

DY-MARK SILVER GAL PROTECTION SPRAY

Chemwatch Independent Material Safety Data Sheet
Issue Date: 3-Oct-2011
NC317ECP

CHEMWATCH 4994-16
Version No:7
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Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME

DY-MARK SILVER GAL PROTECTION SPRAY

PROPER SHIPPING NAME

AEROSOLS

PRODUCT USE

■ Application is by spray atomisation from a hand held aerosol pack.
Anti-corrosive spray.

SUPPLIER

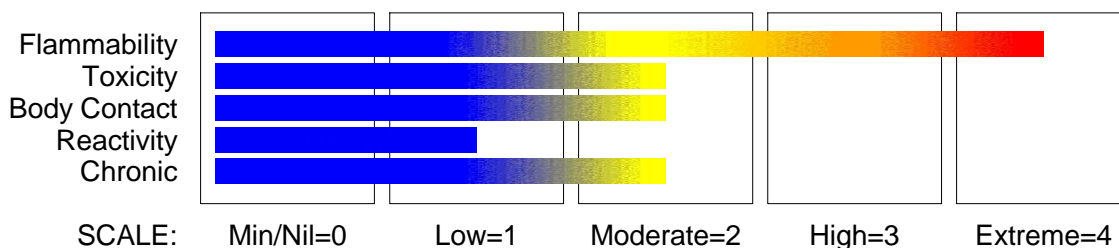
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Section 2 - HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE

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

CHEMWATCH HAZARD RATINGS



RISK

Risk Codes

R12
R22
R36/38
R44
R48/20

Risk Phrases

- Extremely flammable.
- Harmful if swallowed.
- Irritating to eyes and skin.
- Risk of explosion if heated under confinement.
- Harmful: danger of serious damage to health by prolonged exposure through inhalation.
- Possible risk of harm to the unborn child.
- Vapours may cause drowsiness and dizziness.

R63(3)
R67

SAFETY

Safety Codes
S16

Safety Phrases

- Keep away from sources of ignition. No smoking.

continued...

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Section 2 - HAZARDS IDENTIFICATION

| | |
|------|--|
| S23 | • Do not breathe gas/ fumes/ vapour/ spray. |
| S24 | • Avoid contact with skin. |
| S25 | • Avoid contact with eyes. |
| S36 | • Wear suitable protective clothing. |
| S37 | • Wear suitable gloves. |
| S39 | • Wear eye/ face protection. |
| S51 | • Use only in well ventilated areas. |
| S09 | • Keep container in a well ventilated place. |
| S53 | • Avoid exposure - obtain special instructions before use. |
| S401 | • To clean the floor and all objects contaminated by this material, use water and detergent. |
| S07 | • Keep container tightly closed. |
| S13 | • Keep away from food, drink and animal feeding stuffs. |
| S26 | • In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre. |
| S46 | • If swallowed, IMMEDIATELY contact Doctor or Poisons Information Centre (show this container or label). |
| S60 | • This material and its container must be disposed of as hazardous waste. |

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

| NAME | CAS RN | % |
|--|-------------|-------|
| toluene | 108-88-3 | 30-60 |
| aluminium | 7429-90-5 | 1-10 |
| propylene glycol monomethyl ether - alpha isomer | 107-98-2 | <1 |
| hydrocarbon propellant | 68476-85-7. | 30-60 |

Section 4 - FIRST AID MEASURES

SWALLOWED

- - Avoid giving milk or oils.
- Avoid giving alcohol.
- Not considered a normal route of entry.
- If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

EYE

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

SKIN

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

INHALED

- If aerosols, fumes or combustion products are inhaled:

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Section 4 - FIRST AID MEASURES

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

NOTES TO PHYSICIAN

- Treat symptomatically.

Following acute or short term repeated exposures to toluene:

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

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

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

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

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

- SMALL FIRE:

- Water spray, dry chemical or CO₂

LARGE FIRE:

- Water spray or fog.

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.

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Section 5 - FIRE FIGHTING MEASURES

- DO NOT approach containers suspected to be hot.
 - Cool fire exposed containers with water spray from a protected location.
 - If safe to do so, remove containers from path of fire.
 - Equipment should be thoroughly decontaminated after use.
- When any large container (including road and rail tankers) is involved in a fire, consider evacuation by 100 metres in all directions.

