DY-MARK RUST REFORMER AEROSOL

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
DY-MARK RUST REFORMER AEROSOL

PROPER SHIPPING NAME
AEROSOLS

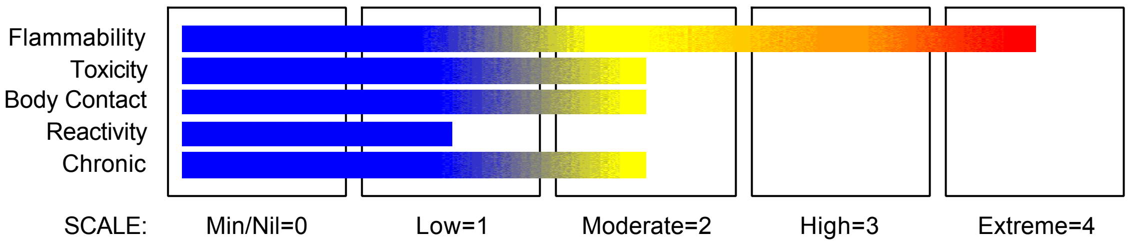
PRODUCT USE
■ Application is by spray atomisation from a hand held aerosol pack.

SUPPLIER
Company: Dy- Mark Pty Ltd
Address:
89 Formation Street
Wacol
QLD, 4076
Australia
Telephone: +61 7 3271 2222
Fax: +61 7 3271 2751
Email: info@dymark.com.au

Section 2 - HAZARDS IDENTIFICATION

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

CHEMWATCH HAZARD RATINGS



RISK
■ Extremely flammable.

SAFETY
■ Keep away from sources of ignition. No smoking.

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■ Harmful by inhalation, in contact with skin and if swallowed.
■ Irritating to eyes and skin.
■ Limited evidence of a carcinogenic effect.
■ Risk of explosion if heated under confinement.
■ Harmful: danger of serious damage to health by prolonged exposure through inhalation.
■ Harmful to aquatic organisms.
■ Possible risk of harm to the unborn child.

■ Repeated exposure may cause skin dryness and cracking.

■ Vapours may cause drowsiness and dizziness.

■ Cumulative effects may result following exposure*.
■ May produce discomfort of the respiratory system*.
■ May possibly affect fertility*.
* (limited evidence).

■ Do not breathe gas/ fumes/ vapour/ spray.

■ Use only in well ventilated areas.
■ Keep container in a well ventilated place.
■ Avoid exposure - obtain special instructions before use.
■ To clean the floor and all objects contaminated by this material, use water and detergent.

■ Keep container tightly closed.
■ Keep away from food, drink and animal feeding stuffs.
■ In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.
■ If swallowed, IMMEDIATELY contact Doctor or Poisons Information Centre (show this container or label).
■ This material and its container must be disposed of as hazardous waste.

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
acetone	67-64-1	10-30
toluene	108-88-3	10-30
n- butyl acetate	123-86-4	10-15
white spirit	8052-41-3.	1-10
xylene	1330-20-7	1-10
ethylene glycol monobutyl ether	111-76-2	1-10
ethylbenzene	100-41-4	1-10
additives non- hazardous		10-30
hydrocarbon propellant	68476-85-7.	10-30

Section 4 - FIRST AID MEASURES

SWALLOWED

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

EYE

■ If aerosols come in contact with the eyes:

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- 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:

- 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

■ Followed acute or short term repeated exposures to ethylene glycol monoalkyl ethers and their acetates:

- Hepatic metabolism produces ethylene glycol as a metabolite.
- Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.
- Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure. [Ellenhorn and Barceloux: Medical Toxicology].

Treat symptomatically.

For acute or short term repeated exposures to ethylene glycol:

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

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

For acute or short term repeated exposures to acetone:

- Symptoms of acetone exposure approximate ethanol intoxication.
- About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours

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following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.

- There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care. [Ellenhorn and Barceloux: Medical Toxicology]

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

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

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

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

Oral Management:
- No GASTRIC LAVAGE OR EMETIC
- Encourage oral fluids.

Systemic Management:
- Monitor blood glucose and arterial pH.
- Ventilate if respiratory depression occurs.
- If patient unconscious, monitor renal function.
- Symptomatic and supportive care.

The Chemical Incident Management Handbook:
Guy's and St. Thomas' Hospital Trust, 2000

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

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

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

Following acute or short term repeated exposures to toluene:
- Toluene is absorbed across the alveolar barrier, the blood/air mixture being 11.2/15.6 (at 37 degrees C.) The concentration of toluene, in expired breath, is of the order of 18 ppm following sustained exposure to 100 ppm. The tissue/blood proportion is 1/3 except in adipose where the proportion is 8/10.
- Metabolism by microsomal mono-oxygenation, results in the production of hippuric acid. This may be detected in the urine in amounts between 0.5 and 2.5 g/24 hr which represents, on average 0.8 gm/gm of creatinine. The biological half-life of hippuric acid is in the order of 1-2 hours.
- Primary threat to life from ingestion and/or inhalation is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (eg cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 <50 mm Hg or pCO2 > 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial damage has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.

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- 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.

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

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Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

■ SMALL FIRE:

- Water spray, dry chemical or CO_2

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.

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- 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 monoxide (CO), carbon dioxide (CO2), phosphorus oxides (POx), other pyrolysis products typical of burning organic material.
Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.

FIRE INCOMPATIBILITY

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

HAZCHEM

2YE

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

- 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.

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

PROTECTIVE ACTIONS FOR SPILL

COULD NOT FIND ISOPAZON.WMF

From IERG (Canada/Australia)

Isolation Distance	-
Downwind Protection Distance	8 metres
IERG Number	49

FOOTNOTES

1 PROTECTIVE ACTION ZONE is defined as the area in which people are at risk of harmful exposure. This zone assumes that random changes in wind direction confines the vapour plume to an area within 30 degrees on either side of the predominant wind direction, resulting in a crosswind protective action distance equal to the downwind protective action distance.

2 PROTECTIVE ACTIONS should be initiated to the extent possible, beginning with those closest to the spill and working away from the site in the downwind direction. Within the protective action zone a level of vapour concentration may exist resulting in nearly all unprotected persons becoming incapacitated and unable to take protective action and/or incurring serious or irreversible health effects.

3 INITIAL ISOLATION ZONE is determined as an area, including upwind of the incident, within which a high probability of localised wind reversal may expose nearly all persons without appropriate protection to life-threatening concentrations of the material.

4 SMALL SPILLS involve a leaking package of 200 litres (55 US gallons) or less, such as a drum (jerrican or box with inner containers). Larger packages leaking less than 200 litres and compressed gas leaking from a small cylinder are also considered "small spills".

LARGE SPILLS involve many small leaking packages or a leaking package of greater than 200 litres, such as a cargo tank, portable tank or a "one-tonne" compressed gas cylinder.

5 Guide 126 is taken from the US DOT emergency response guide book.

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6 IERG information is derived from CANUTEC - Transport Canada.

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
n-butyl acetate	3000ppm

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

toluene	300ppm
n-butyl acetate	200ppm

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

toluene	50ppm
n-butyl acetate	5ppm

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 conditions are maintained.

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SUITABLE CONTAINER

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

STORAGE INCOMPATIBILITY

- Avoid reaction with oxidising agents.

PACKAGING MATERIAL INCOMPATIBILITIES

Chemical Name	Container Type
Xylene	" ABS plastic" , " Buna N (Nitrile)" , CPVC, EPDM, Hypalonr, " Natural rubber" , Neoprene, Polycarbonate, Polyurethane, PVC, Silicone, Tygonr

STORAGE REQUIREMENTS

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

SAFE STORAGE WITH OTHER CLASSIFIED CHEMICALS

+	X	X	X	X	+

+: *May be stored together*
O: *May be stored together with specific preventions*
X: *Must not be stored together*

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m³	STEL ppm	STEL mg/m³	Notes
Australia Exposure Standards	Dy- Mark Rust Reformer Aerosol (Xylene (o- , m- , p- isomers))	80	350	150	655	
Australia Exposure Standards	acetone (Acetone)	500	1185	1000	2375	
Australia Exposure Standards	toluene (Toluene)	50	191	150	574	Sk

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Source	Material	TWA ppm	TWA mg/m³	STEL ppm	STEL mg/m³	Notes
Australia Exposure Standards	n- butyl acetate (n-Butyl acetate)	150	713	200	950	
Australia Exposure Standards	white spirit (White spirits)		790			(see Chapter 16)
Australia Exposure Standards	white spirit (Petrol (gasoline))		900			(see Chapter 16)
Australia Exposure Standards	ethylene glycol monobutyl ether (2-Butoxyethanol)	20	96.9	50	242	Sk
Australia Exposure Standards	ethylbenzene (Ethyl benzene)	100	434	125	543	
Australia Exposure Standards	hydrocarbon propellant (LPG (liquified petroleum gas))	1000	1800			

EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m3)	Revised IDLH Value (ppm)
acetone		2,500 [LEL]
toluene		500
n-butyl acetate		1,700 [LEL]
white spirit	20,000	
xylene		900
ethylene glycol monobutyl ether		700 [Unch]
ethylbenzene		800 [LEL]
hydrocarbon propellant		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.

