Extremely flammable aserosol.
H312	Harmful in contact with skin.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H336	May cause drowsiness of dizziness.
H411	Toxic to aquatic life with long lasting effects.
AUH044	Risk of explosion if heated under confinement.

PRECAUTIONARY STATEMENTS(S) PREVENTION

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Pressurized container: Do not pierce or burn, even after use.
P261	Avoid breathing mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

PRECAUTIONARY STATEMENT(S) RESPONSE

P305 + P351 + P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P321	Specific treatment (see advice on this label).
P322	Specific measures (see advice on this label).
P362	Take off contaminated clothing and wash before reuse.
P302 + P352	IF ON SKIN: Wash with plenty of water and soap.
P337 + P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P304 + P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P332+P313	Skin irritation occurs: Get medical advice/attention.

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 authorized hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

SUBSTANCES

SEE SECTION BELOW FOR COMPOSITION OF MIXTURES

MIXTURES

CAS No.	% [Weight]	Name
1330-20-7	30-60	xylene
7429-90-5	1-10	aluminium
Not Available	1-10	resin, proprietary
7779-90-0	1-5	zinc phosphate
107-98-2	<1	propylene glycol monomethyl ether - alpha isomer
68476-85-7	10-30	hydrocarbon propellant
115-10-6	10-30	dimethyl ether

SECTION 4 FIRST AID MEASURES

DESCRIPTION OF FIRST AID MEASURES

Eye Contact	If aerosols come in contact with the eyes: • Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. • Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. • Transport to hospital or doctor without delay. • Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If solids or aerosol mists are deposited upon the skin: • Flush skin and hair with running water (and soap if available). • Remove any adhering solids with industrial skin cleansing cream. • DO NOT use solvents. • Seek medical attention in the event of irritation.
Inhalation	If aerosols, fumes or combustion products are inhaled: • Remove to fresh air. • Lay patient down. Keep warm and rested. • Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. • If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. • Transport to hospital, or doctor.
Ingestion	• 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 LOWER ALYL 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.
- 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

FOR ACUTE OR SHORT TERM REPEATED EXPOSURES TO XYLENE:

- Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended.
- The use of charcoal and cathartics is equivocal.
- Pulmonary absorption is rapid with about 60-65% retained at rest.
- Primary threat to life from 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 (pO_2 < 50 mm Hg or pCO_2 > 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.

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	Comments
Methylhippu-ric acids in urine	1.5 gm/gm creatinine 2 mg/min	End of shift Last 4hrs of shift	

SECTION 5 FIREFIGHTING MEASURES

EXTINGUISHING MEDIA

- **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	• Liquid and vapour are highly flammable. • Severe fire hazard when exposed to heat or flame. • Vapour forms an explosive mixture with air. • Severe explosion hazard, in the form of vapour, when exposed to flame or spark. • Vapour may travel a considerable distance to source of ignition. • Heating may cause expansion or decomposition with violent container rupture. • Aerosol cans may explode on exposure to naked flames. • Rupturing containers may rocket and scatter burning materials. • Hazards may not be restricted to pressure effects. • May emit acrid, poisonous or corrosive fumes. • On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: • carbon dioxide (CO2) • other pyrolysis products typical of burning organic material. • Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions. • When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

PERSONAL PRECAUTIONS, PROTECTIVE EQUIPMENT AND EMERGENCY PROCEDURES - SEE SECTION 8
ENVIRONMENTAL PRECAUTIONS - SEE SECTION 12
ADVICE FOR FIREFIGHTERS

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	• 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. Chemical Class: aromatic hydrocarbons For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
Feathers - pillow	1	throw	pitchfork	DGC, RT
cross-linked polymer - particulate	2	shovel	shovel	R, W, SS
cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	3	shovel	shovel	R, I, P
treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I
wood fiber - pillow	4	throw	pitchfork	R, P, DGC, RT
LAND SPILL - MEDIUM				
cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I
sorbent clay - particulate	2	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W,SS, DGC
feathers - pillow	3	throw	skiploader	DGC, RT
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC

- Shut off all possible sources of ignition and increase ventilation.
- Use extreme caution to prevent violent reaction.
- DO NOT enter confined space where gas may have collected.
- Keep area clear until gas has dispersed.
- 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 SDS.