FIRE/EXPLOSION HAZARD

- - Liquid and vapour are highly flammable.
- Severe fire hazard when exposed to heat or flame.
- Vapour forms an explosive mixture with air.
- Severe explosion hazard, in the form of vapour, when exposed to flame or spark.
- Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion or decomposition with violent container rupture.
- Aerosol cans may explode on exposure to naked flames.
- Rupturing containers may rocket and scatter burning materials.
- Hazards may not be restricted to pressure effects.
- May emit acrid, poisonous or corrosive fumes.
- On combustion, may emit toxic fumes of carbon monoxide (CO).

Combustion products include: carbon monoxide (CO).

Combustible. Will burn if ignited, 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.

FIRE INCOMPATIBILITY

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

HAZCHEM

2YE

Personal Protective Equipment

Breathing apparatus.

Gas tight chemical resistant suit.

Limit exposure duration to 1 BA set 30 mins.

Section 6 - ACCIDENTAL RELEASE MEASURES

MINOR SPILLS

- - Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Wear protective clothing, impervious gloves and safety glasses.
- Shut off all possible sources of ignition and increase ventilation.
- Wipe up.
- If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.
- Undamaged cans should be gathered and stowed safely.

MAJOR SPILLS

- - Remove leaking cylinders to a safe place if possible.
- Release pressure under safe, controlled conditions by opening the valve.
- 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.

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Section 6 - ACCIDENTAL RELEASE MEASURES

- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water courses
- No smoking, naked lights or ignition sources.
- Increase ventilation.
- Stop leak if safe to do so.
- Water spray or fog may be used to disperse / absorb vapour.
- Absorb or cover spill with sand, earth, inert materials or vermiculite.
- If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.
- Undamaged cans should be gathered and stowed safely.
- Collect residues and seal in labelled drums for disposal.

EMERGENCY RESPONSE PLANNING GUIDELINES (ERPG)

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

life-threatening health effects is:

toluene 1000ppm

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

toluene 300ppm

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

toluene 50ppm

American Industrial Hygiene Association (AIHA)

Ingredients considered according to the following cutoffs

| | | | |
|-----------------|----------|---------------|---------|
| Very Toxic (T+) | >= 0.1% | Toxic (T) | >= 3.0% |
| R50 | >= 0.25% | Corrosive (C) | >= 5.0% |
| R51 | >= 2.5% | | |
| else | >= 10% | | |

where percentage is percentage of ingredient found in the mixture

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

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

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Section 7 - HANDLING AND STORAGE

conditions are maintained.

SUITABLE CONTAINER

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

STORAGE INCOMPATIBILITY

- - Avoid reaction with oxidising agents.

STORAGE REQUIREMENTS

- - Store below 38 deg. C.
- Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can.
- Store in original containers in approved flammable liquid storage area.
- DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
- No smoking, naked lights, heat or ignition sources.
- Keep containers securely sealed. Contents under pressure.
- Store away from incompatible materials.
- Store in a cool, dry, well ventilated area.
- Avoid storage at temperatures higher than 40 deg C.
- Store in an upright position.
- Protect containers against physical damage.
- Check regularly for spills and leaks.
- Observe manufacturer's storing and handling recommendations.

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

| Source | Material | TWA ppm | TWA mg/m ³ | STEL ppm | STEL mg/m ³ | Notes |
|------------------------------|--|---------|-----------------------|----------|------------------------|------------------|
| Australia Exposure Standards | Dy- Mark Silver Gal Protection Spray (Propylene glycol monomethyl ether) | 100 | 369 | 150 | 553 | |
| Australia Exposure Standards | Dy- Mark Silver Gal Protection Spray (Emery (dust) (a)) | | 10 | | | (see Chapter 14) |
| Australia Exposure Standards | Dy- Mark Silver Gal Protection Spray (Aluminium (welding fumes) (as Al)) | | 5 | | | |
| Australia Exposure Standards | Dy- Mark Silver Gal Protection Spray (Aluminium (metal dust)) | | 10 | | | |
| Australia Exposure Standards | toluene (Toluene) | 50 | 191 | 150 | 574 | Sk |
| Australia Exposure Standards | hydrocarbon propellant (LPG (liquified petroleum gas)) | 1000 | 1800 | | | |

EMERGENCY EXPOSURE LIMITS

| Material | Revised | IDLH |
|----------|---------|------|
| toluene | 84 | 500 |

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hydrocarbon propellant

0

2, 000 [LEL]

NOTES

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

MATERIAL DATA

DY-MARK SILVER GAL PROTECTION SPRAY:

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

- 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).

