



# All Purpose Thinners (Toluene Formulation)

## Recochem Inc.

Chemwatch Hazard Alert Code: 4

Part Number: 17000

Version No: 1.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 23/08/2022

Print Date: 23/08/2022

L.GHS.AUS.EN

### SECTION 1 Identification of the substance / mixture and of the company / undertaking

#### Product Identifier

**Product name:** All Purpose Thinners (Toluene Formulation)

**Synonyms:**

Not Available

**Proper shipping name:** FLAMMABLE LIQUID, N.O.S. Flammable Liquid N.O.S. (65% Liquid Hydrocarbons)

**Other means of identification:** Not Available

#### Relevant identified uses of the substance or mixture and uses advised against

**Relevant identified uses:**

Paint thinner and cleaner for spray lines and spray guns

#### Details of the supplier of the safety data sheet

Registered company name	Recochem Inc.
Address	1809 Lytton Road QLD 4178 Australia
Telephone	+61 7 3308 5200
Fax	+61 7 3308 5201
Website	<a href="http://www.recochem.com.au">www.recochem.com.au</a>
Email	recoast@recochem.com

#### Emergency telephone number

Association / Organisation	Poisons Information
Emergency telephone numbers	Australia: 13 11 26
Other emergency telephone numbers	New Zealand: 0800 764 766

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

Poisons Schedule	5
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Specific Target Organ Toxicity - Repeated Exposure Category 2, Flammable Liquids Category 2, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Reproductive Toxicity Category 2, Aspiration Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

#### Label elements

##### Hazard pictogram(s)



Signal word: **Danger**

##### Hazard statement(s)

H319: Causes serious eye irritation.

H336: May cause drowsiness or dizziness.

H373: May cause damage to organs through prolonged or repeated exposure. (Nervous system, Respiratory system) (Inhalation)

H225: Highly flammable liquid and vapour.

H302: Harmful if swallowed.

H315: Causes skin irritation.

H361fd: Suspected of damaging fertility. Suspected of damaging the unborn child.

H304: May be fatal if swallowed and enters airways.

H412: Harmful to aquatic life with long lasting effects.

##### Precautionary statement(s) Prevention

P201: Obtain special instructions before use.

P210: Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.

P260: Do not breathe mist/vapours/spray.

P271: Use only a well-ventilated area.

P280: Wear protective gloves, protective clothing, eye protection and face protection.

P240: Ground and bond container and receiving equipment.

P241: Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.

- P242:** Use non-sparking tools.  
**P243:** Take action to prevent static discharges.  
**P264:** Wash all exposed external body areas thoroughly after handling.  
**P270:** Do not eat, drink or smoke when using this product.  
**P273:** Avoid release to the environment.

#### Precautionary statement(s) Response

- P301+P310:** IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.  
**P331:** Do NOT induce vomiting.  
**P308+P313:** IF exposed or concerned: Get medical advice/ attention.  
**P370+P378:** In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.  
**P305+P351+P338:** IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.  
**P337+P313:** If eye irritation persists: Get medical advice/attention.  
**P301+P312:** IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.  
**P302+P352:** IF ON SKIN: Wash with plenty of water.  
**P303+P361+P353:** IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].  
**P304+P340:** IF INHALED: Remove person to fresh air and keep comfortable for breathing.  
**P330:** Rinse mouth.  
**P332+P313:** If skin irritation occurs: Get medical advice/attention.  
**P362+P364:** Take off contaminated clothing and wash it before reuse.

#### Precautionary statement(s) Storage

- P403+P235:** Store in a well-ventilated place. Keep cool.  
**P405:** Store locked up.

#### Precautionary statement(s) Disposal

- P501:** Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

Not Applicable

### SECTION 3 Composition / information on ingredients

#### Substances

See section below for composition of Mixtures

#### Mixtures

CAS No	%[weight]	Name
108-88-3	30-40	<u>toluene</u>
64742-89-8.	30	<u>solvent naphtha petroleum, light aliphatic</u>
67-64-1	20-30	<u>acetone</u>
64-17-5	<10	<u>ethanol</u>
111-76-2	<5	<u>ethylene glycol monobutyl ether</u>

**Legend:** 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L;  
 \* EU IOELVs available

### SECTION 4 First aid measures

#### Description of first aid measures

##### Eye Contact

- If this product comes in contact with the eyes:
- Wash out immediately 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.
  - Seek medical attention without delay; if pain persists or recurs seek medical attention.
  - Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

##### Skin Contact

- If skin contact occurs:
- Immediately remove all contaminated clothing, including footwear.
  - Flush skin and hair with running water (and soap if available).
  - Seek medical attention in event of irritation.

##### Inhalation

- If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested.
- Prosthesis such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- Transport to hospital, or doctor, without delay.

##### Ingestion

- If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.
- **If swallowed do NOT induce vomiting.**
- If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- Observe the patient carefully.
- Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.

## All Purpose Thinners (Toluene Formulation)

- Seek medical advice.
- Avoid giving milk or oils.
- Avoid giving alcohol.

### Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

For petroleum distillates

- In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
- Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
- Positive pressure ventilation may be necessary.
- Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
- After the initial episode, individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment. Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
- Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
- Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur. Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.

BP America Product Safety & Toxicology Department

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 simple ketones:

### BASIC TREATMENT

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

### ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Consider intubation at first sign of upper airway obstruction resulting from oedema.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

### EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Consult a toxicologist as necessary.

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

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

Following acute or short term repeated exposures to toluene:

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

### BIOLOGICAL EXPOSURE INDEX - BEI

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

Continued...

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

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

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

## SECTION 5 Firefighting measures

### Extinguishing media

#### Special hazards arising from the substrate or mixture

##### Fire Incompatibility

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

#### Advice for firefighters

##### Fire Fighting

##### Fire/Explosion Hazard

- Liquid and vapour are highly flammable.
- Severe fire hazard when exposed to heat, flame and/or oxidisers.
- Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO).

Combustion products include:

carbon dioxide (CO<sub>2</sub>)

other pyrolysis products typical of burning organic material.

**Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions.

#### HAZCHEM

•3YE

## SECTION 6 Accidental release measures

### Personal precautions, protective equipment and emergency procedures

See section 8

### Environmental precautions

See section 12

### Methods and material for containment and cleaning up

#### Minor Spills

- Remove all ignition sources.
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact with the substance, by using protective equipment.
- Contain and absorb small quantities with vermiculite or other absorbent material.
- Wipe up.
- Collect residues in a flammable waste container.

#### Major Spills

- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear full body protective clothing with breathing apparatus.
- Prevent, by all means available, spillage from entering drains or water courses.
- Consider evacuation (or protect in place).
- 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.
- Contain or absorb spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- If contamination of drains or waterways occurs, advise emergency services.

Chemical Class: aromatic hydrocarbons

For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
--------------	------	-------------	------------	-------------

#### LAND SPILL - SMALL

Feathers - pillow	1	throw	pitchfork	DGC, RT
cross-linked polymer - particulate	2	shovel	shovel	R,W,SS
cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	3	shovel	shovel	R, I, P,
treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I

## All Purpose Thinners (Toluene Formulation)

wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT
---------------------	---	-------	-----------	---------------

### LAND SPILL - MEDIUM

cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
feathers - pillow	3	throw	skiploader	DGC, RT
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC

#### Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT: Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

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

## SECTION 7 Handling and storage

### Precautions for safe handling

#### Safe handling

- Containers, even those that have been emptied, may contain explosive vapours.
- Do NOT cut, drill, grind, weld or perform similar operations on or near containers.

#### Contains low boiling substance:

Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.

- Check for bulging containers.
- Vent periodically
- Always release caps or seals slowly to ensure slow dissipation of vapours
- Electrostatic discharge may be generated during pumping - this may result in fire.
- Ensure electrical continuity by bonding and grounding (earthing) all equipment.
- Restrict line velocity during pumping in order to avoid generation of electrostatic discharge ( $\leq 1$  m/sec until fill pipe submerged to twice its diameter, then  $\leq 7$  m/sec).
- Avoid splash filling.
- Do NOT use compressed air for filling discharging or handling operations.
- 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, heat or ignition sources.
- When handling, **DO NOT eat, drink or smoke.**
- Vapour may ignite on pumping or pouring due to static electricity.
- **DO NOT use plastic buckets.**
- Earth and secure metal containers when dispensing or pouring product.
- Use spark-free tools when handling.
- Avoid contact with incompatible materials.
- Keep containers securely sealed.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- Use good occupational work practice.
- Observe manufacturer's storage and handling recommendations contained within this SDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.

#### Other information

- Store in original containers in approved flame-proof area.
- No smoking, naked lights, heat or ignition sources.
- **DO NOT store in pits, depressions, basements or areas where vapours may be trapped.**
- Keep containers securely sealed.
- Store away from incompatible materials in a cool, dry well ventilated area.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storage and handling recommendations contained within this SDS.

### Conditions for safe storage, including any incompatibilities

#### Suitable container

- Packing as supplied by manufacturer.
- Plastic containers may only be used if approved for flammable liquid.
- Check that containers are clearly labelled and free from leaks.
- For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure.
- For materials with a viscosity of at least 2680 cSt. (23 deg. C)
- For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)
- Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used.
- Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages
- In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.

**Storage incompatibility**

## Toluene:

- reacts violently with strong oxidisers, bromine, bromine trifluoride, chlorine, hydrochloric acid/ sulfuric acid mixture, 1,3-dichloro-5,5-dimethyl-2,4-imidazolidindione, dinitrogen tetraoxide, fluorine, concentrated nitric acid, nitrogen dioxide, silver chloride, sulfur dichloride, uranium fluoride, vinyl acetate
- forms explosive mixtures with strong acids, strong oxidisers, silver perchlorate, tetranitromethane
- is incompatible with bis-toluenediazo oxide
- attacks some plastics, rubber and coatings
- may generate electrostatic charges, due to low conductivity, on flow or agitation.
- Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates.

## For alkyl aromatics:

The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.

- Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen
- Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.
- Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.
- Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.
- Alkali metals accelerate the oxidation while CO<sub>2</sub> as co-oxidant enhances the selectivity.
- Microwave conditions give improved yields of the oxidation products.
- Photo-oxidation products may occur following reaction with hydroxyl radicals and NO<sub>x</sub> - these may be components of photochemical smogs.

## Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007

- Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents.
- Aromatics can react exothermically with bases and with diazo compounds.

## Acetone:

- may react violently with chloroform, activated charcoal, aliphatic amines, bromine, bromine trifluoride, chlorotriazine, chromic(IV) acid, chromic(VI) acid, chromium trioxide, chromyl chloride, hexachloromelamine, iodine heptafluoride, iodoform, liquid oxygen, nitrosyl chloride, nitrosyl perchlorate, nitryl perchlorate, perchloromelamine, peroxomonosulfuric acid, platinum, potassium tert-butoxide, strong acids, sulfur dichloride, trichloromelamine, xenon tetrafluoride
- reacts violently with bromoform and chloroform in the presence of alkalis or in contact with alkaline surfaces.
- may form unstable and explosive peroxides in contact with strong oxidisers, fluorine, hydrogen peroxide (90%), sodium perchlorate, 2-methyl-1,3-butadiene
- can increase the explosive sensitivity of nitromethane on contact flow or agitation may generate electrostatic charges due to low conductivity
- dissolves or attacks most rubber, resins, and plastics (polyethylenes, polyester, vinyl ester, PVC, Neoprene, Viton)

## Ketones in this group:

- are reactive with many acids and bases liberating heat and flammable gases (e.g., H<sub>2</sub>).
- react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H<sub>2</sub>) and heat.
- are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides.
- react violently with aldehydes, HNO<sub>3</sub> (nitric acid), HNO<sub>3</sub> + H<sub>2</sub>O<sub>2</sub> (mixture of nitric acid and hydrogen peroxide), and HClO<sub>4</sub> (perchloric acid).
- may react with hydrogen peroxide to form unstable peroxides; many are heat- and shock-sensitive explosives.