SECTION 7 HANDLING AND STORAGE

PRECAUTIONS FOR SAFE HANDLING

Safe handling	The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid. • DO NOT allow clothing wet with material to stay in contact with skin • 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 SDS. • Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other Information	• Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can • Store in original containers in approved flammable liquid storage area. • DO NOT store in pits, depressions, basements or areas where vapours may be trapped. • No smoking, naked lights, heat or ignition sources. • Keep containers securely sealed. Contents under pressure. • Store away from incompatible materials. • Store in a cool, dry, well ventilated area. • Avoid storage at temperatures higher than 40 deg C. • Store in an upright position. • Protect containers against physical damage. • Check regularly for spills and leaks. • Observe manufacturer's storage and handling recommendations contained within this SDS.

CONDITIONS FOR SAFE STORAGE, INCLUDING ANY INCOMPATIBILITIES

Suitable container	• Aerosol dispenser. • Check that containers are clearly labelled.
Storage Incompatibility	• Reacts with acids producing flammable / explosive hydrogen (H2) gas • Avoid reaction with oxidising agents

LEGEND	
	X — MUST NOT BE STORED TOGETHER 0 — MAY BE STORED TOGETHER WITH SPECIFIC PREVENTIONS + — MAY BE STORED TOGETHER

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

CONTROL PARAMETERS - OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredients	Sampling Time	TWA	STEL	Peak	Notes
Australia Exposure Standards	xylene	Xylene (o-, m-, p- isomers)	80ppm / 350mg / m3	655mg /m3 / 150ppm	Not Available	Not Available
Australia Exposure Standards	aluminium powder coated	Aluminium (metal dust)	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium powder coated	Aluminium (welding fumes) (as Al)	5mg / m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium powder coated	Aluminium, pyro powders (as Al)	5mg / m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether	100 ppm / 369 mg/m3	553 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	hydrocarbon propellant	LPG (liquified petroleum gas)	1000 ppm / 1800 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	Dimethyl ether	Dimethyl ether	400 ppm / 760 mg/m3	950mg / m3 / 500ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredients	Material Name	TEEL-1	TEEL-2	TEEL-3
xylene	Xylenes	Not Available	Not Available	Not Available
zinc phosphate	Zinc phosphate (3:2)	12mg / m3	36mg / m3	220mg / m3
propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether; (Ucar Triol HG-170)	100ppm	160ppm	660ppm
zinc phosphate	Zinc phosphate (3:2)	12mg/m3	36mg/m3	220mg/m3
hydrocarbon propellant	Liquified petroleum gas; (L.P.G.)	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm
dimethyl ether	Methyl ether; (Dimethyl ether)	300ppm	3800*ppm	7200*ppm

Ingredient	Original IDLH	Revised IDLH
xylene	900ppm	Not Available
aluminium powder coated	Not Available	Not Available
zinc phosphate	Not Available	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available	Not Available
hydrocarbon propellant	2,000 ppm	Not Available
dimethyl ether	Not Available	Not Available

MATERIAL DATA

FOR LIQUEFIED PETROLEUM GASES (LPG):
TLV TWA: 1000 ppm, 1800 mg/m3 (as LPG)
ES TWA: 1000 ppm, 1800 mg/m3 (as LPG)
OES TWA: 1000 ppm, 1750 mg/m3; STEL: 1250 ppm, 2180 mg/m3 (as LPG)
IDLH Level: 2000 ppm (lower explosive limit)
No chronic systemic effects have been reported from occupational exposure to LPG. The TLV-TWA is based on good hygiene practices and is thought to minimise the risk of fire or explosion.
Odour Safety Factor(OSF)
OSF=0.16 (hydrocarbon propellant)

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

FOR ALUMINIUM OXIDE AND PYROPHORIC GRADES OF ALUMINIUM:
Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA. The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

FOR ALUMINIUM OXIDE:
The experimental and clinical data indicate that aluminium oxide acts as an "inert" material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.
[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition

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 PROPYLENE GLYCOL MONOMETHYL ETHER (PGME)
Odour Threshold: 10 ppm.
The TLV-TWA is protective against discomfort caused by odour, against eye and skin irritation, and chronic effects (including possible liver and kidney damage).
Individuals exposed to 100 ppm reported a transient unpleasant odour with slight eye irritation after about 1 or 2 hours. At 300 ppm, mild irritation of the eyes and nose developed within 5 minutes; some individuals found the irritation hardly bearable after about an hour. A concentration of 750 ppm was highly irritating. Signs of central nervous system depression developed at 1000 ppm. Neurological, clinical chemical and general medical examinations showed no other conspicuous toxicity. Concentrations of the beta-isomer, 2-methoxy-1-propyl acetate are low in commercial grades of PGME and teratogenic effects associated with this isomer are expected to be absent.
Odour Safety Factor(OSF)
OSF=10 (propylene glycol monomethyl ether) for xylenes: IDLH Level: 900 ppm
Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)

NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).
Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.
Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially contribute to the dose received by inhalation.
Odour Safety Factor(OSF)
OSF=4 (XYLENE)

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 harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

EXPOSURE CONTROLS

Appropriate engineering controls

CARE: Use of a quantity of this material in confined space or poorly ventilated area, where rapid build up of concentrated atmosphere may occur, could require increased ventilation and/or protective gear.

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.

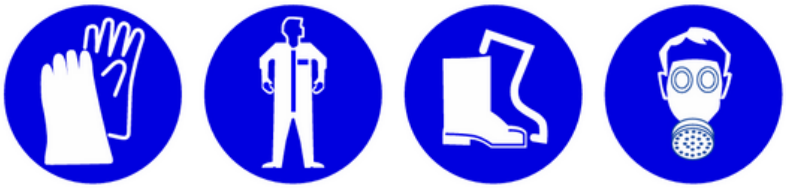
Type of Contaminant	Speed
aerosols, (released at low velocity into zone of active generation)	0.5-1m/s
direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5m/s (200-500 f/min.)

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] • Close fitting gas tight goggles • DO NOT wear contact lenses. • 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]
Skin protection	See Hand protection below
Hands/feet protection	No special equipment needed when handling small quantities. OTHERWISE: • For potentially moderate exposures: • Wear general protective gloves, eg. light weight rubber gloves. • For potentially heavy exposures: • Wear chemical protective gloves, eg. PVC. and safety footwear.
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. OTHERWISE: • Overalls. • Skin cleansing cream. • Eyewash unit. • Do not spray on hot surfaces. • The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton. • Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. BRETHERICK: Handbook of Reactive Chemical Hazards.

RECOMMENDED MATERIAL(S)

GLOVE SELECTION INDEX

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 *computergenerated* selection:

Bright Silver (Hot Dip in a Can)

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
TEFLON	C
VITON	C
VITON	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

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.

^ - Full-face
A(All classes) = Organic vapours
B AUS or B1 = Acid gasses

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AX-AUS		AX-PAPR-AUS / Class 1
up to 50 x ES	-	AX-AUS / Class 1	-
up to 100 x ES	-	AX-2	AX-PAPR-2 ^

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)

- 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.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used
- Generally not applicable.

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

INFORMATION ON BASIC PHYSICAL AND CHIMICAL PROPERTIES

Appearance	Silver coloured flammable liquid with a solvent odour; not miscible with water. Supplied as an aerosol pack. Contents under pressure. Contains highly flammable hydrocarbon/ ether propellant. Note that all of the monopropylene glycol ethers may exist in two isomeric forms, alpha or beta. The alpha form, which is thermodynamically favored during synthesis, consists of a secondary alcohol configuration. The beta form consists of a primary alcohol. The two isomeric forms are shown above. The di- and tripropylene glycol ethers may form up to 4 and 8 isomeric forms, respectively. Even so, all isomers exhibit either the “alpha” or “beta” configuration, existing as secondary or primary alcohols, respectively. The distribution of isomeric forms for the di- and tripropylene glycols, as with the mono-PGEs, also results in predominantly the alpha form (i.e., a secondary alcohol). It should be noted that only the alpha isomer and isomeric mixtures (consisting predominantly of the alpha isomer) are produced commercially; the purified beta isomer is not produced at this time.