ODOUR SAFETY FACTOR (OSF)
OSF=0.042 (white spirit)
■ Exposed individuals are NOT reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

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

Classification into classes follows:

Class	OSF	Description

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

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MATERIAL DATA

ACETONE:
ETHYLBENZENE:
ETHYLENE GLYCOL MONOBUTYL ETHER:
N-BUTYL ACETATE:

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

Odour Safety Factor (OSF) is determined to fall into either Class A or B.
The Odour Safety Factor (OSF) is defined as:
OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm
Classification into classes follows:

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

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DY-MARK RUST REFORMER AEROSOL:
ETHYLBENZENE:

■ for ethyl benzene:
Odour Threshold Value: 0.46-0.60 ppm
NOTE: Detector tubes for ethylbenzene, measuring in excess of 30 ppm, are commercially available.
Ethyl benzene produces irritation of the skin and mucous membranes and appears to produce acute and chronic effects on the central nervous system. Animal experiments also suggest the effects of chronic exposure include damage to the liver, kidneys and testes. In spite of structural similarities to benzene, the material does not appear to cause damage to the haemopoietic system. The TLV-TWA is thought to be protective

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against skin and eye irritation. Exposure at this concentration probably will not result in systemic effects.

Subjects exposed at 200 ppm experienced transient irritation of the eyes; at 1000 ppm there was eye irritation with profuse lachrymation; at 200 ppm eye irritation and lachrymation were immediate and severe accompanied by moderate nasal irritation, constriction in the chest and vertigo; at 5000 ppm exposure produced intolerable irritation of the eyes and throat.

Odour Safety Factor(OSF)

OSF=43 (ETHYL BENZENE).

DY-MARK RUST REFORMER AEROSOL:
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).

ACETONE:
DY-MARK RUST REFORMER AEROSOL:

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

Saturation vapour concentration: 237000 ppm @ 20 C

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

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

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

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

Odour Safety Factor(OSF)

OSF=38 (ACETONE).

DY-MARK RUST REFORMER AEROSOL:
WHITE SPIRIT:

■ Odour Threshold Value: 34 ppm (detection), 97 ppm (recognition)

NOTE: Detector tubes for benzene, measuring in excess of 0.5 ppm, are commercially available. The relative quality of epidemiological data and quantitative health risk assessments related to documented and theoretical leukaemic deaths constitute the basis of the TLV-recommendation.

One study [Dow Chemical] demonstrates a significant fourfold increase in myelogenous leukaemia for workers exposed to average benzene concentrations of about 5 ppm for an average of 9 years and that 2 out of four

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individuals in the study who died from leukaemia were characterised as having been exposed to average benzene levels below 2 ppm. Based on such findings the estimated risk of leukaemia in workers exposed at daily benzene concentrations of 10 ppm for 40 years is 155 times that of unexposed workers; at 1 ppm the risk falls to 1.7 times whilst at 0.1 ppm the risk is about the same in the two groups. A revision of the TLV-TWA to 0.1 ppm was proposed in 1990 but this has been revised upwards as result of industry initiatives.

Typical toxicities displayed following inhalation:

- At 25 ppm (8 hours): no effect
- 50-150 ppm: signs of intoxication within 5 hours
- 500-1500 ppm: signs of intoxication within 1 hour
- 7500 ppm: severe intoxication within 30-60 minutes
- 20000 ppm: fatal within 5-10 minutes

Some jurisdictions require that health surveillance be conducted on occupationally exposed workers. Some surveillance should emphasise (i) demography, occupational and medical history and health advice (ii) baseline blood sample for haematological profile (iii) records of personal exposure.

DY-MARK RUST REFORMER AEROSOL:
N-BUTYL ACETATE:

■ For n-butyl acetate

Odour Threshold Value: 0.0063 ppm (detection), 0.038-12 ppm (recognition)

Exposure at or below the recommended TLV-TWA is thought to prevent significant irritation of the eyes and respiratory passages as well as narcotic effects. In light of the lack of substantive evidence regarding teratogenicity and a review of acute oral data a STEL is considered inappropriate.

Odour Safety Factor(OSF)

OSF=3.8E2 (n-BUTYL ACETATE).

TOLUENE:
XYLENE:

■ 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.

DY-MARK RUST REFORMER AEROSOL:
XYLENE:

■ 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).

DY-MARK RUST REFORMER AEROSOL:
ETHYLENE GLYCOL MONOBUTYL ETHER:

■ For ethylene glycol monobutyl ether (2-butoxyethanol)

continued...

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

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

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL).

WHITE SPIRIT:

■ Odour threshold: 0.25 ppm.

The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m3 is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.

Odour Safety Factor (OSF)

OSF=0.042 (gasoline).

For white spirit:

Low and high odour thresholds of 5.25 and 157.5 mg/m3, respectively, were considered to provide a rather useful index of odour as a warning property.

The TLV-TWA is calculated from data on the toxicities of the major ingredients and is intended to minimise the potential for irritative and narcotic effects, polyneuropathy and kidney damage produced by vapours.

The NIOSH (USA) REL-TWA of 60 ppm is the same for all refined petroleum solvents. NIOSH published an occupational "action level" of 350 mg/m3 for exposure to Stoddard solvent, assuming a 10-hour work shift and a 40-hour work-week. The NIOSH-REL ceiling of 1800 mg/m3 was established to protect workers from short-term effects that might produce vertigo or other adverse effects which might increase the risk of occupational accidents. Combined (gross) percutaneous absorption and inhalation exposure (at concentrations associated with nausea) are thought, by some, to be responsible for the development of frank hepatic toxicity and jaundice.

Odour Safety Factor (OSF)

OSF=0.042 (white spirit).

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

continued...

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].

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.

BRETHERICK: Handbook of Reactive Chemical Hazards.

RESPIRATOR

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

■ General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.
Provide adequate ventilation in warehouse or closed storage areas.
Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents

continued...

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.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

■ Supplied as an aerosol pack. Contents under PRESSURE. Contains highly flammable hydrocarbon propellant.
Black flammable liquid; 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

acetone	
■ log Kow (Prager 1995):	- 0.24
■ log Kow (Sangster 1997):	- 0.24
toluene	
■ log Kow (Sangster 1997):	2.73
n- butyl acetate	
■ log Kow (Prager 1995):	1.82
■ log Kow (Sangster 1997):	1.78
xylene	
■ log Kow (Prager 1995):	3.12- 3.20
ethylene glycol monobutyl ether	
■ log Kow (Prager 1995):	0.83
■ log Kow (Sangster 1997):	0.8
ethylbenzene	

continued...

■ log Kow (Prager 1995):	3.15
■ log Kow (Sangster 1997):	3.15

Section 10 - CHEMICAL STABILITY

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.
Severe acute exposure to ethylene glycol monobutyl ether, by ingestion, may cause kidney damage, haemoglobinuria, (blood in urine) and is potentially fatal.
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

■ 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.
Not considered to be a risk because of the extreme volatility of the gas.
Two drops of the ethylbenzene in to the conjunctival sac produced only slight irritation of the conjunctival membrane but no corneal injury.
When instilled in rabbit eyes ethylene glycol monobutyl ether produced pain, conjunctival irritation, and transient corneal injury.
The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration.
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.

SKIN

■ Skin contact with the material may be harmful; systemic effects may result following absorption.
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.

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Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.
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.
The mean rate of absorption of liquid ethyl benzene applied to 17.3 cm2 area of the forearm of seven volunteers for 10-15 minutes was determined to be 38 mg/cm2/hr. Immersion of the whole hand in aqueous solutions of ethyl benzene (112-156 mg/l) for 1 hour yielded mean absorption rates of 118 and 215.7 ug/cm2/hr. The rate of absorption is thus greater than that of aniline, benzene, nitrobenzene, carbon disulfide and styrene.
Repeated application of the undiluted product to the abdominal area of rabbits (10-20 applications over 2-4 weeks) resulted in erythema, oedema and superficial necrosis. The material did not appear to be absorbed through the skin in sufficient quantity to produce outward signs of toxicity.
Ethylene glycol monobutyl ether (2-butoxyethanol) penetrates the skin easily and toxic effects via this route may be more likely than by inhalation. Percutaneous uptake rate in the guinea pig was estimated to be 0.25 umole/min/cm2.