DY-MARK SILVER GAL PROTECTION SPRAY:

TOLUENE:

- For toluene:

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

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

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

Odour Safety Factor(OSF)

OSF=17 (TOLUENE).

ALUMINIUM:

DY-MARK SILVER GAL PROTECTION SPRAY:

- 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.

ALUMINIUM:

DY-MARK SILVER GAL PROTECTION SPRAY:

- 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.

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TOLUENE:

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

HYDROCARBON PROPELLANT:

■ For butane:

Odour Threshold Value: 2591 ppm (recognition)

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

Odour Safety Factor(OSF)

OSF=0.22 (n-BUTANE).

For propane

Odour Safety Factor(OSF)

OSF=0.16 (PROPANE).

PERSONAL PROTECTION

EYE

■ - Safety glasses with side shields.

- Chemical goggles.

- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent].

HANDS/FEET

■ - No special equipment needed when handling small quantities.

- OTHERWISE:

- For potentially moderate exposures:

- Wear general protective gloves, eg. light weight rubber gloves.

- For potentially heavy exposures:

- Wear chemical protective gloves, eg. PVC. and safety footwear.

OTHER

■ No special equipment needed when handling small quantities.

OTHERWISE:

- Overalls.

- Skin cleansing cream.

- Eyewash unit.

- Do not spray on hot surfaces.

- The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton.

- Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost.

BRETHERRICK: Handbook of Reactive Chemical Hazards.

RESPIRATOR

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

■ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour

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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.

- Positive pressure, full face, air-supplied breathing apparatus should be used for work in enclosed spaces if a leak is suspected or the primary containment is to be opened (e.g. for a cylinder change)

- Air-supplied breathing apparatus is required where release of gas from primary containment is either suspected or demonstrated.

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

ENGINEERING CONTROLS

■ Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.

Provide adequate ventilation in warehouse or closed storage areas.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

■ Supplied as an aerosol pack. Contents under PRESSURE. Contains highly flammable hydrocarbon propellant. Silver coloured flammable liquid with solvent odour; does not mix with water.

PHYSICAL PROPERTIES

Liquid.

Gas.

Does not mix with water.

| | | | |
|---------------------------|-------------------|---------------------------------|----------------|
| State | Liquid | Molecular Weight | Not Applicable |
| Melting Range (°C) | Not Available | Viscosity | Not Available |
| Boiling Range (°C) | Not Available | Solubility in water (g/L) | Immiscible |
| Flash Point (°C) | - 81 (propellant) | pH (1% solution) | Not Applicable |
| Decomposition Temp (°C) | Not Available | pH (as supplied) | Not Applicable |
| Autoignition Temp (°C) | Not Available | Vapour Pressure (kPa) | Not Available |
| Upper Explosive Limit (%) | Not Available | Specific Gravity (water=1) | Not Available |
| Lower Explosive Limit (%) | Not Available | Relative Vapour Density (air=1) | Not Available |
| Volatile Component (%vol) | Not Available | Evaporation Rate | Not Available |

toluene

log Kow (Sangster 1997):

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Section 10 - STABILITY AND REACTIVITY

CONDITIONS CONTRIBUTING TO INSTABILITY

- - Elevated temperatures.
 - Presence of open flame.
 - Product is considered stable.
 - Hazardous polymerisation will not occur.
- For incompatible materials - refer to Section 7 - Handling and Storage.*

Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

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

Not normally a hazard due to physical form of product.

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

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

EYE

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

The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.

There is evidence that material may produce eye irritation in some persons and produce eye damage 24 hours or more after instillation. Severe inflammation may be expected with pain. There may be damage to the cornea. Unless treatment is prompt and adequate there may be permanent loss of vision. Conjunctivitis can occur following repeated exposure.

SKIN

- The material may cause moderate inflammation of the skin either following direct contact or after a delay of some time. Repeated exposure can cause contact dermatitis which is characterised by redness, swelling and blistering.

Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.

Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Thus it may cause itching and skin reaction and inflammation.

Spray mist may produce discomfort.

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

Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

INHALED

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

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

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

The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule,

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Section 11 - TOXICOLOGICAL INFORMATION

these compounds may also act as general anaesthetics.

Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.

Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.

Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed.

Inhalation of toxic gases may cause:

- Central Nervous System effects including depression, headache, confusion, dizziness, stupor, coma and seizures;
- respiratory: acute lung swellings, shortness of breath, wheezing, rapid breathing, other symptoms and respiratory arrest;
- heart: collapse, irregular heartbeats and cardiac arrest;
- gastrointestinal: irritation, ulcers, nausea and vomiting (may be bloody), and abdominal pain.

Central nervous system (CNS) depression may include general 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. Vapour is heavier than air and may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.

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

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

Inhaling high concentrations of mixed hydrocarbons can cause narcosis, with nausea, vomiting and lightheadedness. Low molecular weight (C2-C12) hydrocarbons can irritate mucous membranes and cause incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and stupor. Massive exposures can lead to severe central nervous system depression, deep coma and death. Convulsions can occur due to brain irritation and/or lack of oxygen. Permanent scarring may occur, with epileptic seizures and brain bleeds occurring months after exposure. Respiratory system effects include inflammation of the lungs with oedema and bleeding. Lighter species mainly cause kidney and nerve damage; the heavier paraffins and olefins are especially irritant to the respiratory system. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce sensation loss and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce multiple nerve damage. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue, vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons can sensitise the heart and may cause ventricular fibrillation, leading to death.

Exposure to hydrocarbons may result in irregularity of heart beat. Symptoms of moderate poisoning may include dizziness, headache, nausea. Serious poisoning can result in decreased respiratory function, this may lead to unconsciousness and death. C4 hydrocarbons are especially dangerous to the nervous system. Inhalation of petroleum gases (partly due to olefin impurities) can induce sleep. Serious cases can result in cyanosis due to reduced oxygen concentration and hence asphyxiation, with symptoms of fast breathing, mental dullness, inco- ordination, poor judgment, nausea and vomiting; leading to unconsciousness and death.

CHRONIC HEALTH EFFECTS

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

Based on experience with animal studies, exposure to the material may result in toxic effects to the development of the foetus, at levels which do not cause significant toxic effects to the mother.

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

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make an assessment.

Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure.

Animal testing shows long term exposure to aluminium oxides may cause lung disease and cancer, depending on the size of the particle. The smaller the size, the greater the tendencies of causing harm.

Constant or exposure over long periods to mixed hydrocarbons may produce stupor with dizziness, weakness and visual disturbance, weight loss and anaemia, and reduced liver and kidney function. Skin exposure may result in drying and cracking and redness of the skin. Chronic exposure to lighter hydrocarbons can cause nerve damage, peripheral neuropathy, bone marrow dysfunction and psychiatric disorders as well as damage the liver and kidneys.

Intentional abuse (glue sniffing) or occupational exposure to toluene can result in chronic habituation.

Chronic abuse has caused inco-ordination, tremors of the extremities (due to widespread cerebrum withering), headache, abnormal speech, temporary memory loss, convulsions, coma, drowsiness, reduced colour perception, blindness, nystagmus (rapid, involuntary eye movements), hearing loss leading to deafness and mild dementia.

Toluene addicts often display a range of disease phenomena in their nervous systems. Toluene abuse can cause kidney disease but occupational toluene exposures usually do not cause it. Chronic exposure to toluene can damage the heart and the blood, especially causing heartbeat irregularities. High concentrations of toluene can harm the unborn baby and the developing infant.

TOXICITY AND IRRITATION

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

HYDROCARBON PROPELLANT:

ALUMINIUM:

■ No significant acute toxicological data identified in literature search.

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

DY-MARK SILVER GAL PROTECTION SPRAY:

■ for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no

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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 sensitizers.

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.

TOLUENE:

DY-MARK SILVER GAL PROTECTION SPRAY:

■ 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.

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Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea. Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death. Toluene can also strip the skin of lipids causing dermatitis.

Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression.

Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days.

Subchronic/Chronic Effects:

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

Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitizer and fatal cardiotoxin.

Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L.