Physical state	Liquid	Relative density (Water = 1)	Not Available
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 & boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	-81 (propellant)	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	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

INFORMATION ON TOXICOLOGICAL EFFECTS

Inhaled	• Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. • 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. • 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. Common, generalised symptoms associated with toxic gas inhalation include: • 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. Inhalation hazard is increased at higher temperatures. 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 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. 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. Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue. Xylene is a central nervous system depressant. 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.

SECTION 11 TOXICOLOGICAL INFORMATION

INFORMATION ON TOXICOLOGICAL EFFECTS

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 • Ingestion of alkyl ethers may produce stupor, blurred vision, headache, dizziness and irritation of the nose and throat. Respiratory distress and asphyxia may result.
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either • produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or • produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Spray mist may produce discomfort Open cuts, abraded or irritated skin should not be exposed to this material Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression.
Eye	• 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. • 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. • Eye contact with alkyl ethers (vapours or liquid) may produce irritation, redness and lachrymation.
Chronic	• Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. • There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. • Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. • Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.

SECTION 11 TOXICOLOGICAL INFORMATION

INFORMATION ON TOXICOLOGICAL EFFECTS

Chronic (continued)	When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity. Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction. There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs. Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic. Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer. After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans. At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.

Chronic (continued)	Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively. Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations. Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA). Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995] Principal route of occupational exposure to the gas is by inhalation. Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mix ed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms. Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers. Xylene has been classed as a developmental toxin in some jurisdictions. Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis). Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

BRIGHT SILVER (HOT DIP IN A CAN)

TOXICITY	IRRITATION
Not Available	Not Available

XYLENE

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >1700mg / kg[2]	Eye (human): 200ppm - irritant
Inhalation (rat) LC50: 4994.295mg / l / 4h[2]	Eye (rabbit): 5mg / 24h - SEVERE
Oral (mouse) LD50: 2119mg / kg[2]	Eye (rabbit): 87mg - mild
	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit):500mg / 24h - moderate
	Skin: adverse effect observed (irritating)[1]

ALUMINIUM POWDER COATED

TOXICITY	IRRITATION
Oral (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Skin: no adverse effect observed (not irritating)[1]

ZINC PHOSPHATE

TOXICITY	IRRITATION
Oral (rat) LD50: >5000mg / kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Skin: no adverse effect observed (not irritating)[1]

PROPYLENE GLYCOL MONOMETHYL ETHER -ALPHA ISOMER

TOXICITY	IRRITATION
dermal (rat) LD50: >2000mg / kg[2]	Eye (rabbit) 230 mg mild
Inhalation (rat) LC50: 12485.7375 mg/l/5h.d[2]	Eye (rabbit) 500 mg/24 h. - mild
Oral (rat) LD50: 3739 mg/kg[2]	Eye (rabbit): 100 mg SEVERE
	Skin (rabbit) 500 mg open - mild

HYDROCARBON PROPELLANT

TOXICITY	IRRITATION
Not Available	Not Available

DIMETHYL ETHER

TOXICITY	IRRITATION
Inhalation (rat) LC50: 309mg / l / 4h[2]	Not Available

LEGEND

1. VALUE OBTAINED FROM EUROPE ECHA REGISTERED SUBSTANCES - ACUTE TOXICITY
2.* VALUE OBTAINED FROM MANUFACTURER'S SDS. UNLESS OTHERWISE SPECIFIED DATA EXTRACTED FROM RTECS - REGISTER OF TOXIC EFFECT OF CHEMICAL SUBSTANCES

Chronic (continued)	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. For 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 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 lowestobserved-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day).

Bright Silver (Hot Dip in a Can) (Continued)	**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.
XYLENE	Reproductive effector in rats The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
PROPYLENE GLYCOL MONOMETHYL ETHER ALPHA ISOMER	NOTE: For PGE - mixed isomers: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.
Bright Silver (Hot Dip in a Can) & PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Bright Silver (Hot Dip in a Can) & PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER (Continued)	As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/ m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non irritating. None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/ kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. In vitro, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic in vivo. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.
Bright Silver (Hot Dip in a Can) & ALUMINIUM POWDER COATED & HYDRO CARBON PROPELLANT	No significant acute toxicological data identified in literature search.
Bright Silver (Hot Dip in a Can) & HYDROCARBON PROPELLANT	for Petroleum Hydrocarbon Gases: 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.