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

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

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.
When humans were exposed to the 100 and 200 ppm for 8 hours about 45-65% is retained in the body. Only traces of unchanged ethyl benzene are excreted in expired air following termination of inhalation exposure.
Humans exposed to concentrations of 23-85 ppm excreted most of the retained dose in the urine (mainly as metabolites). Guinea pigs that died from exposure had intense congestion of the lungs and generalised visceral hyperaemia. Rats exposed for three days at 8700 mg/m3 (2000 ppm) showed changes in the levels of dopamine and noradrenaline in various parts of the brain.
Ethylene glycol monobutyl ether (2-butoxyethanol) and its metabolite butoxyacetic acid are haemolytic agents, causing red blood cell destruction.
On the basis of industrial experience and volunteer short-term exposure humans are shown to be less susceptible than experimental animals to exposure. In 8-hour exposures at concentrations of 200 or 100 ppm no objective effects were seen other than raised urinary excretion of the metabolite butoxyacetic acid. No increased osmotic fragility of the red blood cell is observed. Subjectively these concentrations were uncomfortable with mild eye, nose and throat irritation occurring. No clinical signs of adverse effects nor

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subjective complaints were produced when male volunteers were exposed for 2 hours to 20 ppm during light physical exercise. Other studies have established that the most sensitive indicators of toxic effect observed from many of the glycol ethers is an increase in erythrocyte osmotic fragility in rats. This appears to be related to the development of haemoglobinuria at higher exposure levels.
Headache, fatigue, tiredness, irritability and digestive disturbances (nausea, loss of appetite and bloating) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Temporary memory loss, kidney impairment, temporary confusion and some evidence of disturbance of liver function was reported in workers grossly exposed to xylene (1%). One death was noted, with autopsy revealing lung congestion, oedema and local bleeding of alveoli. Inhaling xylene at 100 ppm for 5-6 hours can increase reaction time and cause slight inco-ordination. Tolerance developed during the work week, but was lost over the weekend. Physical exercise may reduce tolerance. About 4-8% of total absorbed xylene accumulates in fat.
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.
Inhalation of acetone causes central nervous system depression, light-headedness, incoherent speech, inco-ordination, stupor, low blood pressure, fast pulse, metabolic acidosis, high blood sugar and ketosis. Rarely, convulsions and tubular necrosis may be evident. Other symptoms of exposure may include restlessness, headache, vomiting, low blood pressure and rapid and irregular pulse, eye and throat irritation, weakness of the legs and dizziness. Inhalation of high concentrations may produce dryness of the mouth and throat, nausea, inco-ordinated movement, loss of co-ordinated speech, drowsiness, and in severe cases, coma. Inhalation of acetone vapours over long periods causes irritation of the airways, coughing and headache. Rats exposed to a concentration of 5.22% for 1 hour showed clear signs of sleepiness; deaths occurred at 12.66%.
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

■ Harmful: danger of serious damage to health by prolonged exposure through inhalation.
Harmful: danger of serious damage to health by prolonged exposure through inhalation.
This material can cause serious damage if one is exposed to it for long periods. It can be assumed that it contains a substance which can produce severe defects. This has been demonstrated via both short- and long-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.
Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.
Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure.

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Based on experience with similar materials, there is a possibility that exposure to the material may reduce fertility in humans at levels which do not cause other toxic effects.
Principal route of occupational exposure to the gas is by inhalation.
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.
Industrial workers exposed to a maximum level of ethylbenzene 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.
Prolonged and repeated exposure may be harmful to the central nervous system (CNS), upper respiratory tract, and/ or may cause liver disorders. It may also cause drying, scaling and blistering of the skin.
Rats and mice exposed to ethylbenzene for 6 hours daily, 5 days a week for 104 and 103 weeks respectively showed a statistically significant increase in kidney tumours in male and female rats, lung tumours in male mice, and liver tumours in female mice exposed to 750 ppm ethylbenzene.
Women exposed to xylene in the first 3 months of pregnancy showed a slightly increased risk of miscarriage and birth defects. Evaluation of workers chronically exposed to xylene has demonstrated lack of genetic toxicity. Exposure to xylene has been associated with increased rates of blood cancer, but this may be complicated by exposure to other substances, including benzene. Animal testing found no evidence of cancer-causing activity.
Workers exposed to acetone for long periods showed inflammation of the airways, stomach and small bowel, attacks of giddiness and loss of strength. Exposure to acetone may enhance the liver toxicity of chlorinated solvents.

TOXICITY AND IRRITATION

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

■ 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 on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylgloxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances.
Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys.
Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (400 ppm and greater) of ethylbenzene
In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncitial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland.
In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted

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in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity
Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination.
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 lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .
Developmental/Reproductive Toxicity
Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.
Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy
Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m3 toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m3 8 hours/day during days 1-21 of gestation. No maternal deaths or

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toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m3 toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m3. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.
Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.
Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .
Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .
Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites
Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.
for acetone:
The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15, 665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice.
Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.
The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.

XYLENE:
ETHYLENE GLYCOL MONOBUTYL ETHER:
ETHYLBENZENE:
N-BUTYL ACETATE:
■ The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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ETHYLENE GLYCOL MONOBUTYL ETHER:
ETHYLBENZENE:
ACETONE:
■ 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.

N-BUTYL ACETATE:
XYLENE:
TOLUENE:
■ 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.

ACETONE:

TOXICITY	IRRITATION
Oral (man) TDLo: 2857 mg/kg	Eye (human): 500 ppm - Irritant
Oral (rat) LD50: 5800 mg/kg	Eye (rabbit): 3.95 mg - SEVERE
Inhalation (human) TCLo: 500 ppm	Eye (rabbit): 20mg/24hr - Moderate
Inhalation (man) TCLo: 12000 ppm/4 hr	Skin (rabbit):395mg (open) - Mild
Inhalation (man) TCLo: 10 mg/m³/6 hr	Skin (rabbit): 500 mg/24hr - Mild
Inhalation (rat) LC50: 50100 mg/m³/8 hr	
Dermal (rabbit) LD50: 20000 mg/kg	

■ for acetone:
The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15, 665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice.
Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.
The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.

TOLUENE:

TOXICITY	IRRITATION
Oral (human) LDLo: 50 mg/kg	Skin (rabbit):20 mg/24h- Moderate
Oral (rat) LD50: 636 mg/kg	Skin (rabbit):500 mg - Moderate
Inhalation (human) TCLo: 100 ppm	Eye (rabbit):0.87 mg - Mild
Inhalation (man) TCLo: 200 ppm	Eye (rabbit): 2mg/24h - SEVERE

continued...

Inhalation (rat) LC50: >26700 ppm/1h
Dermal (rabbit) LD50: 12124 mg/kg
Eye (rabbit):100 mg/30sec - Mild

■ 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 lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .
Developmental/Reproductive Toxicity
Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.
Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy
Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m3 toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m3 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m3 toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m3. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the

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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.

N-BUTYL ACETATE:

TOXICITY	IRRITATION
Oral (rat) LD50: 13100 mg/kg	Skin (rabbit): 500 mg/24h- Moderate
Dermal (rabbit) LD50: 3200 mg/kg*	Eye (rabbit): 20 mg (open)- SEVERE
Inhalation (human) TCLo: 200 ppm	Eye (rabbit): 20 mg/24h - Moderate
Inhalation (rat) LC50: 2000 ppm/4h	Eye (human): 300 mg
Inhalation (Human) TCLo: 200 ppm/4h * [PPG]	
Oral (Rat) LD50: 10768 mg/kg	
Inhalation (Rat) LC50: 390 ppm/4h	
Intraperitoneal (Mouse) LD50: 1230 mg/kg	
Oral (Rabbit) LD50: 3200 mg/kg	
Oral (Guinea pig) LD50: 4700 mg/kg	
Intraperitoneal (Guinea pig) LD: 1500 mg/kg	

WHITE SPIRIT:

TOXICITY	IRRITATION
Inhalation (human) TCLo: 600 mg/m³/8h	Nil Reported
Oral (rat) LD50: >5000 mg/kg	Eye (human): 470 ppm/15m
Inhalation (rat) LC50: >5500 mg/m³/4h	Eye (rabbit): 500 mg/24h Moderate

■ for petroleum:
This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.
This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.
This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents
Carcinogenicity: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.
Mutagenicity: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.
Reproductive Toxicity: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.
Human Effects: Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and

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may make the skin more susceptible to irritation and penetration by other materials.

Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.
white spirit, as CAS RN 8052-41-3

XYLENE:

TOXICITY	IRRITATION
Oral (human) LDLo: 50 mg/kg	Skin (rabbit):500 mg/24h Moderate
Oral (rat) LD50: 4300 mg/kg	Eye (human): 200 ppm Irritant
Inhalation (human) TCLo: 200 ppm	Eye (rabbit): 87 mg Mild
Inhalation (man) LCLo: 10000 ppm/6h	Eye (rabbit): 5 mg/24h SEVERE
Inhalation (rat) LC50: 5000 ppm/4h	
Oral (Human) LD: 50 mg/kg	
Inhalation (Human) TCLo: 200 ppm/4h	
Intraperitoneal (Rat) LD50: 2459 mg/kg	
Subcutaneous (Rat) LD50: 1700 mg/kg	
Oral (Mouse) LD50: 2119 mg/kg	
Intraperitoneal (Mouse) LD50: 1548 mg/kg	
Intravenous (Rabbit) LD: 129 mg/kg	
Inhalation (Guinea) pig: LC 450 ppm/4h	

■ 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.
Reproductive effector in rats

ETHYLENE GLYCOL MONOBUTYL ETHER:

TOXICITY	IRRITATION
Oral (rat) LD50: 470 mg/kg	Skin (rabbit): 500 mg, open; Mild
Dermal (rabbit) LD50: 220 mg/kg	Eye (rabbit): 100 mg/24h- Moderate
Inhalation (human) TCLo: 100 ppm	Eye (rabbit): 100 mg SEVERE
Inhalation (human) TCLo: 195 ppm/8h * [Union Carbide]	
Inhalation (Rat) LC50: 450 ppm *	

■ For ethylene glycol:
Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.
dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.
Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate

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crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

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

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases.Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

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

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

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

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crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.
Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.
Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.
Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.
Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.
Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available in vivo and in vitro laboratory studies provide consistently negative genotoxicity results for ethylene glycol.
For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):
Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.
EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.
Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE in vitro than those of rats.
Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA in vitro and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA in vitro.
Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in S. typhimurium strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. In vitro cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not

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genotoxic.
Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity
Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).
Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.
The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).
Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.
At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.
Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.
Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland.
In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.
1: NTP Toxicology Program Technical report Series 484, March 2000.
NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes.

ETHYLBENZENE:

TOXICITY	IRRITATION
Oral (rat) LD50: 3500 mg/kg	Skin (rabbit): 15 mg/24h Mild
Inhalation (human) TCLo: 100 ppm/8h	Eye (rabbit): 500 mg - SEVERE
Inhalation (rat) LCLo: 4000 ppm/4h	
Intraperitoneal (mouse) LD50: 2642 mg/kg	
Dermal (rabbit) LD50: 17800 mg/kg	
Inhalation (Rat) LC: 4000 ppm/4h	

■ Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-

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enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylglyoxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances.
Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys. Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (400 ppm and greater) of ethylbenzene
In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncitial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland.
In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity
Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination.
NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.
WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
Liver changes, utheral tract, effects on fertility, foetotoxicity,
specific developmental abnormalities (musculoskeletal system) recorded.

HYDROCARBON PROPELLANT:
■ Not available. Refer to individual constituents.

REPROTOXIN

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

Section 12 - ECOLOGICAL INFORMATION

Refer to data for ingredients, which follows:

ACETONE:
TOLUENE:
N-BUTYL ACETATE:
WHITE SPIRIT:
XYLENE:
ETHYLENE GLYCOL MONOBUTYL ETHER:
ETHYLBENZENE:
HYDROCARBON PROPELLANT:
DY-MARK RUST REFORMER AEROSOL:

continued...

■ DO NOT discharge into sewer or waterways.

XYLENE:
ETHYLBENZENE:
DY-MARK RUST REFORMER AEROSOL:
■ Harmful to aquatic organisms.

ETHYLBENZENE:
DY-MARK RUST REFORMER AEROSOL:
■ For ethylbenzene:
log Kow, 3.15
log Koc : 1.98-3.04
Koc : 164
log Kom : 1.73-3.23
Vapour Pressure, 1270 Pa (1.27 kPa)
Half-life (hr) air : 0.24-85.6
Half-life (hr) H2O surface water : 5-240
Half-life (hr) H2O ground : 144-5472
Half-life (hr) soil : 72-240
Henry's Pa m3 /mol: 748-887
Henry's atm m3 /mol: 8.44E-03
ThOD : 3.17
BCF : 3.15-146
log BCF : 1.19-2.67
Water solubility, 169 mg/l at 25 C
Environmental fate:
Ethylbenzene partitions to air from water and soil, and is degraded in air. Ethylbenzene is volatile and when released will quickly vaporize. Photodegradation is the primary route of removal in the environment. Photodegradation is estimated with a half-life of 1 day. Ethylbenzene is considered inherently biodegradable and removal from water occurs primarily by evaporation but in the summer biodegradation plays a key role in the removal process. Level I and Level III fugacity modeling indicate that partitioning is primarily to the air compartment, 98 and 96%, respectively. Ethylbenzene is inherently biodegradable in water and in soil under aerobic conditions, and not rapidly biodegradable in anaerobic conditions. Ethylbenzene is expected to be moderately adsorbed to soil.
Based on measured data, ethylbenzene is not expected to bioaccumulate (BCF 1.1-15).
Ecotoxicity:
In acute aquatic toxicity testing LC50 values range approximately between 1 and 10 mg/l. In acute aquatic fish tests (fresh water species), the 96-hr LC50 for Pimephales promelas and Oncorhynchus mykiss are 12.1 and 4.2 mg/L, respectively. Data are available in the saltwater species Menidia menidia and give results within the same range as for the fresh water species with a 96-hr LC50 = 5.1 mg/L. In fresh water invertebrate species Daphnia magna and Ceriodaphia dubia, 48-hr LC50 values were 1.81 and 3.2 mg/L, respectively. Additional data is available in the saltwater species Crangon franciscorium (96-hr LC50 = 0.49 mg/L) and Mysidopsis bahia (96-hr LC50 = 2.6 mg/L). In 96-hr algal toxicity testing, results indicate that ethylbenzene inhibits algae growth in Selenastrum capricornatum at 3.6 mg/L and in Skeletonema costatum at 7.7 mg/L.

ETHYLENE GLYCOL MONOBUTYL ETHER:
DY-MARK RUST REFORMER AEROSOL:
■ For ethylene glycol monoalkyl ethers and their acetates:
Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE)
Environmental fate:
The ethers, like other simple glycol ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions.

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Level III fugacity modeling indicates that category members, when released to air and water, will partition predominately to water and, to a lesser extent, to air and soil. Estimates of soil and sediment partition coefficients (Kocs ranging from 1- 10) suggest that category members would exhibit high soil mobility. Estimated bioconcentration factors (log BCF) range from 0.463 to 0.732. Biodegradation studies indicate that all category members are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment.
Ecotoxicity:
Glycol ether acetates do not hydrolyse rapidly into their corresponding glycol ethers in water under environmental conditions. The LC50 or EC50 values for EGHE are lower than those for EGPE and EGBE (which have shorter chain lengths and lower log Kow values). Overall, the LC50 values for the glycol ethers in aquatic species range from 94 to > 5000 mg/L. For EGHE, the 96-hour LC50 for Brachydanio rerio (zebra fish) is between 94 and mg/L, the 48-hour EC50 for Daphnia magna was 145 mg/L and the 72-hour EC50 values for biomass and growth rate of algae (Scenedesmus subspicatus) were 98 and 198 mg/L, respectively. LC50/EC50 values for EGPE and EGBE in aquatic species are 835 mg/l or greater.
Aquatic toxicity data for EGBEA show a 96-hour LC50 of 28.3 mg/L for rainbow trout (Oncorhynchus mykiss), a 48-hour LC50 of 37-143 mg/L for Daphnia magna, a 72-hour EC50 of greater than 500 mg/L for biomass or growth rate of algae (Scenedesmus subspicatus and Pseudokirchneriella subcapitata, respectively), and a 7-day EC10 of 30.4 mg/L and a NOEC of 16.4 mg/L for reproduction in Ceriodaphnia dubia.

