842UR Silver Conductive Coating

MG Chemicals UK Limited

Version No: **A-2.00**
Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: **05/11/2021**
Revision Date: **24/02/2022**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

1.1. Product Identifier

Product name	842UR
Synonyms	SDS Code: 842UR-Liquid; 842UR-12ML, 842UR-150ML, 842UR-850ML, 842UR-3.6L | UFI:6VK0-70GG-M00Y-X7NM
Other means of identification	Silver Conductive Coating

1.2. Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Polyurethane Conductive Coating
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	**MG Chemicals UK Limited**	**MG Chemicals (Head office)**
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	1210 Corporate Drive Ontario L7L 5R6 Canada
Telephone	+(44) 1663 362888	+(1) 800-340-0772
Fax	Not Available	+(1) 800-340-0773
Website	Not Available	www.mgchemicals.com
Email	sales@mgchemicals.com	Info@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	**Verisk 3E (Access code: 335388)**
Emergency telephone numbers	+(44) 20 35147487
Other emergency telephone numbers	+(0) 800 680 0425

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H225 - Flammable Liquids Category 2, H319 - Serious Eye Damage/Eye Irritation Category 2, H361 - Reproductive Toxicity Category 2, H317 - Sensitisation (Skin) Category 1, H410 - Hazardous to the Aquatic Environment Long-Term Hazard Category 1, H351 - Carcinogenicity Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567*

2.2. Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H225	Highly flammable liquid and vapour.
H319	Causes serious eye irritation.
H361	Suspected of damaging fertility or the unborn child.
H317	May cause an allergic skin reaction.
H410	Very toxic to aquatic life with long lasting effects.
H351	Suspected of causing cancer.

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Supplementary statement(s)

Not Applicable

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.
P233	Keep container tightly closed.
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.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

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.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].

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.

2.3. Other hazards

Inhalation and/or ingestion may produce health damage*.

Cumulative effects may result following exposure*.

May produce discomfort of the respiratory system and skin*.

Repeated exposure potentially causes skin dryness and cracking*.

HARMFUL: may cause lung damage if swallowed

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	SCL / M-Factor	Nanoform Particle Characteristics
1.7440-22-4 2.231-131-3 3.Not Available 4.Not Available	30	silver	Not Applicable	Not Available	Not Available
1.616-38-6 2.210-478-4 3.607-013-00-6 4.Not Available	21	dimethyl carbonate	Flammable Liquids Category 2; H225 [2]	Not Available	Not Available
1.67-64-1 2.200-662-2 3.606-001-00-8 4.Not Available	16	acetone * -	Flammable Liquids Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H225, H319, H336 [2]	Not Available	Not Available

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1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	SCL / M-Factor	Nanoform Particle Characteristics
1.108-65-6 2.203-603-9 3.607-195-00-7 4.Not Available	16	propylene glycol monomethyl ether acetate, alpha-isomer * -	Flammable Liquids Category 3; H226 [2]	Not Available	Not Available
1.108-10-1 2.203-550-1 3.606-004-00-4 4.Not Available	5	methyl isobutyl ketone * -	Flammable Liquids Category 2, Acute Toxicity (Inhalation) Category 4, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3; H225, H332, H319, H335 [2]	Not Available	Not Available
1.85940-94-9 2.Not Available 3.Not Available 4.Not Available	4	hexamethylene diisocyanate homopolymer, MEK-oxime blocked	Acute Toxicity (Inhalation) Category 4, Sensitisation (Skin) Category 1, Sensitisation (Respiratory) Category 1; H332, H317, H334, EUH204 [1]	Not Available	Not Available
1.64742-95-6. 2.247-093-6|265-199-0 3.649-356-00-4 4.Not Available	1	naphtha petroleum, light aromatic solvent **[e]**	Flammable Liquids Category 3, Acute Toxicity (Oral) Category 5, Skin Corrosion/Irritation Category 3, Serious Eye Damage/Eye Irritation Category 2B, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Aspiration Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H226, H303, H316, H320, H336, H305, H411 [1]	Not Available	Not Available
1.95-63-6 2.202-436-9 3.601-043-00-3 4.Not Available	1	1,2,4-trimethyl benzene * -	Flammable Liquids Category 3, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3 , Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H226, H332, H315, H319, H335, H411 [2]	Not Available	Not Available
1.98-82-8 2.202-704-5 3.601-024-00-X 4.Not Available	0.2	cumene * -	Flammable Liquids Category 3, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3 , Aspiration Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H226, H335, H304, H411 [2]	Not Available	Not Available
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties*				