A significant property of most ketones is that the hydrogen atoms on the carbons next to the carbonyl group are relatively acidic when compared to hydrogen atoms in typical hydrocarbons. Under strongly basic conditions these hydrogen atoms may be abstracted to form an enolate anion. This property allows ketones, especially methyl ketones, to participate in condensation reactions with other ketones and aldehydes. This type of condensation reaction is favoured by high substrate concentrations and high pH (greater than 1 wt% NaOH).

**SECTION 8 Exposure controls / personal protection****Control parameters****Occupational Exposure Limits (OEL)****INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	toluene	Toluene	50 ppm / 191 mg/m <sup>3</sup>	574 mg/m <sup>3</sup> / 150 ppm	Not Available	Not Available
Australia Exposure Standards	solvent naphtha petroleum, light aliphatic	Oil mist, refined mineral	5 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	acetone	Acetone	500 ppm / 1185 mg/m <sup>3</sup>	2375 mg/m <sup>3</sup> / 1000 ppm	Not Available	Not Available
Australia Exposure Standards	ethanol	Ethyl alcohol	1000 ppm / 1880 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m <sup>3</sup>	242 mg/m <sup>3</sup> / 50 ppm	Not Available	Not Available

**MATERIAL DATA**

## IFRA Prohibited Fragrance Substance

The International Fragrance Association (IFRA) Standards form the basis for the globally accepted and recognized risk management system for the safe use of fragrance ingredients and are part of the IFRA Code of Practice. This is the self-regulating system of the industry, based on risk assessments carried out by an independent Expert Panel

## For ethanol:

Odour Threshold Value: 49-716 ppm (detection), 101 ppm (recognition)

Eye and respiratory tract irritation do not appear to occur at exposure levels of less than 5000 ppm and the TLV-TWA is thought to provide an adequate margin of safety against such effects. Experiments in man show that inhalation of 1000 ppm caused slight symptoms of poisoning and 5000 ppm caused strong stupor and morbid sleepiness. Subjects exposed to 5000 ppm to 10000 ppm experienced smarting of the eyes and nose and coughing. Symptoms disappeared within minutes. Inhalation also causes local irritating effects to the eyes and upper respiratory tract, headaches, sensation of heat intraocular tension, stupor, fatigue and a need to sleep. At 15000 ppm there was continuous lachrymation and coughing.

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)

## All Purpose Thinners (Toluene Formulation)

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:

ClassOSF 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  |

NOTE E: Substances with specific effects on human health that are classified as carcinogenic, mutagenic and/ or toxic for reproduction in categories 1 or 2 are ascribed Note E if they are classified as very toxic (T+), toxic (T) or harmful (Xn). For these substances the risk phrases R20, R21, R22, R23, R24, R25, R26, R27, R28, R39, R68, R48 and R65 and all combinations of these risk phrases shall be preceded by the word "Also".

R45-23: May cause cancer. Also toxic by inhalation

This note applies only to certain complex oil-derived substances in Annex VI.

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

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)

for benzene

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 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.

for: hexane, isomers (excluding n-hexane)

The TLV-TWA is thought to be protective against nausea, headache, upper respiratory tract irritation and CNS depression. The STEL is added to prevent objective depression of the CNS. The lower value ascribed

to n-hexane is due to the neurotoxicity of its metabolites, principally 5-hydroxy-2-hexanone and 2,5-hexanedione. It is considered unlikely that other hexanes follow the same metabolic route. It should be noted however that the n-hexane TLV-TWA also applies to commercial hexane having a concentration of greater than 5% n-hexane.

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)

For ethylene glycol monobutyl ether (2-butoxyethanol)

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)

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

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

### Exposure controls

#### Appropriate engineering controls

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

The basic types of engineering controls are:

Continued...

## All Purpose Thinners (Toluene Formulation)

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

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

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

- Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
- Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.
- Open-vessel systems are prohibited.
- Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.
- Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.
- For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).
- Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
- Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.

### Personal protection



### Eye and face protection

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

### Skin protection

See Hand protection below

### Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

### Body protection

See Other protection below

### Other protection

- Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent]
- Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent]
- Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely.
- Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood.
- Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Overalls.
- PVC Apron.
- PVC protective suit may be required if exposure severe.
- Eyewash unit.
- Ensure there is ready access to a safety shower.
- Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.
- For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).
- Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot and shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

#### Appearance

:

Colourless liquid

#### Physical state

Liquid

#### Relative density (Water = 1)

0.78 - 0.82

#### Odour

Not Available

#### Partition coefficient n-octanol / water

Not Available

## All Purpose Thinners (Toluene Formulation)

<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	480 - 536
<b>pH (as supplied)</b>	Not Available	<b>Decomposition temperature (°C)</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	50 - 166	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	-30	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	HIGHLY FLAMMABLE.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	19.0	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	0.8	<b>Volatile Component (%vol)</b>	100
<b>Vapour pressure (kPa)</b>	30	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Partly miscible	<b>pH as a solution (Not Available%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	>1	<b>VOC g/L</b>	800

### SECTION 10 Stability and reactivity

#### Reactivity:

See section 7

#### Chemical stability :

- Unstable in the presence of incompatible materials.
- Product is considered stable.
- Hazardous polymerisation will not occur.

#### Possibility of hazardous reactions :

See section 7

#### Conditions to avoid :

See section 7

#### Incompatible materials :

See section 7

#### Hazardous decomposition products :

See section 5

### SECTION 11 Toxicological information

#### Information on toxicological effects

##### Inhaled

Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.

Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.

Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.

The most common signs of inhalation overexposure to ethanol, in animals, include ataxia, incoordination and drowsiness for those surviving narcosis. The narcotic dose for rats, after 2 hours of exposure, is 19260 ppm.

Inhalation hazard is increased at higher temperatures.

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

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

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

The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.

Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.

The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation.