XYLENE:
DY-MARK RUST REFORMER AEROSOL:
■ 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

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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-dimethyl-p-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.

N-BUTYL ACETATE:
DY-MARK RUST REFORMER AEROSOL:
■ For n-butyl acetate:
Half-life (hr) air : 144
Half-life (hr) H2O surface water : 178-27156
Henry's atm m3 /mol: 3.20E-04
BOD 5 if unstated: 0.15-1.02,7%
COD : 78%
ThOD : 2.207
BCF : 4-14
Environmental Fate:
TERRESTRIAL FATE: An estimated Koc value of 200 determined from a measured log Kow of 1.78 indicates that n-butyl acetate is expected to have moderate mobility in soil. Volatilisation of n-butyl acetate is expected from moist soil surfaces given its Henry's Law constant of 2.8x10-4 atm-cu m/mole. Volatilisation from dry soil surfaces is expected based on a measured vapor pressure of 11.5 mm Hg. Using a standard BOD dilution technique and a sewage inoculum, theoretical BODs of 56 % to 86 % were observed during 5-20 day incubation periods, which suggests that n-butyl acetate may biodegrade in soil.
AQUATIC FATE: An estimated Koc value indicates that n-butyl acetate is not expected to adsorb to suspended solids and sediment in water. Butyl acetate is expected to volatilise from water surfaces based on a Henry's Law constant of 2.8x10-4 atm-cu m/mole. Estimated half-lives for a model river and model lake are 7 and 127, hours respectively. An estimated BCF value of 10 based on the log Kow, suggests that bioconcentration in aquatic organisms is low. Using a filtered sewage seed, 5-day and 20-day theoretical BODs of 58 % and 83 % were measured in freshwater dilution tests; 5-day and 20-day theoretical BODs of 40 % and 61 % were measured in salt water. A 5-day theoretical BOD of 56.8 % and 51.8 % were measured for n-butyl acetate in distilled water and seawater, respectively. Hydrolysis may be an important environmental fate for this compound based upon experimentally determined hydrolysis half-lives of 114 and 11 days at pH 8 and 9 respectively.
ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the

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atmosphere, n-butyl acetate, which has a vapour pressure of 11.5 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapour-phase n-butyl acetate 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 4 days
Environmental fate:
Fish LC50 (96 h, 23 C): island silverside (Menidia beryllina) 185 ppm (static bioassay in synthetic seawater, mild aeration applied after 24 h); bluegill sunfish (Lepomis macrochirus) 100 ppm (static bioassay in fresh water, mild aeration applied after 24 h)
Fish EC50 (96 h): fathead minnow (Pimephales promelas) 18 mg/l (affected fish lost equilibrium prior to death)
Daphnia LC50 (48 h): 44 ppm
Algal LC50 (96 h): Scenedesmus 320 ppm.

TOLUENE:
DY-MARK RUST REFORMER AEROSOL:
■ For toluene:
log Kow : 2.1-3
log Koc : 1.12-2.85
Koc : 37-260
log Kom : 1.39-2.89
Half-life (hr) air : 2.4-104
Half-life (hr) H2O surface water : 5.55-528
Half-life (hr) H2O ground : 168-2628
Half-life (hr) soil : <48-240
Henry's Pa m3 /mol: 518-694
Henry's atm m3 /mol: 5.94E-03
BOD 5 0.86-2.12, 5%
COD : 0.7-2.52,21-27%
ThOD : 3.13
BCF : 1.67-380
log BCF : 0.22-3.28
Environmental fate:
Transport: The majority of toluene evaporates to the atmosphere from the water and soil.It is moderately retarded by adsorption to soils rich in organic material (Koc = 259), therefore, transport to ground water is dependent on the soil composition. In unsaturated topsoil containing organic material, it has been estimated that 97% of the toluene is adsorbed to the soil and only about 2% is in the soil-water phase and transported with flowing groundwater. There is little retardation in sandy soils and 2-13% of the toluene was estimated to migrate with flowing water; the remainder was volatilised, biodegraded, or unaccounted for. In saturated deep soils with no soil-air phase, about 48% may be transported with flowing groundwater.
Transformation/Persistence:
Air - The main degradation pathway for toluene in the atmosphere is reaction with photochemically produced hydroxyl radicals. The estimated atmospheric half life for toluene is about 13 hours. Toluene is also oxidised by reactions with atmospheric nitrogen dioxide, oxygen, and ozone, but these are minor degradation pathways. Photolysis is not considered a significant degradative pathway for toluene
Soil - In surface soil, volatilisation to air is an important fate process for toluene. Biodegradation of toluene has been demonstrated in the laboratory to occur with a half life of about 1 hour. In the environment, biodegradation of toluene to carbon dioxide occurs with a typical half life of 1-7 days.
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

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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).

ACETONE:
DY-MARK RUST REFORMER AEROSOL:
■ for acetone:
log Kow: -0.24
Half-life (hr) air: 312-1896
Half-life (hr) H2O surface water: 20
Henry's atm m3 /mol: 3.67E-05
BOD 5: 0.31-1.76,46-55%
COD: 1.12-2.07
ThOD: 2.2
BCF: 0.69
Environmental fate:
Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water
In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.
Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.
Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.
Acetone does not concentrate in the food chain.
Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period
Drinking Water Standard: none available.
Soil Guidelines: none available.
Air Quality Standards: none available.
Ecotoxicity:
Testing shows that acetone exhibits a low order of toxicity
Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l
Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l
Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l
Aquatic invertebrate 2100 - 16700 mg/l
Aquatic plant NOEC: 5400-7500 mg/l
Daphnia magna chronic NOEC 1660 mg/l
Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (Tribolium confusum) and the flour moth (Ephestia kuehniella) were exposed to an airborne acetone concentration of 61.5 mg/m3. The LT50 values

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for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.
The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (Entosiphon sulcatum) which yielded a 3-day NOEC of 28 mg/L.
■ For ketones:
Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds
Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrateThe higher molecular weight ketones do no form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions
Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (e.g., ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable.
Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.

DY-MARK RUST REFORMER AEROSOL:
■ 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.

ACETONE:

■ Fish LC50 (96hr.) (mg/l):	8300- 40000
■ Daphnia magna EC50 (48hr.) (mg/l):	10
■ log Kow (Prager 1995):	- 0.24
■ log Kow (Sangster 1997):	- 0.24
■ log Pow (Verschueren 1983):	- 0.24
■ BOD5:	122%
■ ThOD:	72
■ Half- life Soil - High (hours):	168
■ Half- life Soil - Low (hours):	24
■ Half- life Air - High (hours):	2790
■ Half- life Air - Low (hours):	279

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■ Half- life Surface water - High (hours):	168
■ Half- life Surface water - Low (hours):	24
■ Half- life Ground water - High (hours):	336
■ Half- life Ground water - Low (hours):	48
■ Aqueous biodegradation - Aerobic - High (hours):	168
■ Aqueous biodegradation - Aerobic - Low (hours):	24
■ Aqueous biodegradation - Anaerobic - High (hours):	672
■ Aqueous biodegradation - Anaerobic - Low (hours):	96
■ Aqueous biodegradation - Removal secondary treatment - High (hours):	75%
■ Aqueous biodegradation - Removal secondary treatment - Low (hours):	54%
■ Aqueous photolysis half- life - High (hours):	270
■ Photooxidation half- life water - High (hours):	3.97E+06
■ Photooxidation half- life water - Low (hours):	9.92E+04
■ Photooxidation half- life air - High (hours):	2790
■ Photooxidation half- life air - Low (hours):	279

TOLUENE:

■ Hazardous Air Pollutant:	Yes
■ Fish LC50 (96hr.) (mg/l):	7.3- 22.8
■ BCF<100:	13.2 (EELS
■ log Kow (Sangster 1997):	2.73
■ log Pow (Verschueren 1983):	2.69
■ BOD5:	5%
■ COD:	21%
■ ThOD:	3.13
■ Half- life Soil - High (hours):	528
■ Half- life Soil - Low (hours):	96
■ Half- life Air - High (hours):	104
■ Half- life Air - Low (hours):	10
■ Half- life Surface water - High (hours):	528
■ Half- life Surface water - Low (hours):	96
■ Half- life Ground water - High (hours):	672
■ Half- life Ground water - Low (hours):	168
■ Aqueous biodegradation - Aerobic - High (hours):	528
■ Aqueous biodegradation - Aerobic - Low (hours):	96
■ Aqueous biodegradation - Anaerobic - High (hours):	5040
■ Aqueous biodegradation - Anaerobic - Low (hours):	1344
■ Aqueous biodegradation - Removal secondary treatment - High (hours):	75%
■ Photolysis maximum light absorption - High (nano- m):	268
■ Photolysis maximum light absorption - Low (nano- m):	253.5
■ Photooxidation half- life water - High (hours):	1284
■ Photooxidation half- life water - Low (hours):	321
■ Photooxidation half- life air - High (hours):	104
■ Photooxidation half- life air - Low (hours):	10