SECTION 4 First aid measures

4.1. 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. ▸ Prostheses 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. Following uptake by inhalation, move person to an area free from risk of further exposure. Oxygen or artificial respiration should be administered as needed. Asthmatic-type symptoms may develop and may be immediate or delayed up to several hours. Treatment is essentially symptomatic. A physician should be consulted.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. 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 acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac

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monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

for simple esters:

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 (5 ml/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.
- 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

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For sub-chronic and chronic exposures to isocyanates:
- This material may be a potent pulmonary sensitiser which causes bronchospasm even in patients without prior airway hyperreactivity.
- Clinical symptoms of exposure involve mucosal irritation of respiratory and gastrointestinal tracts.
- Conjunctival irritation, skin inflammation (erythema, pain vesiculation) and gastrointestinal disturbances occur soon after exposure.
- Pulmonary symptoms include cough, burning, substernal pain and dyspnoea.
- Some cross-sensitivity occurs between different isocyanates.
- Noncardiogenic pulmonary oedema and bronchospasm are the most serious consequences of exposure. Markedly symptomatic patients should receive oxygen, ventilatory support and an intravenous line.
- Treatment for asthma includes inhaled sympathomimetics (epinephrine [adrenalin], terbutaline) and steroids.
- Activated charcoal (1 g/kg) and a cathartic (sorbitol, magnesium citrate) may be useful for ingestion.
- Mydriatics, systemic analgesics and topical antibiotics (Sulamyd) may be used for corneal abrasions.
- There is no effective therapy for sensitised workers.

[Ellenhorn and Barceloux; Medical Toxicology]

NOTE: Isocyanates cause airway restriction in naive individuals with the degree of response dependant on the concentration and duration of exposure. They induce smooth muscle contraction which leads to bronchoconstrictive episodes. Acute changes in lung function, such as decreased FEV1, may not represent sensitivity.

[Karol & Jin, Frontiers in Molecular Toxicology, pp 56-61, 1992]

Personnel who work with isocyanates, isocyanate prepolymers or polyisocyanates should have a pre-placement medical examination and periodic examinations thereafter, including a pulmonary function test. Anyone with a medical history of chronic respiratory disease, asthmatic or bronchial attacks, indications of allergic responses, recurrent eczema or sensitisation conditions of the skin should not handle or work with isocyanates. Anyone who develops chronic respiratory distress when working with isocyanates should be removed from exposure and examined by a physician. Further exposure must be avoided if a sensitivity to isocyanates or polyisocyanates has developed.

SECTION 5 Firefighting measures

5.1. Extinguishing media

- **DO NOT** use halogenated fire extinguishing agents.

Metal dust fires need to be smothered with sand, inert dry powders.

DO NOT USE WATER, CO_2 or FOAM.
- Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- Chemical reaction with CO_2 may produce flammable and explosive methane.
- If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.

5.2. 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

5.3. Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Consider evacuation (or protect in place). ▸ Fight fire from a safe distance, with adequate cover. ▸ If safe, switch off electrical equipment until vapour fire hazard removed. ▸ Use water delivered as a fine spray to control the fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools.

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	▸ **Do not** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	Combustion products include: carbon dioxide (CO2) ▸ **DO NOT** disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ▸ **DO NOT** use water or foam as generation of explosive hydrogen may result. With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present. Metal powders, while generally regarded as non-combustible: ▸ May burn when metal is finely divided and energy input is high. ▸ May react explosively with water. ▸ May be ignited by friction, heat, sparks or flame. ▸ May **REIGNITE** after fire is extinguished. ▸ Will burn with intense heat. Note: ▸ Metal dust fires are slow moving but intense and difficult to extinguish. ▸ Containers may explode on heating. ▸ Dusts or fumes may form explosive mixtures with air. ▸ Gases generated in fire may be poisonous, corrosive or irritating. ▸ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▸ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids ▸ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning. isocyanates and minor amounts of hydrogen cyanide nitrogen oxides (NOx) other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions. When heated at high temperatures many isocyanates decompose rapidly generating a vapour which pressurises containers, possibly to the point of rupture. Release of toxic and/or flammable isocyanate vapours may then occur ▸ Burns with acrid black smoke.