## All Purpose Thinners (Toluene Formulation)

### Ingestion

Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.

Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).

The material is not thought to produce adverse health effects following ingestion (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum.

Ingestion of ethanol (ethyl alcohol, "alcohol") may produce nausea, vomiting, bleeding from the digestive tract, abdominal pain, and diarrhoea. Effects on the body:

Blood concentration	Effects
<1.5 g/L	Mild: impaired vision, co-ordination and reaction time; emotional instability
1.5-3.0 g/L	Moderate: Slurred speech, confusion, inco-ordination, emotional instability, disturbances in perception and senses, possible blackouts, and impaired objective performance in standardized tests. Possible double vision, flushing, fast heart rate, sweating and incontinence. Slow breathing may occur rarely and fast breathing may develop in cases of metabolic acidosis, low blood sugar and low blood potassium. Central nervous system depression may progress to coma.
3-5 g/L	Severe: cold clammy skin, low body temperature and low blood pressure. Atrial fibrillation and heart block have been reported. Depression of breathing may occur, respiratory failure may follow serious poisoning, choking on vomit may result in lung inflammation and swelling. Convulsions due to severe low blood sugar may also occur. Acute liver inflammation may develop.

At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes)

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

### Skin Contact

The material may accentuate any pre-existing dermatitis condition

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

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

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

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

- produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or
- produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.

Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

### Eye

Direct contact of the eye with ethanol may cause immediate stinging and burning with reflex closure of the lid and tearing, transient injury of the corneal epithelium and hyperaemia of the conjunctiva. Foreign-body type discomfort may persist for up to 2 days but healing is usually spontaneous and complete.

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.

Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.

### Chronic

Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.

Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.

Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents.

Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size.

Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1).

(1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996

## All Purpose Thinners (Toluene Formulation)

Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

### Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar

naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of an alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.

Chronic toluene habituation occurs following intentional abuse (glue sniffing) or from occupational exposure. Ataxia, incoordination and tremors of the hands and feet (as a consequence of diffuse cerebral atrophy), headache, abnormal speech, transient memory loss, convulsions, coma, drowsiness, reduced colour perception, frank blindness, nystagmus (rapid, involuntary eye-movements), hearing loss leading to deafness and mild dementia have all been associated with chronic abuse. Peripheral nerve damage, encephalopathy, giant axonopathy electrolyte disturbances in the cerebrospinal fluid and abnormal computer tomographic (CT scans) are common amongst toluene addicts. Although toluene abuse has been linked with kidney disease, this does not commonly appear in cases of occupational toluene exposures. Cardiac and haematological toxicity are however associated with chronic toluene exposures. Cardiac arrhythmia, multifocal and premature ventricular contractions and supraventricular tachycardia are present in 20% of patients who abused toluene-containing paints. Previous suggestions that chronic toluene inhalation produced human peripheral neuropathy have been discounted. However central nervous system (CNS) depression is well documented where blood toluene exceeds 2.2 mg%. Toluene abusers can achieve transient circulating concentrations of 6.5 %. Amongst workers exposed for a median time of 29 years, to toluene, no subacute effects on neuroathenic complaints and psychometric test results could be established.

The prenatal toxicity of very high toluene concentrations has been documented for several animal species and man. Malformations indicative of specific teratogenicity have not generally been found. Neonatal toxicity, described in the literature, takes the form of embryo death or delayed foetal growth and delayed skeletal system development. Permanent damage of children has been seen only when mothers have suffered from chronic intoxication as a result of "sniffing".

Chronic exposure to benzene may cause headache, fatigue, loss of appetite and lassitude with incipient blood effects including anaemia and blood changes. Benzene is a myelotoxicant known to suppress bone- marrow cell proliferation and to induce haematologic disorders in humans and animals. Signs of benzene-induced aplastic anaemia include suppression of leukocytes (leukopenia), red cells (anaemia), platelets (thrombocytopenia) or all three cell types (pancytopenia). Classic symptoms include weakness, purpura, and haemorrhage. The most significant toxic effect is insidious and often reversible injury to the blood forming tissue. Leukaemia may develop. Occupational exposures have shown a relationship between exposure to benzene and production of myelogenous leukaemia. There may also be a relationship between benzene exposure and the production of lymphoma and multiple myeloma. In chronic exposure, workers exhibit signs of central nervous system lesions and impairment of hearing.

Benzene haemotoxicity and leukaemogenicity involve metabolism, growth factor regulation, oxidative stress, DNA damage, cell regulation, and apoptosis. (Yoon et al Environmental Health Perspectives, 111, pp 1411-1420, 2003)

All Purpose Thinners (Toluene Formulation)	TOXICITY	IRRITATION
	Not Available	Not Available

  

toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg <sup>[2]</sup>	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation(Rat) LC50; >13350 ppm4h <sup>[2]</sup>	Eye (rabbit):0.87 mg - mild
	Oral (Rat) LD50: 636 mg/kg <sup>[2]</sup>	Eye (rabbit):100 mg/30sec - mild
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>	

  

solvent naphtha petroleum, light aliphatic	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation(Rat) LC50; >4.42 mg/L4h <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50; >4500 mg/kg <sup>[1]</sup>	

  

acetone	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 20000 mg/kg <sup>[2]</sup>	Eye (human): 500 ppm - irritant
	Inhalation(Mouse) LC50; 44 mg/L4h <sup>[2]</sup>	Eye (rabbit): 20mg/24hr -moderate
	Oral (Rat) LD50; 5800 mg/kg <sup>[2]</sup>	Eye (rabbit): 3.95 mg - SEVERE
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 500 mg/24hr - mild
		Skin (rabbit):395mg (open) - mild
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>

## All Purpose Thinners (Toluene Formulation)

	TOXICITY	IRRITATION
ethanol	Dermal (rabbit) LD50: 17100 mg/kg <sup>[1]</sup>	Eye (rabbit): 500 mg SEVERE
	Inhalation(Rat) LC50: 64000 ppm4h <sup>[2]</sup>	Eye (rabbit):100mg/24hr-moderate
	Oral (Rat) LD50: 7060 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit):20 mg/24hr-moderate
		Skin (rabbit):400 mg (open)-mild
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
ethylene glycol monobutyl ether	dermal (guinea pig) LD50: 210 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg SEVERE
	Inhalation(Rat) LC50: 2.21 mg/14h <sup>[2]</sup>	Eye (rabbit): 100 mg/24h-moderate
	Oral (Rat) LD50: 300 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 500 mg, open; mild
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>

**Legend:** 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.\* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

## All Purpose Thinners (Toluene Formulation)

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

Data demonstrate that during inhalation exposure, aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure.

Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism.

The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.

## SOLVENT NAPHTHA PETROLEUM, LIGHT ALIPHATIC

For Low Boiling Point Naphthas (LBPNS):

**Acute toxicity:**

LBPNS generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure

Most LBPNS are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices.

**Sensitisation:**

LBPNS do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies

**Repeat dose toxicity:**

The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPNS substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNS in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNS, were considered species- and sex-specific. These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.

Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNS. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3

No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats

No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNS and very few non-cancer chronic toxicity studies were identified for other LBPNS. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3

A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.

**Genotoxicity:**

Although few genotoxicity studies were identified for the site-restricted LBPNS, the genotoxicity of several other LBPNS substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results.

For in vivo genotoxicity tests, LBPNS exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells.

## All Purpose Thinners (Toluene Formulation)

For *in vitro* genotoxicity studies, LBPNS were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNS exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay. Mixed results were observed for UDS and the mouse lymphoma assay.

While the majority of *in vivo* genotoxicity results for LBPNS substances are negative, the potential for genotoxicity of LBPNS as a group cannot be discounted based on the mixed *in vitro* genotoxicity results.

### Carcinogenicity:

Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect

s of human exposure to LBPNS substances.

No inhalation studies assessing the carcinogenicity of the site-restricted LBPNS were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m<sup>3</sup> of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously. Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol. However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPNS group.

Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPNS substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPNS, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans).

Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNS. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light straight-run naphtha and naphtha. Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.

### Reproductive/ Developmental toxicity:

No reproductive or developmental toxicity was observed for the majority of LBPNS substances evaluated. Most of these studies were carried out by inhalation exposure in rodents. NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m<sup>3</sup> (CAS RN 8052-41-3) to 27 687 mg/m<sup>3</sup> (CAS RN 64741-63-5) for the LBPNS groups per evaluated, and from 7690 mg/m<sup>3</sup> to 27 059 mg/m<sup>3</sup> for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m<sup>3</sup>, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted. For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13.

For most LBPNS, no treatment-related developmental effects were observed by the different routes of exposure. However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m<sup>3</sup> delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

### Low Boiling Point Naphthas [Site-Restricted]

For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation.

Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans.

Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants).

Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus.

Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials.

Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.

## ETHYLENE GLYCOL MONOBUTYL ETHER

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. \*\* ASCC (NZ) SDS

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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/m<sup>3</sup>) for EGHE, LC50 > 400ppm (2620 mg/m<sup>3</sup>) for EGBEA to LC50 > 2132 ppm (9061 mg/m<sup>3</sup>) 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 sensitizers 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* cytogenetic 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 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

## All Purpose Thinners (Toluene Formulation)

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/m<sup>3</sup> and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m<sup>3</sup>), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m<sup>3</sup>), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m<sup>3</sup>) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m<sup>3</sup> (rabbit-EGPE), 100 ppm or 425 mg/m<sup>3</sup> (rat-EGPE), 50 ppm or 241 mg/m<sup>3</sup> (rat EGBE) and 100 ppm or 483 mg/m<sup>3</sup> (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m<sup>3</sup> (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 foetotoxicity 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.

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 CO<sub>2</sub>, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO<sub>2</sub>, 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 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 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 embryotoxicity 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.

### All Purpose Thinners (Toluene Formulation) & SOLVENT NAPHTHA PETROLEUM, LIGHT ALIPHATIC

Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.

The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.

## All Purpose Thinners (Toluene Formulation)

### All Purpose Thinners (Toluene Formulation) & TOLUENE

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** - Sternbral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m<sup>3</sup> 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/m<sup>3</sup> 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/m<sup>3</sup> 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/m<sup>3</sup>. 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.

### All Purpose Thinners (Toluene Formulation) & ACETONE

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/m<sup>3</sup> and in rats at 26,100 mg/m<sup>3</sup>. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m<sup>3</sup> for both rats and mice.

Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m<sup>3</sup>, 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/m<sup>3</sup> have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m<sup>3</sup> 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/m<sup>3</sup> or greater.

### TOLUENE & ETHANOL

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

### ACETONE & ETHYLENE GLYCOL MONOBUTYL ETHER

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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

#### Legend:

- ✗ - Data either not available or does not fill the criteria for classification
- ✓ - Data available to make classification

## SECTION 12 Ecological information

### Toxicity

## All Purpose Thinners (Toluene Formulation)

All Purpose Thinners (Toluene Formulation)	Endpoint	Test Duration (hr)	Species	Value	Source
		Not Available	Not Available	Not Available	Not Available

  

toluene	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	3.78mg/L	5
	NOEC(ECx)	168h	Crustacea	0.74mg/L	5
	LC50	96h	Fish	5-35mg/l	4
	EC50	96h	Algae or other aquatic plants	>376.71mg/L	4

  

solvent naphtha petroleum, light aliphatic	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	<0.1mg/l	1
	EC50	72h	Algae or other aquatic plants	6.5mg/l	1
	LC50	96h	Fish	>100000mg/L	4
	EC50	96h	Algae or other aquatic plants	64mg/l	2

  

acetone	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	12h	Fish	0.001mg/L	4
	EC50	48h	Crustacea	6098.4mg/L	5
	LC50	96h	Fish	3744.6-5000.7mg/L	4
	EC50	96h	Algae or other aquatic plants	9.873-27.684mg/l	4

  

ethanol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	275mg/l	2
	EC50	48h	Crustacea	>79mg/L	4
	LC50	96h	Fish	>100mg/l	2
	EC50	96h	Algae or other aquatic plants	<0.001mg/L	4

  

ethylene glycol monobutyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	623mg/l	2
	EC50	48h	Crustacea	164mg/l	2
	EC10(ECx)	48h	Crustacea	7.2mg/l	2
	LC50	96h	Fish	1700mg/l	Not Available
	EC50	96h	Algae or other aquatic plants	720mg/l	2

**Legend:** 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

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

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

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

For Aromatic Substances Series:

Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.