Bright Silver (Hot Dip in a Can) & HYDROCARBON PROPELLANT (Continued)	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 **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). **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: 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). **Genotoxicity:** ***In vitro*:** The majority of the Petroleum Hydrocarbon Gases Category components are negative for in vitro genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian in vitro test systems. ***In vivo*:** The majority of the Petroleum Hydrocarbon Gases Category components are negative for in vivo genotoxicity. The exceptions are benzene and 1,3-butadiene, which are genotoxic in in vivo test systems. **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: 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).
Bright Silver (Hot Dip in a Can) & XYLENE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.

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

LEGEND

✗ - DATA EITHER NOT AVAILABLE OR DOES NOT FILL THE CRITERIA FOR CLASSIFICATION
✓ - DATA AVAILABLE TO MAKE CLASSIFICATION

SECTION 12 ECOLOGICAL INFORMATION | TOXICITY

BRIGHT SILVER (HOT DIP IN A CAN)

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

XYLENE

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	2.6mg/L	2
EC50	48	Crustacea	1.8mg/L	2
EC50	72	Algae or other aquatic plants	3.2mg/L	2
NOEC	73	Algae or other aquatic plants	0.44mg/L	2

ALUMINIUM POWDER COATED

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	5-540mg/L	2
EC50	48	Crustacea	>100mg/L	4
EC50	96	Algae or other aquatic plants	20.565mg/L	2
NOEC	504	Crustacea	1-866mg/L	2

ZINC PHOSPHATE

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	0.001-0.58mg/L	2
EC50	48	Crustacea	0.001-0.833mg/L	2
NOEC	72	Algae or other aquatic plants	.00038608mg/L	2

PROPYLENE GLYCOL MONOMETHYL ETHER -ALPHA ISOME

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>=1-mg/L	2
EC50	48	Crustacea	>=1-mg/L	2
EC50	96	Algae or other aquatic plants	>1-mg/L	2
EC0	48	Crustacea	>=1-mg/L	2
NOEC	48	Crustacea	>=1-mg/L	2

HYDROCARBON PROPELLANT

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	24.11mg/L	2
EC50	96	Algae or other aquatic plants	7.71mg/L	2
LC50	96	Fish	24.11mg/L	2
LC50	96	Algae or other aquatic plants	7.71mg/L	2

DIMETHYL ETHER

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	1-783.04mg/L	2
EC50	48	Crustacea	>4400.0mg/L	2
EC50	96	Algae or other aquatic plants	154.917mg/L	2
NOEC	48	Crustacea	>4000mg/L	1

LEGEND

EXTRACTED FROM 1. IUCLID TOXICITY DATA | 2. EUROPE ECHA REGISTERED SUBSTANCES - ECOTOXICOLOGICAL INFORMATION - AQUATIC TOXICITY| 3. EPIWIN SUITE | 3.12 (QSAR) - AQUATIC TOXICITY DATA (ESTIMATED) | 4. US EPA, ECOTOX DATABASE - AQUATIC TOXICITY DATA | 5. ECETOC AQUATIC HAZARD ASSESSMENT DATA | 6. NITE (JAPAN) - BIOCONCENTRATION DATA | 7. METI (JAPAN) - BIOCONCENTRATION DATA | 8. VENDOR DATA

Harmful 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.
When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the oxygen transfer between the air and the water
Oils of any kind can cause:

- drowning of water-fowl due to lack of buoyancy, loss of insulating capacity of feathers, starvation and vulnerability to predators due to lack of mobility
- lethal effects on fish by coating gill surfaces, preventing respiration
- asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom and
- adverse aesthetic effects of fouled shoreline and beaches

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.

for propylene glycol ethers:
Environmental fate: Most are liquids at room temperature and all are water-soluble. Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM)