SECTION 6 Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. 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	Chemical Class: ester and ethers For release onto land: recommended sorbents listed in order of priority. SORBENT TYPE | RANK | APPLICATION | COLLECTION | LIMITATIONS LAND SPILL - SMALL cross-linked polymer - particulate | 1 | shovel | shovel | R, W, SS cross-linked polymer - pillow | 1 | throw | pitchfork | R, DGC, RT sorbent clay - particulate | 2 | shovel | shovel | R,I, P wood fiber - particulate | 3 | shovel | shovel | R, W, P, DGC wood fiber - pillow | 3 | throw | pitchfork | R, P, DGC, RT treated wood fiber - pillow | 3 | throw | pitchfork | DGC, RT LAND SPILL - MEDIUM cross-linked polymer - particulate | 1 | blower | skiploader | R,W, SS cross-linked polymer - pillow | 2 | throw | skiploader | R, DGC, RT sorbent clay - particulate | 3 | blower | skiploader | R, I, P polypropylene - particulate | 3 | blower | skiploader | W, SS, DGC expanded mineral - particulate | 4 | blower | skiploader | R, I, W, P, DGC wood fiber - particulate | 4 | blower | skiploader | R, W, P, DGC

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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

- Liquid Isocyanates and high isocyanate vapour concentrations will penetrate seals on self contained breathing apparatus - SCBA should be used inside encapsulating suit where this exposure may occur.

For isocyanate spills of less than 40 litres (2 m2):

- Evacuate area from everybody not dealing with the emergency, keep them upwind and prevent further access, remove ignition sources and, if inside building, ventilate area as well as possible.
- Notify supervision and others as necessary.
- Put on personal protective equipment (suitable respiratory protection, face and eye protection, protective suit, gloves and impermeable boots).
- Control source of leakage (where applicable).
- Dike the spill to prevent spreading and to contain additions of decontaminating solution.
- Prevent the material from entering drains.
- Estimate spill pool volume or area.
- Absorb and decontaminate. - Completely cover the spill with wet sand, wet earth, vermiculite or other similar absorbent. - Add neutraliser (for suitable formulations: see below) to the adsorbent materials (equal to that of estimated spill pool volume). Intensify contact between spill, absorbent and neutraliser by carefully mixing with a rake and allow to react for 15 minutes
- Shovel absorbent/decontaminant solution mixture into a steel drum.
- Decontaminate surface. - Pour an equal amount of neutraliser solution over contaminated surface. - Scrub area with a stiff bristle brush, using moderate pressure. - Completely cover decontaminant with vermiculite or other similar absorbent. - After 5 minutes, shovel absorbent/decontamination solution mixture into the same steel drum used above.
- Monitor for residual isocyanate. If surface is decontaminated, proceed to next step. If contamination persists, repeat decontaminate procedure immediately above
- Place loosely covered drum (release of carbon dioxide) outside for at least 72 hours. Label waste-containing drum appropriately. Remove waste materials for incineration.
- Decontaminate and remove personal protective equipment.
- Return to normal operation.
- Conduct accident investigation and consider measures to prevent reoccurrence.

Decontamination:
Treat isocyanate spills with sufficient amounts of isocyanate decontaminant preparation ('neutralising fluid'). Isocyanates and polyisocyanates are generally not miscible with water. Liquid surfactants are necessary to allow better dispersion of isocyanate and neutralising fluids/ preparations. Alkaline neutralisers react faster than water/surfactant mixtures alone.
Typically, such a preparation may consist of:
Sawdust: 20 parts by weight Kieselguhr 40 parts by weight plus a mixture of {ammonia (s.g. 0.880) 8% v/v non-ionic surfactant 2% v/v water 90% v/v}.
Let stand for 24 hours
Three commonly used neutralising fluids each exhibit advantages in different situations.
Formulation A :
liquid surfactant 0.2-2%
sodium carbonate 5-10%
water to 100%
Formulation B
liquid surfactant 0.2-2%
concentrated ammonia 3-8%
water to 100%
Formulation C
ethanol, isopropanol or butanol 50%
concentrated ammonia 5%
water to 100%

After application of any of these formulae, let stand for 24 hours.