N-BUTYL ACETATE:

■ Fish LC50 (96hr.) (mg/l):	18
■ Daphnia magna EC50 (48hr.) (mg/l):	44
■ log Kow (Prager 1995):	1.82
■ Fish LC50 (96hr.) (mg/l):	100- 185
■ Daphnia magna EC50 (48hr.) (mg/l):	44
■ Algae IC50 (72hr.) (mg/l):	280
■ log Kow (Sangster 1997):	1.78
■ COD:	78%

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WHITE SPIRIT:
■ For petroleum derivatives:
Chemical analysis for all individual compounds in a petroleum bulk product released to the environment is generally unrealistic due to the complexity of these mixtures and the laboratory expense. Determining the chemical composition of a petroleum release is further complicated by hydrodynamic, abiotic, and biotic processes that act on the release to change the chemical character.
The longer the release is exposed to the environment, the greater the change in chemical character and the harder it is to obtain accurate analytical results reflecting the identity of the release. After extensive weathering, detailed knowledge of the original bulk product is often less valuable than current site-specific information on a more focused set of hydrocarbon components. Health assessment efforts are frequently frustrated by three primary problems: (1) the inability to identify and quantify the individual compounds released to the environment as a consequence of a petroleum spill; (2) the lack of information characterizing the fate of the individual compounds in petroleum mixtures; and (3) the lack of specific health guidance values for the majority of chemicals present in petroleum products. To define the public health implications associated with exposure to petroleum hydrocarbons, it is necessary to have a basic understanding of petroleum properties, compositions, and the physical, chemical, biological, and toxicological properties of the compounds most often identified as the key chemicals of concern.
Environmental fate:
Petroleum products released to the environment migrate through soil via two general pathways: (1) as bulk oil flow infiltrating the soil under the forces of gravity and capillary action, and (2) as individual compounds separating from the bulk petroleum mixture and dissolving in air or water. When bulk oil flow occurs, it results in little or no separation of the individual compounds from the product mixture and the infiltration rate is usually fast relative to the dissolution rate. Many compounds that are insoluble and immobile in water are soluble in bulk oil and will migrate along with the bulk oil flow. Factors affecting the rate of bulk oil infiltration include soil moisture content, vegetation, terrain, climate, rate of release (e.g., catastrophic versus slow leakage), soil particle size (e.g., sand versus clay), and oil viscosity (e.g., gasoline versus motor oil).
As bulk oil migrates through the soil column, a small amount of the product mass is retained by soil particles. The bulk product retained by the soil particles is known as "residual saturation".
Depending upon the persistence of the bulk oil, residual saturation can potentially reside in the soil for years. Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater. Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater. When the amount of product released to the environment is small relative to the volume of available soil, all of the product is converted to residual saturation and downward migration of the bulk product usually ceases prior to affecting groundwater resources. Adverse impacts to groundwater may still occur if rain water infiltrates through soil containing residual saturation and initiates the downward migration of individual compounds. When the amount of product released is large relative to the volume of available soil, the downward migration of bulk product ceases as water-saturated pore spaces are encountered. If the density of the bulk product is less than that of water, the product tends to "float" along the interface between the water saturated and unsaturated zones and spread horizontally in a pancake-like layer, usually in the direction of groundwater flow. Almost all motor and heating oils are less dense than water. If the density of the bulk product is greater than that of water, the product will continue to migrate downward through the water table aquifer under the continued influence of gravity. Downward migration ceases when the product is converted to residual saturation or when an impermeable surface is encountered.
As the bulk product migrates through the soil column, individual compounds may separate from the mixture and migrate independently. Chemical transport properties such as volatility, solubility, and sorption potential are often used to evaluate and predict which compounds will likely separate from the mixture. Since petroleum products are complex mixtures of hundreds of compounds, the compounds characterized by relatively high vapor pressures tend to volatilise and enter the vapor phase. The exact composition of these vapors depends on the composition of the original product. Using gasoline as an example, compounds such as butane, propane, benzene,

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toluene, ethylbenzene and xylene are preferentially volatilised. Because volatility represents transfer of the compound from the product or liquid phase to the air phase, it is expected that the concentration of that compound in the product or liquid phase will decrease as the concentration in the air phase increases.
In general, compounds having a vapor pressure in excess of 10-2 mm Hg are more likely to be present in the air phase than in the liquid phase. Compounds characterized by vapor pressures less than 10-7 mm Hg are more likely to be associated with the liquid phase. Compounds possessing vapor pressures that are less than 10-2 mm Hg, but greater than 10-7 mm Hg, will have a tendency to exist in both the air and the liquid phases.
Lighter petroleum products such as gasoline contain constituents with higher water solubility and volatility and lower sorption potential than heavier petroleum products such as fuel oil.
Data compiled from gasoline spills and laboratory studies indicate that these light-fraction hydrocarbons tend to migrate readily through soil, potentially threatening or affecting groundwater supplies. In contrast, petroleum products with heavier molecular weight constituents, such as fuel oil, are generally more persistent in soils, due to their relatively low water solubility and volatility and high sorption capacity.
Solubility generally decreases with increasing molecular weight of the hydrocarbon compounds. For compounds having similar molecular weights, the aromatic hydrocarbons are more water soluble and mobile in water than the aliphatic hydrocarbons and branched aliphatics are less water-soluble than straight-chained aliphatics.
Aromatic compounds in petroleum fuels may comprise as much as 50% by weight; aromatic compounds in the C6-C13, range made up approximately 95% of the compounds dissolved in water.
Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds. Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media.
The final products of microbial degradation are carbon dioxide, water, and microbial biomass. The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics, and the aromatics in the C10-C22 range are the most readily biodegradable; n-alkanes, n-alkyl aromatics, and aromatics in the C5-C9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilisation and thus are unavailable in most environments; n-alkanes in the C1-C4 ranges are biodegradable only by a narrow range of specialized hydrocarbon degraders; and n-alkanes, n-alkyl aromatics, and aromatics above C22 are generally not available to degrading microorganisms.
Hydrocarbons with condensed ring structures, such as PAHs with four or more rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded.
A large proportion of the water-soluble fraction of the petroleum product may be degraded as the compounds go into solution. As a result, the remaining product may become enriched in the alicyclics, the highly branched aliphatics, and PAHs with many fused rings.
In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. Anaerobic decomposition of petroleum hydrocarbons leads to extremely low rates of degradation. The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is, greater than 7. The moisture content of the contaminated soil will affect biodegradation of oils due to dissolution of the residual compounds, dispersive actions, and the need for microbial metabolism to sustain high activity. The moisture content in soil affects microbial locomotion, solute diffusion, substrate supply, and the removal of metabolic by-products. Biodegradation rates in soils are also affected by the volume of product released to the environment. At concentrations of 0.5% of oil by volume, the degradation rate in soil is fairly independent of oil concentrations. However, as oil concentration rises, the first order degradation rate decreases and the oil degradation half-life increases. Ultimately, when the oil reaches saturation conditions in the soil (i.e., 30-50% oil), biodegradation virtually ceases.
Excessive moisture will limit the gaseous supply of oxygen for enhanced decomposition of petroleum hydrocarbons. Most studies indicate that optimum moisture content is within 50-70% of the water holding capacity.
All biological transformations are affected by temperature. Generally, as the temperature increases, biological activity tends to increase up to a temperature where enzyme denaturation occurs. The presence of oil should increase soil temperature, particularly at the surface. The darker color increases the heat capacity by adsorbing more radiation. The optimal temperature for biodegradation to occur ranges from 18 C to

continued...