Environmental fate: Log octanol-water partition coefficients (log Kow's) range from 0.309 for TPM to 1.523 for DPnB. Calculated BCFs range from 1.47 for DPnB to 3.16 for DPMA and TPM, indicating low bioaccumulation. Henry's Law Constants, which indicate propensity to partition from water to air, are low for all category members, ranging from 5.7 x 10-9 atm-m3/mole for TPM to 2.7 x10-9 atm-m3/mole for PnB. Fugacity modeling indicates that most propylene glycol ethers are likely to partition roughly equally into the soil and water compartments in the environment with small to negligible amounts remaining in other environmental compartments (air, sediment, and aquatic biota). Propylene glycol ethers are unlikely to persist in the environment. Once in air, the half-life of the category members due to direct reactions with photochemically generated hydroxyl radicals, range from 2.0 hours for TPM to 4.6 hours for PnB. In water, most members of this family are "readily biodegradable" under aerobic conditions. (DPMA degraded within 28 days (and within the specified 10-day window) but only using pre-adapted or "acclimated" inoculum.). In soil, biodegradation is rapid for PM and PMA.

Ecotoxicity:
Acute aquatic toxicity testing indicates low toxicity for both ethers and acetates. For ethers, effect concentrations are > 500 mg/L. For acetates, effect concentrations are > 151 mg/L. Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms.

When released to dry soil most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. Environmental processes may also be important in changing solubilities.

Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects. A metal ion is considered infinitely persistent because it cannot degrade further.

The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation. The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.
Environmental processes may enhance bioavailability. 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.
Anthrcene 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 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 Gasescan 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 xylenes :
log Koc : 2.05-3.08
Koc : 25.4-204
Half-life (hr) air : 0.24-42
Half-life (hr) H2O surface water : 24-672
Half-life (hr) H2O ground : 336-8640
Half-life (hr) soil : 52-672
Henry's Pa m3 /mol: 637-879
Henry's atm m3 /mol: 7.68E-03
BOD 5 if unstated: 1.4,1%
COD : 2.56,13%
ThOD : 3.125
BCF : 23
log BCF : 1.17-2.41

Environmental Fate
Terrestrial fate:: Measured Koc values of 166 and 182, indicate that 3-xylene is expected to have moderate mobility in soil. Volatilisation of p-xylene is expected to be important from moist soil surfaces given a measured Henry's Law constant of 7.18x10-3 atm-cu m/mole. The potential for volatilisation of 3-xylene from dry soil surfaces may exist based on a measured vapor pressure of 8.29 mm Hg. p-Xylene may be degraded during its passage through soil). The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. p-Xylene, present in soil samples contaminated with jet fuel, was completely degraded aerobically within 5 days. In aquifer studies under anaerobic conditions, p-xylene was degraded, usually within several weeks, with the production of 3-methylbenzylfumaric acid, 3-methylbenzylsuccinic acid, 3-methylbenzoate, and 3-methylbenzaldehyde as metabolites.

Aquatic fate: Koc values indicate that p-xylene may adsorb to suspended solids and sediment in water. p-Xylene is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. BCF values of 14.8, 23.4, and 6, measured in goldfish, eels, and clams, respectively, indicate that bioconcentration in aquatic organisms is low. p-Xylene in water with added humic substances was 50% degraded following 3 hours irradiation suggesting that indirect photooxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. Although p-xylene is biodegradable and has been observed to degrade in pond water, there are insufficient data to assess the rate of this process in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater in several studies; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high.

Atmospheric fate: Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere primarily by reaction with photochemically produced hydroxyl radicals, with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylenes' susceptibility to photochemical oxidation in the troposphere is to the extent that they may contribute to photochemical smog formation. According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and from its vapour pressure, p-xylene, is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase p-xylene is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated

to be about 16 hours. A half-life of 1.0 hr in summer and 10 hr in winter was measured for the reaction of p-xylene with photochemically-produced hydroxyl radicals.

p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers, with loss rates varying from 9-42% per hr. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzylnitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethylp-benzoquinone, 2,4-dimethylphenol, 6-nitro 2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6 dimethylphenol.