Formulation B reacts faster than Formulation A. However, ammonia-based neutralisers should be used only under well-ventilated conditions to avoid overexposure to ammonia or if members of the emergency team wear suitable respiratory protection. Formulation C is especially suitable for cleaning of equipment from unreacted isocyanate and neutralizing under freezing conditions. Regard has to be taken to the flammability of the alcoholic solution.

- Avoid contamination with water, alkalies and detergent solutions.
- Material reacts with water and generates gas, pressurises containers with even drum rupture resulting.
- **DO NOT** reseal container if contamination is suspected.
- Open all containers with care.
- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- 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 spill with sand, earth or vermiculite.
- Use only spark-free shovels and explosion proof equipment.
- Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

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6.4. Reference to other sections

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

SECTION 7 Handling and storage

7.1. 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. The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe ▸ **DO NOT** concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential. ▸ Any static discharge is also a source of hazard. ▸ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina. ▸ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage. ▸ Add inhibitor to any distillate as required. ▸ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely. The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example. Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised. ▸ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date. ▸ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▸ Unopened containers received from the supplier should be safe to store for 18 months. ▸ Opened containers should not be stored for more than 12 months. **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 ▸ 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.
Fire and explosion protection	See section 5
Other information	for commercial quantities of isocyanates: · Isocyanates should be stored in adequately bunded areas. Nothing else should be kept within the same bunding. Pre-polymers need not be segregated. Drums of isocyanates should be stored under cover, out of direct sunlight, protected from rain, protected from physical damage and well away from moisture, acids and alkalis. · Where isocyanates are stored at elevated temperatures to prevent solidifying, adequate controls should be installed to prevent the high temperatures and precautions against fire should be taken. · Where stored in tanks, the more reactive isocyanates should be blanketed with a non-reactive gas such as nitrogen and equipped with absorptive type breather valve (to prevent vapour emissions).. · Transfer systems for isocyanates in bulk storage should be fully enclosed and use pump or vacuum systems. Warning signs, in appropriate languages, should be posted where necessary. · Areas in which polyurethane foam products are stored should be supplied with good general ventilation. Residual amounts of unreacted isocyanate may be present in the finished foam, resulting in hazardous atmospheric concentrations. · Ideal storage temperature range is dependent on the specific polymer due to viscosity and melting point differences between the polymers. Use 25 deg C (77 deg F) to 30 deg C (86 deg F) as a guideline to most liquid isocyanates for optimum storage temperature. If some isocyanates are stored at or below a temperature of 25 deg C (77 deg F), crystallization and settling of the isocyanate may occur. Storage in a cold warehouse can cause crystals to form. These crystals can settle to the bottom of the container. If crystals do form, they can be melted easily with moderate heat. It is suggested that a container the size of a drum be warmed for 16-24 hours at sufficient temperature to melt the crystals. When the crystals are melted, the container should be agitated by rolling or stirring, until the contents are homogenous. Since heated isocyanate will generate vapors more rapidly than product stored at 25 deg C (77 deg F), be sure to follow the precautions under the Personal Protection. ▸ 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.