Atmospheric Fate: PAHs are "semi-volatile substances" which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.

Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes > naphthalenes. Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.

For Ethanol:

log Kow: -0.31 to -0.32;

Koc 1: Estimated BCF= 3;

Half-life (hr) air: 144;

Half-life (hr) H2O surface water: 144;

Henry's atm m<sup>3</sup>/mol: 6.29E-06;

BOD 5 if unstated: 0.93-1.67,63%

COD: 1.99-2.11,97%;

ThOD : 2.1.

Environmental Fate: Terrestrial - Ethanol quickly biodegrades in soil but may leach into ground water; most is lost by evaporation. Ethanol is expected to have very high mobility in soil. Volatilization of ethanol from moist soil surfaces is expected to be an important fate process. The potential for volatilization of ethanol from dry soil surfaces may exist. Biodegradation is expected to be an important fate process for ethanol based on half-lives on the order of a few days for ethanol in sandy soil/groundwater microcosms.

Atmospheric Fate: Ethanol is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase ethanol is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 5 days. Ethanol readily degraded by reaction with photochemically produced hydroxy radicals; release into air will result in photodegradation and wet deposition.

Aquatic Fate: When released into water ethanol readily evaporates and is biodegradable. Ethanol is not expected to adsorb to suspended solids and sediment. Volatilization from water surfaces is expected and volatilization half-lives for a model river and model lake are 3 and 39 days, respectively. Bioconcentration in aquatic organisms is considered to be low. Hydrolysis and photolysis in sunlit surface waters is not expected to be an important environmental fate process for ethanol and is unlikely to be persistent in aquatic environments.

For petroleum distillates:

Environmental fate:

When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.

As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.

Continued...

## All Purpose Thinners (Toluene Formulation)

Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus, when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants. The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes.

The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.

### Biodegradation:

Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons. Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:

- (1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
- (2) isoalkanes;
- (3) alkenes;
- (4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
- (5) monoaromatics;
- (6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
- (7) higher molecular weight cycloalkanes (which may degrade very slowly).

Three weathering processes—dissolution in water, volatilization and biodegradation—typically result in the depletion of the more readily soluble, volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.

When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile; this is referred to as residual NAPL. Above the retention capacity, the NAPL becomes mobile and will move within the soil.

### Bioaccumulation:

Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances.

Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with  $\log K_{ow} > -4.5$ .

In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.

Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs.

These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however, one study suggests that some alkyl-PAHs may biomagnify. While only BSAFs were found for some PAHs, it is possible that BSAFs will be  $> 1$  for invertebrates, given that they do not have the same metabolic competency as fish.

In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.

Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.

This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF  $> 5000$ ) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal.

Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish.

### Ecotoxicity:

Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and *Daphnia magna*, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. *Daphnia magna* had a 24-hour LC50 of 1.8 mg/L. The values varied greatly for aquatic species such as rainbow trout and *Daphnia magna*, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. *Daphnia magna* had a 24-hour LC50 of 1.8 mg/L.

The tropical mysid *Metamysidopsis insularis* was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L. This species has been shown to be as sensitive as temperate mysids to toxicants. However, this study used nominal concentrations, and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (*Crangon crangon*) a 96-hour LC50 of 22 mg/L was determined. A "gas oil" was also tested and a 96-hour LC50 of 12 mg/L was determined.

The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species. The diatom *Phaeodactylum tricornutum* showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga *Isochrysis galbana* was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L. Finally, the green algae *Chlorella salina* was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L. All populations of phytoplankton returned to a steady state within 5 days of exposure.

In sandy soils, earthworm (*Eisenia fetida*) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight loss was recorded.

Nephrotoxic effects of diesel fuel have been documented in several animal and human studies. Some species of birds (mallard ducks in particular) are generally resistant to the toxic effects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality.

### For n-hexane:

$\log K_{ow}$ : 3.17-3.94

BOD 5 if unstated: 2.21

COD: 0.04

ThOD: 3.52

### Environmental fate:

**Transport and Partitioning:** The physical properties of n-hexane that affect its transport and partitioning in the environment are: water solubility of 9.5 mg/L;  $\log K_{ow}$  (octanol/water partition coefficient), estimated as 3.29; Henry's law constant, 1.69 atm-m<sup>3</sup> mol; vapor pressure, 150 mm Hg at 25 °C; and  $\log K_{oc}$  in the range of 2.90 to 3.61. As with many alkanes, experimental methods for the estimation of the  $K_{oc}$  parameter are lacking, so that estimates must be made based on theoretical considerations.

The dominant transport process from water is volatilization. Based on mathematical models the half-life for n-hexane in bodies of water with any degree of turbulent mixing (e.g., rivers) would be less than 3 hours. For standing bodies of water (e.g. small ponds), a half-life no longer than one week (6.8 days) is estimated. Based on the  $\log$  octanol/water partition coefficient (i.e.  $\log K_{oc}$ ) and the estimated  $\log$  sorption coefficient (i.e.  $\log K_{oc}$ ) n-hexane is not expected to become concentrated in biota. A calculated bioconcentration factor (BCF) of 453 for a fathead minnow further suggests a low potential for n-hexane to bioconcentrate or bioaccumulate in trophic food chains.