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

Developmental/Reproductive Toxicity

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

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

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

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

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

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

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

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TOLUENE:

TOXICITY

Oral (human) LDLo: 50 mg/kg

Oral (rat) LD50: 636 mg/kg

Inhalation (human) TClO: 100 ppm

Inhalation (man) TClO: 200 ppm

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

Dermal (rabbit) LD50: 12124 mg/kg

IRRITATION

Skin (rabbit):20 mg/24h- Moderate

Skin (rabbit):500 mg - Moderate

Eye (rabbit):0.87 mg - Mild

Eye (rabbit): 2mg/24h - SEVERE

Eye (rabbit):100 mg/30sec - Mild

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

TOXICITY

Oral (rat) LD50: 3739 mg/kg

Inhalation (human) TClO: 3000 ppm

Inhalation (rat) LC50: 10000 ppm/5 h.

Dermal (rabbit) LD50: 13000 mg/kg

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.

IRRITATION

Skin (rabbit) 500 mg Open - Mild

Eye (rabbit) 230 mg Mild

Eye (rabbit) 500 mg/24 h. - Mild

Eye (rabbit): 100 mg SEVERE

HYDROCARBON PROPELLANT:

- inhalation of the gas.

CARCINOGEN

Toluene

International Agency for Research on Cancer
(IARC) - Agents Reviewed by the IARC
Monographs

Group

3

REPROTOXIN

toluene

ILO Chemicals in the
electronics industry
that have toxic effects
on reproduction

Reduced fertility or
sterility

SKIN

toluene

Australia Exposure Standards - Skin

Notes

Sk

Section 12 - ECOLOGICAL INFORMATION

ALUMINIUM:

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

HYDROCARBON PROPELLANT:

TOLUENE:

- DO NOT discharge into sewer or waterways.

TOLUENE:

- For toluene:

log Kow : 2.1-3

log Koc : 1.12-2.85

Koc : 37-260

log Kom : 1.39-2.89

Half-life (hr) air : 2.4-104

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

Half-life (hr) H₂O ground : 168-2628

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

Henry's Pa m³ /mol: 518-694

Henry's atm m³ /mol: 5.94E-03

BOD 5 0.86-2.12, 5%

COD : 0.7-2.52, 21-27%

ThOD : 3.13

BCF : 1.67-380

log BCF : 0.22-3.28

Environmental fate:

Transport: The majority of toluene evaporates to the atmosphere from the water and soil. It is moderately retarded by adsorption to soils rich in organic material (K_{oc} = 259), therefore, transport to ground water is dependent on the soil composition. In unsaturated topsoil containing organic material, it has been estimated that 97% of the toluene is adsorbed to the soil and only about 2% is in the soil-water phase and transported with flowing groundwater. There is little retardation in sandy soils and 2-13% of the toluene was estimated to migrate with flowing water; the remainder was volatilised, biodegraded, or unaccounted for. In saturated deep soils with no soil-air phase, about 48% may be transported with flowing groundwater.

Transformation/Persistence:

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

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

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

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

Ecotoxicity:

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

Fish LC₅₀ (96 h): fathead minnow (*Pimephales promelas*) 12.6-72 mg/l; *Lepomis macrochirus* 13-24 mg/l; guppy (*Poecilia reticulata*) 28.2-59.3 mg/l; channel catfish (*Ictalurus punctatus*) 240 mg/l; goldfish (*Carassius auratus*): 22.8-57.68 mg/l

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

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

ALUMINIUM:

■ 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.

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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.

For aluminium and its compounds and salts:

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, $[Al(H_2O)_6]^{3+}$, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., $[Al(H_2O)_5(OH)]^{2+}$, $[Al(H_2O)_4(OH)_2]^+$). 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 crystallised 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

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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 mg/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.

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

■ For glycol ethers:

Environmental fate:

Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures.

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OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity:

Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.

Glycols exert a high oxygen demand for decomposition and once released to the environments cause the death of aquatic organisms if dissolved oxygen is depleted.

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 5.7×10^{-9} atm-m³/mole for TPM to 2.7×10^{-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 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.

log Kow: -0.53

Dissolves rapidly in water; the material is volatile and will partition to the air compartment.

Readily biodegradable.

Degradation by activated sludge in 29 days=90%

Not expected to bioaccumulate

Fish LC50 (96 h): fathead minnow >2000 mg/l

HYDROCARBON PROPELLANT:

■ for Petroleum Hydrocarbon Gases:

Environmental fate:

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

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

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these gases to enter the atmosphere.

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

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

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

Ecotoxicity:

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

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

For isobutane:

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

Environmental Fate

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

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

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

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

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

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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 7.07×10^{-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 1.15×10^{-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.