7.2. Conditions for safe storage, including any incompatibilities

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Suitable container	▸ **CARE**: Packing of high density product in light weight metal or plastic packages may result in container collapse with product release ▸ Heavy gauge metal packages / Heavy gauge metal drums ▸ 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	Methyl isobutyl ketone (MIBK) ▸ forms unstable and explosive peroxides on contact with air and/ or when in contact with hydrogen peroxide ▸ reacts violently with strong oxidisers, aldehydes, aliphatic amines, nitric acid, perchloric acid, potassium tert-butoxide, strong acids, reducing agents ▸ dissolves some plastics, resins and rubber 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 CO2 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 NOx - 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. ▸ Carbonates are incompatible with cerium compounds, germanium, lead diacetate, magnesium, mercurous chloride, silver nitrate ▸ WARNING: Avoid or control reaction with peroxides. All *transition metal* peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. ▸ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive. ▸ Avoid reaction with borohydrides or cyanoborohydrides ▸ Silver or silver salts readily form explosive silver fulminate in the presence of both nitric acid and ethanol. The resulting fulminate is much more sensitive and a more powerful detonator than mercuric fulminate. ▸ Silver and its compounds and salts may also form explosive compounds in the presence of acetylene and nitromethane. ▸ Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid. ▸ Esters react with acids to liberate heat along with alcohols and acids. ▸ Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products. ▸ Heat is also generated by the interaction of esters with caustic solutions. ▸ Flammable hydrogen is generated by mixing esters with alkali metals and hydrides. ▸ Esters may be incompatible with aliphatic amines and nitrates. ▸ Glycol ethers may form peroxides under certain conditions; the potential for peroxide formation is enhanced when these substances are used in processes such as distillation where they are concentrated or even evaporated to near-dryness or dryness; storage under a nitrogen atmosphere is recommended to minimise the possible formation of highly reactive peroxides ▸ Nitrogen blanketing is recommended if transported in containers at temperatures within 15 deg C of the flash-point and at or above the flash-point - large containers may first need to be purged and inerted with nitrogen prior to loading ▸ In the presence of strong bases or the salts of strong bases, at elevated temperatures, the potential exists for runaway reactions. ▸ Contact with aluminium should be avoided; release of hydrogen gas may result- glycol ethers will corrode scratched aluminium surfaces. ▸ May discolour in mild steel/ copper; lined containers, glass or stainless steel is preferred ▸ Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water . Investigation of the hazards associated with use of 2-butoxyethanol for alloy electropolishing showed that mixtures with 50-95% of acid at 20 deg C, or 40-90% at 75 C, were explosive and initiable by sparks. Sparking caused mixtures with 40-50% of acid to become explosive, but 30% solutions appeared safe under static conditions of temperature and concentration. Ketones in this group: ▸ are reactive with many acids and bases liberating heat and flammable gases (e.g., H2). ▸ react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H2) and heat. ▸ are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides. ▸ react violently with aldehydes, HNO3 (nitric acid), HNO3 + H2O2 (mixture of nitric acid and hydrogen peroxide), and HClO4 (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). Propylene glycol monomethyl ether acetate: ▸ may polymerise unless properly inhibited due to peroxide formation ▸ should be isolated from UV light, high temperatures, free radical initiators ▸ may react with strong oxidisers to produce fire and/ or explosion ▸ reacts violently with with sodium peroxide, uranium fluoride ▸ is incompatible with sulfuric acid, nitric acid, caustics, aliphatic amines, isocyanates, boranes ▸ Segregate from alcohol, water. ▸ Avoid strong acids, bases. ▸ Avoid reaction with oxidising agents, bases and strong reducing agents.