In soil, the dominant transport mechanism for n-hexane present near the surface probably is volatilisation (based on its Henry's law constant, water solubility, vapor pressure, and  $K_{oc}$ ). While its estimated  $K_{oc}$  values suggest a moderate ability to sorb to soil particles, n-hexane has a density (0.6603 g/mL at 20 °C) well below that of water and a very low water solubility of 9.5 mg/L. n-Hexane would, therefore, be viewed as a light nonaqueous phase liquid (LNAPL), which would suggest a low potential for leaching into the lower soil depths since the n-hexane would tend to float on the top of the saturated zone of the water table. n-Hexane would generally stay near the soil surface and, if not appreciably sorbed into the soil matrix, would be expected eventually to volatilise to the atmosphere. Exceptions would involve locations with shallow groundwater tables where there were large spills of hexane products. In such cases, the n-hexane could spread out to contaminant a large volume of soil materials.

**Air:** n-Hexane does not absorb ultraviolet (UV) light at 290 nm and is thus not expected to undergo direct photolysis reactions. The dominant tropospheric removal mechanism for n-hexane is generally regarded to be decomposition by hydroxyl radicals. Calculations assuming typical hydroxyl radical concentrations suggest a half-life of approximately 2.9 days. While n-hexane can react with nitrogen oxides to produce ozone precursors under controlled laboratory conditions, the smog-producing potential of n-hexane is very low compared to that of other alkanes or chlorinated VOCs. Hydroxyl ion reactions in the upper troposphere, therefore, are probably the primary mechanisms for n-hexane degradation in the atmosphere. As with most alkanes, n-hexane is resistant to hydrolysis.

**Water:** Although few data are available dealing explicitly with the biodegradation of n-hexane in water, neither hydrolysis nor biodegradation in surface waters appears to be rapid compared with volatilization. In surface waters, as in the atmosphere, alkanes such as n-hexane would be resistant to hydrolysis. Biodegradation is probably the most significant

## All Purpose Thinners (Toluene Formulation)

degradation mechanism in groundwater. The ability of *Pseudomonas mendocina* bacteria to metabolise *n*-hexane in laboratory microcosms simulating groundwater conditions has been documented. Mixed bacterial cultures as well as pure cultures are documented as capable of metabolizing *n*-hexane under aerobic conditions. In general, linear alkanes (such as *n*-hexane) are viewed as the most readily biodegradable fractions in petroleum, particularly when oxygen is present in solution. Once introduced into groundwater, *n*-hexane may be fairly persistent since its degradation by chemical hydrolysis is slow and opportunities for biodegradation may be limited under anoxic conditions or where nutrients such as nitrogen or phosphorus are in limited supply.

**Sediment and Soil:** The most important biodegradation processes involve the conversion of the *n*-hexane to primary alcohols, aldehydes and, ultimately, into fatty acids. Similar processes are encountered with other light hydrocarbons such as heptane. In general, unless the *n*-hexane is buried at some depth within a soil or sediment, volatilisation is generally assumed to occur at a much more rapid rate than chemical or biochemical degradation processes. Once introduced into deeper sediments, *n*-hexane may be fairly persistent.

**Ecotoxicity:**

Fish LC50 (96 h): Oncorhynchus mykiss 4.14 mg/l; Pimephales promelus 2.5 mg/l (flow through); Lepomis macrochirus 4.12 mg/l

Daphnia EC50 (48 h): 3.87 mg/l

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

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

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

**Ecotoxicity:** Ketones are unlikely to bioconcentrate or biomagnify.

For 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 m<sup>3</sup> /mol : 518-694;

Henry's atm m<sup>3</sup> /mol : 5.94;

E-03BOD 5 0.86-2.12, 5%COD - 0.7-2.52,21-27%;

ThOD - 3.13 ; BCF - 1.67-380;

log BCF - 0.22-3.28.

**Atmospheric Fate:** The majority of toluene evaporates to the atmosphere from the water and soil. The main degradation pathway for toluene in the atmosphere is reaction with photochemically produced hydroxyl radicals. The estimated atmospheric half life for toluene is about 13 hours. Toluene is also oxidized by reactions with atmospheric nitrogen dioxide, oxygen, and ozone, but these are minor degradation pathways. Photolysis is not considered a significant degradative pathway for toluene.

**Terrestrial Fate:** Toluene is moderately retarded by adsorption to soils rich in organic material, therefore, transport to ground water is dependent on soil composition. In unsaturated topsoil containing organic material, it has been estimated that 97% of the toluene is adsorbed to the soil and only about 2% is in the soil-water phase and transported with flowing groundwater. There is little retardation in sandy soils and 2-13% of the toluene was estimated to migrate with flowing water; the remainder was volatilized, biodegraded, or unaccounted for. In saturated deep soils with no soil-air phase, about 48% may be transported with flowing groundwater. In surface soil, volatilization to air is an important fate process for toluene. In the environment, biodegradation of toluene to carbon dioxide occurs with a typical half life of 1-7 days.

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

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

for acetone:

log Kow: -0.24

Half-life (hr) air: 312-1896

Half-life (hr) H2O surface water: 20

Henry's atm m<sup>3</sup> /mol: 3.67E-05

BOD 5: 0.31-1.76,46-55%

COD: 1.12-2.07

ThOD: 2.2

BCF: 0.69

**Environmental fate:**

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

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

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

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

Acetone does not concentrate in the food chain.

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

Drinking Water Standard: none available.

Soil Guidelines: none available.

Air Quality Standards: none available.

**Ecotoxicity:**

Testing shows that acetone exhibits a low order of toxicity

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

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

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

Aquatic invertebrate 2100 - 16700 mg/l

Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephesia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m<sup>3</sup>. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

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

**DO NOT discharge into sewer or waterways.**

Continued...

## All Purpose Thinners (Toluene Formulation)

### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)

### Bioaccumulative potential

Ingredient	Bioaccumulation
toluene	LOW (BCF = 90)
acetone	LOW (BCF = 0.69)
ethanol	LOW (LogKOW = -0.31)
ethylene glycol monobutyl ether	LOW (BCF = 2.51)

### Mobility in soil

Ingredient	Mobility
toluene	LOW (KOC = 268)
acetone	HIGH (KOC = 1.981)
ethanol	HIGH (KOC = 1)
ethylene glycol monobutyl ether	HIGH (KOC = 1)

## SECTION 13 Disposal considerations

### Waste treatment methods

#### Product / Packaging disposal

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.

#### Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- Where possible retain label warnings and SDS and observe all notices pertaining to the product.
- **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.
- Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

## SECTION 14 Transport information

### Labels Required

**Marine Pollutant**

NO

**HAZCHEM**

•3YE

	Land transport (ADG)	Air transport (ICAO-IATA / DGR)	Sea transport (IMDG-Code / GGVSee)
UN number: <b>1993</b> UN proper shipping name: <b>FLAMMABLE LIQUID, N.O.S. Flammable Liquid N.O.S. (65% Liquid Hydrocarbons)</b> Transport hazard class(es): <b>3</b> Subrisk: <b>Not Applicable</b> Packing group: <b>II</b>	Environmental hazard: <b>Not Applicable</b> Special provisions: <b>274</b> Limited quantity: <b>1 L</b>	Environmental hazard: <b>Not Applicable</b> ERG Code: <b>3H</b> Special provisions: <b>A3</b> Cargo Only Packing Instructions: <b>364</b> Cargo Only Maximum Qty / Pack: <b>60 L</b> Passenger and Cargo Packing Instructions: <b>353</b> Passenger and Cargo Maximum Qty / Pack: <b>5 L</b> Passenger and Cargo Limited Quantity Packing Instructions: <b>Y341</b> Passenger and Cargo Limited Maximum Qty / Pack: <b>1 L</b>	Environmental hazard: <b>Not Applicable</b> EMS Number: <b>F-E, S-E</b> Special provisions: <b>274</b> Limited Quantities: <b>1 L</b>

**Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code**

Product name	Group
toluene	Not Available
solvent naphtha petroleum, light aliphatic	Not Available
acetone	Not Available
ethanol	Not Available
ethylene glycol monobutyl ether	Not Available

**Transport in bulk in accordance with the ICG Code**

Product name	Ship Type
toluene	Not Available
solvent naphtha petroleum, light aliphatic	Not Available
acetone	Not Available
ethanol	Not Available
ethylene glycol monobutyl ether	Not Available

**SECTION 15 Regulatory information****Safety, health and environmental regulations / legislation specific for the substance or mixture****toluene is found on the following regulatory lists**

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**solvent naphtha petroleum, light aliphatic is found on the following regulatory lists**

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**acetone is found on the following regulatory lists**

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
- Australian Inventory of Industrial Chemicals (AIIC)

**ethanol is found on the following regulatory lists**

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australian Inventory of Industrial Chemicals (AIIC)

**ethylene glycol monobutyl ether is found on the following regulatory lists**

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
- Australian Inventory of Industrial Chemicals (AIIC)
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**National Inventory Status**

## All Purpose Thinners (Toluene Formulation)

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (toluene; solvent naphtha petroleum, light aliphatic; acetone; ethanol; ethylene glycol monobutyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (solvent naphtha petroleum, light aliphatic)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

## SECTION 16 Other information

Revision Date: 23/08/2022

Initial Date: 23/08/2022

## CONTACT POINT

IMMEDIATELY contact the local POISON CONTROL center for your area (24 hours): Alberta 1-800-332-1414 British Columbia 1-800-567-8911 Manitoba 1-855-776-4766 New Brunswick 911 Newfoundland and Labrador 1-866-727-1110 Northwest Territories 1-800-332-1414 Nova Scotia and Prince Edward Island 1-800-565-8161, 1-800-332-1414 or 911 Nunavut 1-800-268-9017 Ontario 1-800-268-9017 Quebec 1-800-463-5060 Saskatchewan 1-866-454-1212 Yukon Territory 867-393-8700 United States 1-800-222-1222 Contactez IMMÉDIATEMENT le centre ANTIPOISON de votre région (24 heures): Alberta 1-800-332-1414 Colombie-Britannique 1-800-567-8911 Manitoba 1-855-776-4766 Nouveau-Brunswick 911 Terre-Neuve-et-Labrador 1-866-727-1110 Territoires du Nord-Ouest 1-800-332-1414 Nouvelle-Écosse et Île-du-Prince-Édouard 1-800-565-8161, 1-800-332-1414 ou 911 Nunavut 1-800-268-9017 Ontario 1-800-268-9017 Québec 1-800-463-5060 Saskatchewan 1-866-454-1212 Territoire du Yukon 867-393-8700 États-Unis: 1-800-222-1222

## Other information

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

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

## Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average  
 PC—STEL: Permissible Concentration-Short Term Exposure Limit  
 IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit.  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 ES: Exposure Standard  
 OSF: Odour Safety Factor  
 NOAEL :No Observed Adverse Effect Level  
 LOAEL: Lowest Observed Adverse Effect Level  
 TLV: Threshold Limit Value  
 LOD: Limit Of Detection  
 OTV: Odour Threshold Value  
 BCF: BioConcentration Factors  
 BEI: Biological Exposure Index  
 AIIC: Australian Inventory of Industrial Chemicals  
 DSL: Domestic Substances List  
 NDSL: Non-Domestic Substances List  
 IECSC: Inventory of Existing Chemical Substance in China  
 EINECS: European Inventory of Existing Commercial Chemical Substances  
 ELINCS: European List of Notified Chemical Substances  
 NLP: No-Longer Polymers  
 ENCS: Existing and New Chemical Substances Inventory  
 KECI: Korea Existing Chemicals Inventory  
 NZIoC: New Zealand Inventory of Chemicals  
 PICCS: Philippine Inventory of Chemicals and Chemical Substances  
 TSCA: Toxic Substances Control Act  
 TCSI: Taiwan Chemical Substance Inventory  
 INSQ: Inventario Nacional de Sustancias Químicas  
 NCI: National Chemical Inventory  
 FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

Powered by AuthorITe, from Chemwatch.