30 C. Minimum rates would be expected at 5 C or lower.

XYLENE:

■ Fish LC50 (96hr.) (mg/l):	13.5
■ BCF<100:	2.14- 2.20
■ log Kow (Prager 1995):	3.12- 3.20
■ Half- life Soil - High (hours):	672
■ Half- life Soil - Low (hours):	168
■ Half- life Air - High (hours):	44
■ Half- life Air - Low (hours):	2.6
■ Half- life Surface water - High (hours):	672
■ Half- life Surface water - Low (hours):	168
■ Half- life Ground water - High (hours):	8640
■ Half- life Ground water - Low (hours):	336
■ Aqueous biodegradation - Aerobic - High (hours):	672
■ Aqueous biodegradation - Aerobic - Low (hours):	168
■ Aqueous biodegradation - Anaerobic - High (hours):	8640
■ Aqueous biodegradation - Anaerobic - Low (hours):	4320
■ Photolysis maximum light absorption - High (nano- m):	269.5
■ Photolysis maximum light absorption - Low (nano- m):	265
■ Photooxidation half- life water - High (hours):	2.70E+08
■ Photooxidation half- life water - Low (hours):	3.90E+05
■ Photooxidation half- life air - High (hours):	44
■ Photooxidation half- life air - Low (hours):	2.6

ETHYLENE GLYCOL MONOBUTYL ETHER:

■ Fish LC50 (96hr.) (mg/l):	1490
■ BCF<100:	0.4
■ log Kow (Prager 1995):	0.83
■ log Kow (Sangster 1997):	0.8
■ Half- life Soil - High (hours):	672
■ Half- life Soil - Low (hours):	168
■ Half- life Air - High (hours):	32.8
■ Half- life Air - Low (hours):	3.28
■ Half- life Surface water - High (hours):	672
■ Half- life Surface water - Low (hours):	168
■ Half- life Ground water - High (hours):	1344
■ Half- life Ground water - Low (hours):	336
■ Aqueous biodegradation - Aerobic - High (hours):	672
■ Aqueous biodegradation - Aerobic - Low (hours):	168
■ Aqueous biodegradation - Anaerobic - High (hours):	2688
■ Aqueous biodegradation - Anaerobic - Low (hours):	672
■ Photooxidation half- life air - High (hours):	32.8
■ Photooxidation half- life air - Low (hours):	3.28
■ Fish LC50 (96hr.) (mg/l):	1250- 1650
■ Daphnia magna EC50 (48hr.) (mg/l):	600- 1000

■ For glycol ethers:
Environmental fate:
Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. 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

continued...

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.
log Kow: 0.76-0.83
Koc: 67
Half-life (hr) air: 17
Henry's atm m^3 /mol: 2.08E-08
BOD 5 if unstated: 0.71
COD: 2.2
Log BCF: 0.4
Fish LC50 (24 h): 983-1650 mg/L
Fish LC50 (96 h): fathead minnow 1700 mg/L **
Invertebrate toxicity:
cell mult. inhib.91-900mg/L
(Daphnia) 48h LC50: >1000 mg/L **
Bioaccumulation: not sig
Effects on algae and plankton: cell mult. inhib.35-900mg/L
Degradation Biological: rapid
processes Abiotic: no hydrol&photol,RxnOH* ** [Union Carbide]

ETHYLBENZENE:

■ Hazardous Air Pollutant:	Yes
■ Fish LC50 (96hr.) (mg/l):	32.0- 97.1
■ Algae IC50 (72hr.) (mg/l):	33- 160
■ Water solubility (g/l):	2.16
■ log Kow (Prager 1995):	3.15
■ log Kow (Sangster 1997):	3.15
■ log Pow (Verschueren 1983):	3.15
■ ThOD:	3.17
■ Half- life Soil - High (hours):	240
■ Half- life Soil - Low (hours):	72
■ Half- life Air - High (hours):	85.6
■ Half- life Air - Low (hours):	8.56
■ Half- life Surface water - High (hours):	240
■ Half- life Surface water - Low (hours):	72
■ Half- life Ground water - High (hours):	5472
■ Half- life Ground water - Low (hours):	144
■ Aqueous biodegradation - Aerobic - High (hours):	240
■ Aqueous biodegradation - Aerobic - Low (hours):	72
■ Aqueous biodegradation - Anaerobic - High (hours):	5472
■ Aqueous biodegradation - Anaerobic - Low (hours):	4224
■ Aqueous biodegradation - Removal secondary treatment - High (hours):	95%
■ Aqueous biodegradation - Removal secondary treatment - Low (hours):	72%
■ Photolysis maximum light absorption - High (nano- m):	269.5
■ Photolysis maximum light absorption - Low (nano- m):	208
■ Photooxidation half- life air - High (hours):	85.6
■ Photooxidation half- life air - Low (hours):	8.56

■ The material is classified as an ecotoxin* because the Fish LC50 (96 hours) is less than or equal to 0.1

continued...

mg/l
* Classification of Substances as Ecotoxic (Dangerous to the Environment)
Appendix 8, Table 1
Compiler's Guide for the Preparation of International Chemical Safety Cards: 1993 Commission of the European Communities.

HYDROCARBON PROPELLANT:
■ For hydrocarbons:
Environmental fate:
The lower molecular weight hydrocarbons are expected to form a "slick" on the surface of waters after release in calm sea conditions. This is expected to evaporate and enter the atmosphere where it will be degraded through reaction with hydroxy radicals.
Some hydrocarbon will become associated with benthic sediments, and it is likely to be spread over a fairly wide area of sea floor. Marine sediments may be either aerobic or anaerobic. The material, in probability, is biodegradable, under aerobic conditions (isomerised olefins and alkenes show variable results). Evidence also suggests that the hydrocarbons may be degradable under anaerobic conditions although such degradation in benthic sediments may be a relatively slow process.
Under aerobic conditions hydrocarbons degrade to water and carbon dioxide, while under anaerobic processes they produce water, methane and carbon dioxide.
Alkenes have low log octanol/water partition coefficients (Kow) of about 1 and estimated bioconcentration factors (BCF) of about 10; aromatics have intermediate values (log Kow values of 2-3 and BCF values of 20-200), while C5 and greater alkanes have fairly high values (log Kow values of about 3-4.5 and BCF values of 100-1,500
The estimated volatilisation half-lives for alkanes and benzene, toluene, ethylbenzene, xylene (BTEX) components were predicted as 7 days in ponds, 1.5 days in rivers, and 6 days in lakes. The volatilisation rate of naphthalene and its substituted derivatives were estimated to be slower.
Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds. Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media.
The final products of microbial degradation are carbon dioxide, water, and microbial biomass. The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics, and the aromatics in the C10-C22 range are the most readily biodegradable; n-alkanes, n-alkyl aromatics, and aromatics in the C5-C9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilisation and thus are unavailable in most environments; n-alkanes in the C1-C4 ranges are biodegradable only by a narrow range of specialised hydrocarbon degraders; and n-alkanes, n-alkyl aromatics, and aromatics above C22 are generally not available to degrading microorganisms.
Hydrocarbons with condensed ring structures, such as PAHs with four or more rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is, greater than 7.
All biological transformations are affected by temperature. Generally, as the temperature increases, biological activity tends to increase up to a temperature where enzyme denaturation occurs.
Atmospheric fate: Alkanes, isoalkanes, and cycloalkanes have half-lives on the order of 1-10 days, whereas alkenes, cycloalkenes, and substituted benzenes have half-lives of 1 day or less. Photochemical oxidation products include aldehydes, hydroxy compounds, nitro compounds, and peroxyacyl nitrates. Alkenes, certain substituted aromatics, and naphthalene are potentially susceptible to direct photolysis.
Ecotoxicity:
Hydrocarbons are hydrophobic (high log Kow and low water solubility). Such substances produce toxicity in aquatic organisms by a mechanism referred to as "non-polar narcosis" or "baseline" toxicity. The hydrophobicity increases and water solubility decreases with increasing carbon number for a particular class of hydrocarbon. Substances with the same carbon number show increased hydrophobicity and decreased solubility

continued...