Ecotoxicity:
for xylenes Fish LC50 (96 h) Pimephales promelas 13.4 mg/l; Oncorhyncus mykiss 8.05 mg/l; Lepomis macrochirus 16.1 mg/l (all flow through values); Pimephales promelas 26.7 (static) Daphnia EC50 948 h): 3.83 mg/l Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/l Gammarus lacustris LC50 (48 h): 0.6 mg/l

For aluminium and its compounds and salts: Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals.Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.

Environmental fate:
Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter. Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.

As an element, aluminum cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminum in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminum can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminum in the environment will depend on the ligands present and the pH. The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion, [Al(H2O)6]3+, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H2O)5(OH)]2+, [Al(H2O)4(OH)2]+). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)2+ and Al(OH)2+, while the solid Al(OH)3 is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)4- is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)3, which crystallise to gibbsite in acid waters.

Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystalized clay mineral species.

Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.

Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.

The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.

Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.

Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, Abies amabilis, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/ concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.

Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.

The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.

Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.
Ecotoxicity:
Freshwater species pH >6.5
Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (Salmo salar) - 106 mg/L; Chronic NOEC (8-28 d): 7 spp,NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for Micropterus sp.

Amphibian: Acute LC50 (4 d): Bufo americanus, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L
Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7 28 d) 3 spp, 0.136-1.72 mg/L
Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L

Freshwater species pH <6.5 (all between pH 4.5 and 6.0)
Fish LC50 (24-96 h): 4 spp, 0.015 (S. trutta) - 4.2 mg/L; chronic data on Salmo trutta, LC50 (21-42 d) 0.015- 0.105 mg/L
Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)

Alga: 1 sp NOEC growth 2.0 mg/L
Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.

The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4.5.4, with a maximum toxicity occurring around pH 5.0.5.2. The inorganic single unit aluminium species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9.

However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.
Drinking Water Standards:
aluminium: 200 ug/l (UK max.)
200 ug/l (WHO guideline)
chloride: 400 mg/l (UK max.)
250 mg/l (WHO guideline)
fluoride: 1.5 mg/l (UK max.)
1.5 mg/l (WHO guideline)
nitrate: 50 mg/l (UK max.)
50 mg/l (WHO guideline)
sulfate: 250 mg/l (UK max.)
Soil Guideline: none available.
Air Quality Standards: none available.

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

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

Environmental Fate
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 stimated 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 5x105 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:
Environmental Fate Terrestrial fate:: An estimated Koc value of 460 determined from a log Kow of 2.36 indicates that propane is expected to have moderate mobility in soil. Volatilisation of propane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 7.07x10-1 atm-cu m/mole, derived from its vapor pressure, 7150 mm Hg, and water solubility, 62.4 mg/L. Propane is expected to volatilise from dry soil surfaces based upon its vapor pressure. Using cell suspensions of microorganisms isolated from soil and water, propane was oxidised to acetone within 24 hours, suggesting that biodegradation may be an important fate process in soil and sediment.

Aquatic fate: The estimated Koc value indicates that propane is expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant. Using this Henry's Law constant volatilisation half lives for a model river and model lake are estimated to be 41 minutes and 2.6 days, respectively.

An estimated BCF of 13.1 using log Kow suggests the potential for bioconcentration in aquatic organisms is low. After 192 hr, the trace concentration of propane contained in gasoline remained unchanged for both a sterile control and a mixed culture sample collected from ground water contaminated with gasoline. This indicates that biodegradation may not be an important fate process in water.

Atmospheric fate: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and vapour pressure, 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, calculated from its rate constant of 1.15x10-12 cu cm/molecule-sec at 25 deg C. Propane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.

DO NOT discharge into sewer or waterways.