Ecotoxicity

| Ingredient | Persistence: Water/Soil | Persistence: Air | Bioaccumulation | Mobility |
|---|----------------------------|----------------------|-----------------|----------|
| Dy- Mark Silver Gal Protection Spray | No Data Available | No Data Available | | |
| toluene | LOW | MED | LOW | MED |
| aluminium | No Data Available | No Data Available | | |
| propylene glycol monomethyl ether - alpha isomer | LOW | LOW | LOW | HIGH |
| hydrocarbon propellant | No Data Available | No Data Available | | |

Section 13 - DISPOSAL CONSIDERATIONS

- - DO NOT allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Consult State Land Waste Management Authority for disposal.
- Discharge contents of damaged aerosol cans at an approved site.
- Allow small quantities to evaporate.
- DO NOT incinerate or puncture aerosol cans.
- Bury residues and emptied aerosol cans at an approved site.

Section 14 - TRANSPORTATION INFORMATION

Labels Required: FLAMMABLE GAS

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Section 14 - TRANSPORTATION INFORMATION

HAZCHEM:

2YE (ADG7)

ADG7:

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

Name and Description: AEROSOLS

Land Transport UNDG:

| | | | |
|-------------------|------|-------------------|------|
| Class or division | 2.1 | Subsidiary risk: | None |
| UN No.: | 1950 | UN packing group: | None |

Shipping Name:AEROSOLS

Air Transport IATA:

| | | | |
|--|------|--|---------|
| UN/ID Number: | 1950 | Packing Group: | - |
| Special provisions: Cargo Only | A145 | | |
| Packing Instructions: Passenger and Cargo | 203 | Maximum Qty/Pack: Passenger and Cargo | 150 kg |
| Packing Instructions: Passenger and Cargo Limited Quantity | Y203 | Maximum Qty/Pack: Passenger and Cargo Limited Quantity | 75 kg |
| Packing Instructions: | 203 | Maximum Qty/Pack: | 30 kg G |

Shipping Name: AEROSOLS, FLAMMABLE

Maritime Transport IMDG:

| | | | |
|-------------|------------|---------------------|------------------------|
| IMDG Class: | 2 | IMDG Subrisk: | SP63 |
| UN Number: | 1950 | Packing Group: | None |
| EMS Number: | F- D, S- U | Special provisions: | 63 190 277 327 344 959 |

Limited Quantities: See SP277
Shipping Name: AEROSOLS

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE None

REGULATIONS

Regulations for ingredients

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

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

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Fragrance Association (IFRA) Standards Prohibited", "WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water"

aluminium (CAS: 7429-90-5) is found on the following regulatory lists;

"Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (AQUA/1 to 6 - inorganic chemicals)", "Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (Domestic water supply - inorganic chemicals)", "Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (IRRIG - inorganic chemicals)", "Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (STOCK - inorganic chemicals)", "Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (Aquatic habitat)", "Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (Domestic water supply quality)", "Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (IRRIG)", "Australia - Australian Capital Territory Environment Protection Regulation Pollutants entering waterways - Agricultural uses (Stock)", "Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "WHO Guidelines for Drinking-water Quality - Chemicals for which guideline values have not been established"

propylene glycol monomethyl ether - alpha isomer (CAS: 107-98-2) is found on the following regulatory lists;

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia Inventory of Chemical Substances (AICS)", "IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk", "International Council of Chemical Associations (ICCA) - High Production Volume List", "International Fragrance Association (IFRA) Survey: Transparency List"

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

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

No data for Dy-Mark Silver Gal Protection Spray (CW: 4994-16)

Section 16 - OTHER INFORMATION

INGREDIENTS WITH MULTIPLE CAS NUMBERS

| Ingredient Name | CAS |
|------------------------|----------------------------|
| hydrocarbon propellant | 68476- 85- 7, 68476- 86- 8 |

REPRODUCTIVE HEALTH GUIDELINES

| Ingredient | ORG | UF | Endpoint | CR | Adeq TLV |
|------------|-----------------------|----|----------|----|----------|
| toluene | 9.6 mg/m ³ | 10 | D | NA | - |

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

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

TLV believed to be adequate to protect reproductive health:

LOD: Limit of detection

Toxic endpoints have also been identified as:

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

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

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

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

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

www.chemwatch.net/references.

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

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This is the end of the MSDS.