with increasing saturation. Quantitative structure activity relationships (QSAR), relating both solubility and toxicity to Kow predict that the water solubility of single chemical substances decreases more rapidly with increasing Kow than does the acute toxicity.
Based on test results, as well as theoretical considerations, the potential for bioaccumulation may be high.
Toxic effects are often observed in species such as blue mussel, daphnia, freshwater green algae, marine copepods and amphipods.
The values of log Kow for individual hydrocarbons increase with increasing carbon number within homologous series of generic types. Quantitative structure activity relationships (QSAR), relating log Kow values of single hydrocarbons to toxicity, show that water solubility decreases more rapidly with increasing Kow than does the concentration causing effects. This relationship varies somewhat with species of hydrocarbon, but it follows that there is a log Kow limit for hydrocarbons, above which, they will not exhibit acute toxicity; this limit is at a log Kow value of about 4 to 5. It has been confirmed experimentally that for fish and invertebrates, paraffinic hydrocarbons with a carbon number of 10 or higher (log Kow >5) show no acute toxicity and that alkylbenzenes with a carbon number of 14 or greater (log Kow >5) similarly show no acute toxicity.
QSAR equations for chronic toxicity also suggest that there should be a point where hydrocarbons with high log Kow values become so insoluble in water that they will not cause chronic toxicity, that is, that there is also a solubility cut-off for chronic toxicity. Thus, paraffinic hydrocarbons with carbon numbers of greater than 14 (log Kow >7.3) should show no measurable chronic toxicity. Experimental support for this cut-off is demonstrated by chronic toxicity studies on lubricant base oils and one “heavy” solvent grade (substances composed of paraffins of C20 and greater) which show no effects after exposures to concentrations well above solubility.
The initial criteria for classification of substances as dangerous to the aquatic environment are based upon acute toxicity data in fish, daphnids and algae. However, for substances that have low solubility and show no acute toxicity, the possibility of a long-term or chronic hazard to the environment is recognised in the R53 phrase or so-called "safety net". The R53 assignment for possible long-term harm is a surrogate for chronic toxicity test results and is triggered by substances that are both bioaccumulative and persistent. The indicators of bioaccumulation and persistence are taken as a BCF > 100 (or log Kow > 3 if no BCF data) and lack of ready biodegradability. For low solubility substances which have direct chronic toxicity data demonstrating no chronic toxicity at 1 mg/L or higher, these data take precedence such that no classification for long term toxicity is required.
■ Drinking Water Standards: hydrocarbon total: 10 ug/l (UK max.).

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
acetone	LOW	HIGH	LOW	HIGH
toluene	LOW	MED	LOW	MED
n- butyl acetate	LOW		LOW	HIGH
xylene	LOW	LOW	LOW	
ethylene glycol monobutyl ether	LOW	LOW	LOW	HIGH
ethylbenzene	LOW	MED	LOW	MED

GESAMP/EHS COMPOSITE LIST - GESAMP Hazard Profiles

Name / Cas No / RTECS No	EHS	TRN	A1a	A1b	A1	A2	B1	B2	C1	C2	C3	D1	D2	D3	E1	E2	E3
Xylene (mixed isomers) / ZE2275000	1408	743	3	NI	3	NR	3	0	0	0	0	2	2		(T)	FE	2

continued...

Legend:
EHS=EHS Number (EHS=GESAMP Working Group on the Evaluation of the Hazards of Harmful Substances Carried by Ships) NRT=Net Register Tonnage, A1a=Bioaccumulation log Pow, A1b=Bioaccumulation BCF, A1=Bioaccumulation, A2=Biodegradation, B1=Acuteaquatic toxicity LC/ECIC50 (mg/l), B2=Chronic aquatic toxicity NOEC (mg/l), C1=Acute mammalian oral toxicity LD50 (mg/kg), C2=Acutemammalian dermal toxicity LD50 (mg/kg), C3=Acute mammalian inhalation toxicity LC50 (mg/kg), D1=Skin irritation & corrosion, D2=Eye irritation& corrosion, D3=Long-term health effects, E1=Tainting, E2=Physical effects on wildlife & benthic habitats, E3=Interference with coastal amenities,
For column A2: R=Readily biodegradable, NR=Not readily biodegradable.
For column D3: C=Carcinogen, M=Mutagenic, R=Reprotoxic, S=Sensitising, A=Aspiration hazard, T=Target organ systemic toxicity, L=Lunginjury, N=Neurotoxic, I=Immunotoxic.
For column E1: NT=Not tainting (tested), T=Tainting test positive.
For column E2: Fp=Persistent floater, F=Floater, S=Sinking substances.
The numerical scales start from 0 (no hazard), while higher numbers reflect increasing hazard.
(GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships)

Section 13 - DISPOSAL CONSIDERATIONS

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

Section 14 - TRANSPORTATION INFORMATION

Labels Required: FLAMMABLE GAS

HAZCHEM:
2YE (ADG7)

Land Transport UNDG:

Class or division:	2	Subsidiary risk:	None
UN No.:	1950	UN packing group:	None

Shipping Name:AEROSOLS

continued...

Air Transport IATA:

ICAO/IATA Class:	2.1	ICAO/IATA Subrisk:	None
UN/ID Number:	1950	Packing Group:	-
Special provisions:	A145		
Cargo Only			
Packing Instructions:	203	Maximum Qty/Pack:	150 kg
Passenger and Cargo		Passenger and Cargo	
Packing Instructions:	203	Maximum Qty/Pack:	75 kg
Passenger and Cargo Limited Quantity		Passenger and Cargo Limited Quantity	
Packing Instructions:	Y203	Maximum Qty/Pack:	30 kg G

Shipping Name: AEROSOLS, FLAMMABLE

Maritime Transport IMDG:

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

Shipping Name: AEROSOLS

GESAMP hazard profiles for this material can be found in section 12 of the MSDS.

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE S5

REGULATIONS

Regulations for ingredients

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

"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 Drugs and Poisons (SUSDP) - Appendix E (Part 2)","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 5","GESAMP/EHS Composite List - GESAMP Hazard Profiles","IMO IBC Code Chapter 18: List of products to which the Code does not apply","IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances","International Fragrance Association (IFRA) Survey: Transparency List","OECD Representative List of High Production Volume (HPV) Chemicals","United Nations List of Precursors and Chemicals Frequently used in the Illicit Manufacture of Narcotic Drugs and Psychotropic Substances Under International Control - Table II"

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

"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 Ecosystem maintenance - Organic chemicals - Non-pesticide anthropogenic organics","Australia - Australian Capital Territory Environment Protection Regulation Pollutants entering waterways - Domestic water quality","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

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Drugs and Poisons (SUSDP) - Appendix E (Part 2)","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix I","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 6","GESAMP/EHS Composite List - GESAMP Hazard Profiles","IMO IBC Code Chapter 17: Summary of minimum requirements","IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs","OECD Representative List of High Production Volume (HPV) Chemicals","United Nations List of Precursors and Chemicals Frequently used in the Illicit Manufacture of Narcotic Drugs and Psychotropic Substances Under International Control - Table II","WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water"

n-butyl acetate (CAS: 123-86-4) 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)","IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk","International Council of Chemical Associations (ICCA) - High Production Volume List","OECD Representative List of High Production Volume (HPV) Chemicals"

white spirit (CAS: 8052-41-3,8042-47-5) is found on the following regulatory lists;
"Australia Exposure Standards","Australia Hazardous Substances","Australia Inventory of Chemical Substances (AICS)","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)", "GESAMP/EHS Composite List - GESAMP Hazard Profiles","IMO IBC Code Chapter 17: Summary of minimum requirements","IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO","International Council of Chemical Associations (ICCA) - High Production Volume List","OECD Representative List of High Production Volume (HPV) Chemicals"

xylene (CAS: 1330-20-7) is found on the following regulatory lists;
"Australia High Volume Industrial Chemical List (HVICL)","Australia Inventory of Chemical Substances (AICS)", "International Council of Chemical Associations (ICCA) - High Production Volume List","OECD Representative List of High Production Volume (HPV) Chemicals"

ethylene glycol monobutyl ether (CAS: 111-76-2) 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)","IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances","International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs","International Fragrance Association (IFRA) Survey: Transparency List","OECD Representative List of High Production Volume (HPV) Chemicals"

ethylbenzene (CAS: 100-41-4) is found on the following regulatory lists;
"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 Ecosystem maintenance - Organic chemicals - Non-pesticide anthropogenic organics","Australia - Australian Capital Territory Environment Protection Regulation Pollutants entering waterways - Domestic water quality","Australia Exposure Standards","Australia Hazardous Substances","Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)","Australia National Pollutant Inventory","GESAMP/EHS Composite List - GESAMP Hazard Profiles","IMO IBC Code Chapter 17: Summary of minimum requirements","IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk","IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO","International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs","OECD Representative List of High Production Volume (HPV) Chemicals","WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water"

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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)","OECD Representative List of High Production Volume (HPV) Chemicals"

No data for Dy-Mark Rust Reformer Aerosol (CW: 4649-98)

Section 16 - OTHER INFORMATION

INGREDIENTS WITH MULTIPLE CAS NUMBERS

Ingredient Name	CAS	
white spirit	8052-41-3,	8042-47-5
hydrocarbon propellant	68476-85-7,	68476-86-8

REPRODUCTIVE HEALTH GUIDELINES

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
toluene	9.6 mg/m3	10	D	NA	-
xylene	1.5 mg/m3	10	D	NA	-
ethylene glycol monobutyl ether	3.6 mg/m3	100	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.