PERSISTENCE AND DEGRADABILITY

Ingredient	Persistence: Water/Soil	Persistence: Air
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
propylene glycol monomethyl ether - alpha isomer	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
dimethyl ether	LOW	LOW

BIOACCUMULATIVE POTENTIAL

Ingredient	Bioaccumulation
xylene	MEDIUM (BCF = 740)
propylene glycol monomethyl ether - alpha isomer	LOW (BCF = 2)
dimethyl ether	LOW (LogKOW = -0.1)

MOBILITY IN SOIL

Ingredient	Bioaccumulation
propylene glycol monomethyl ether - alpha isomer	HIGH (KOC = 1)
dimethyl ether	HIGH (KOC = 1.292)

SECTION 13 DISPOSAL CONSIDERATIONS

WASTE TREATMENT METHODS

Product / Packaging disposal	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: • Reduction • Reuse • Recycling • Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. • DO NOT allow wash water from cleaning or process equipment to enter drains. • It may be necessary to collect all wash water for treatment before disposal. • In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. • Where in doubt contact the responsible authority. • Consult State Land Waste Management Authority for disposal. • Discharge contents of damaged aerosol cans at an approved site. • Allow small quantities to evaporate. • DO NOT incinerate or puncture aerosol cans. • Bury residues and emptied aerosol cans at an approved site.

SECTION 14 TRANSPORT INFORMATION

LABELS REQUIRED

	
Marine Pollutant	
HAZCHEM	Not Applicable

LAND TRANSPORT (ADG)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	Class: 2.1 Subrisk: Not applicable
Packing group	Not applicable
Environmental hazard	Environmentally Hazardous
Special precautions for user	Special provisions: 63 190 277 327 344 381 Limited quantity: 1000ml

AIR TRANSPORT (ICAO-IATA / DGR)

UN number	1950
UN proper shipping name	Aerosols, flammable (engine starting fluid); Aerosols, flammable
Transport hazard class(es	ICAO/IATA Class: 2.1 ICAO / IATA Subrisk: Not Applicable ERG Code: 10L
Packing group	Not Applicable
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A145 A167 A802; A1 A145 A167 A802 Cargo Only Packing Instructions: 203 Cargo Only Maximum Qty / Pack: 150kg Passenger and Cargo Packing Instructions: 203; Forbidden Passenger and Cargo Maximum Qty / Pack: 75 kg; Forbidden Passenger and Cargo Limited Quantity Packing Instructions: Y203; Forbidden Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G; Forbidden

SEA TRANSPORT (IMDG-CODE / GGVSEE)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es	IMDG CLASS: 2.1 IMDG SURISK: Not Applicable
Packing group	Not Applicable
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-D , S-U Special provisions: 63 190 277 327 344 381 959 Limited Quantites: 1000ml

SECTION 15 REGULATORY INFORMATION

SAFETY, HEALTH AND ENVIRONMENTAL REGULATIONS / LEGISLATION SPECIFIC FOR THE SUBSTANCE OR MIXTURE

XYLENE IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Hazardous chemicals which may require Health Monitoring
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) Part 2, Section Seven - Appendix I
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

ALUMINIUM POWDER COATED IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)

ZINC PHOSPHATE IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)

HYDROCARBON PROPELLANT IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5 Chemical Footprint Project - Chemicals of High Concern List

DIMETHYL ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

National Inventory	Status
Australia - AIIC	Yes
Canada - DSL	Yes
Canada - NDSL	No (aluminium powder coated; xylene; propylene glycol monomethyl ether - alpha isomer; dimethyl ether; hydrocarbon propellant)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (aluminium; zinc powder)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (zinc phosphate)
Vietnam - NCI	Yes
Russia - ARIPS	Yes

LEGEND

YES = ALL CAS DECLARED INGREDIENTS ARE ON THE INVENTORY
NO = ONE OR MORE OF THE CAS LISTED INGREDIENTS ARE NOT ON THE INVENTORY AND ARE NOT EXEMPT FROM LISTING (SEE SPECIFIC INGREDIENTS IN BRACKETS)

SECTION 16 OTHER INFORMATION

Revision Date	01/11/2019
Initial Date	28/04/2014

SDS VERSION SUMMARY

Version	Issue Date	Section Updated
9.1.1.1	11/08/2016	Classification, Name
10.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

Other information

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

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

Definitions and abbreviations

- PC - TWA: Permissible Concentration-Time Weighted Average
- PC - STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit
- IDLH: Immediately Dangerous to Life or Health Concentrations
- OSF: Odour Safety Factor
- NOAEL :No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index