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	· Avoid reaction with water, alcohols and detergent solutions. Isocyanates are electrophiles, and as such they are reactive toward a variety of nucleophiles including alcohols, amines, and even water. Upon treatment with an alcohol, an isocyanate forms a urethane linkage. If a di-isocyanate is treated with a compound containing two or more hydroxyl groups, such as a diol or a polyol, polymer chains are formed, which are known as polyurethanes. Reaction between a di-isocyanate and a compound containing two or more amine groups, produces long polymer chains known as polyureas. · Isocyanates and thioisocyanates are incompatible with many classes of compounds, reacting exothermically to release toxic gases. Reactions with amines, strong bases, aldehydes, alcohols, alkali metals, ketones, mercaptans, strong oxidisers, hydrides, phenols, and peroxides can cause vigorous releases of heat. Acids and bases initiate polymerisation reactions in these materials. · Isocyanates also can react with themselves. Aliphatic di-isocyanates can form trimers, which are structurally related to cyanuric acid. Isocyanates participate in Diels-Alder reactions, functioning as dienophiles · Isocyanates easily form adducts with carbodiimides, isothiocyanates, ketenes, or with substrates containing activated CC or CN bonds. · Some isocyanates react with water to form amines and liberate carbon dioxide. This reaction may also generate large volumes of foam and heat. Foaming spaces may produce pressure in confined spaces or containers. Gas generation may pressurise drums to the point of rupture. · Do NOT reseal container if contamination is expected · Open all containers with care · Base-catalysed reactions of isocyanates with alcohols should be carried out in inert solvents. Such reactions in the absence of solvents often occur with explosive violence, · Isocyanates will attack and embrittle some plastics and rubbers. · The isocyanate anion is a pseudohalide (syn pseudohalogen) whose chemistry, resembling that of the true halogens, allows it to substitute for halogens in several classes of chemical compounds.. The behavior and chemical properties of the several pseudohalides are identical to that of the true halide ions. ▸ A range of exothermic decomposition energies for isocyanates is given as 20-30 kJ/mol. ▸ The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment. ▸ For example, in 'open vessel processes' (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below 500 J/g are unlikely to present a danger, whilst those in 'closed vessel processes' (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g. BRETHERICK: Handbook of Reactive Chemical Hazards, 4th Edition Metals exhibit varying degrees of activity. Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but: ▸ can react exothermically with oxidising acids to form noxious gases. ▸ catalyse polymerisation and other reactions, particularly when finely divided ▸ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds. ▸ Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxide formation on exposure to air. ▸ Safe handling is possible in relatively low concentrations of oxygen in an inert gas. ▸ Several pyrophoric metals, stored in glass bottles have ignited when the container is broken on impact. Storage of these materials moist and in metal containers is recommended. ▸ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric. Factors influencing the pyrophoricity of metals are particle size, presence of moisture, nature of the surface of the particle, heat of formation of the oxide, or nitride, mass, hydrogen content, stress, purity and presence of oxide, among others. ▸ Many metals in elemental form react exothermically with compounds having active hydrogen atoms (such as acids and water) to form flammable hydrogen gas and caustic products. ▸ Elemental metals may react with azo/diazo compounds to form explosive products. ▸ Some elemental metals form explosive products with halogenated hydrocarbons.

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
silver	Inhalation 0.1 mg/m³ (Systemic, Chronic) *Inhalation 0.04 mg/m³ (Systemic, Chronic) ** *Oral 1.2 mg/kg bw/day (Systemic, Chronic) **	0.04 µg/L (Water (Fresh)) 0.86 µg/L (Water - Intermittent release) 438.13 mg/kg sediment dw (Sediment (Fresh Water)) 438.13 mg/kg sediment dw (Sediment (Marine)) 1.41 mg/kg soil dw (Soil) 0.025 mg/L (STP)
dimethyl carbonate	Dermal 5 mg/kg bw/day (Systemic, Chronic) Inhalation 34.9 mg/m³ (Systemic, Chronic) *Dermal 2.5 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 8.7 mg/m³ (Systemic, Chronic) ** *Oral 2.5 mg/kg bw/day (Systemic, Chronic) **	0.5 mg/L (Water (Fresh)) 0.05 mg/L (Water - Intermittent release) 1 mg/L (Water (Marine)) 188 mg/L (STP)
acetone	Dermal 186 mg/kg bw/day (Systemic, Chronic) Inhalation 1 210 mg/m³ (Systemic, Chronic) Inhalation 2 420 mg/m³ (Local, Acute) *Dermal 62 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 200 mg/m³ (Systemic, Chronic) ** *Oral 62 mg/kg bw/day (Systemic, Chronic) **	10.6 mg/L (Water (Fresh)) 1.06 mg/L (Water - Intermittent release) 21 mg/L (Water (Marine)) 30.4 mg/kg sediment dw (Sediment (Fresh Water)) 3.04 mg/kg sediment dw (Sediment (Marine)) 29.5 mg/kg soil dw (Soil) 100 mg/L (STP)
propylene glycol monomethyl ether acetate, alpha-isomer	Dermal 796 mg/kg bw/day (Systemic, Chronic) Inhalation 275 mg/m³ (Systemic, Chronic) Inhalation 550 mg/m³ (Local, Acute) *Dermal 320 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 33 mg/m³ (Systemic, Chronic) ** *Oral 36 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 33 mg/m³ (Local, Chronic) **	0.635 mg/L (Water (Fresh)) 0.064 mg/L (Water - Intermittent release) 6.35 mg/L (Water (Marine)) 3.29 mg/kg sediment dw (Sediment (Fresh Water)) 0.329 mg/kg sediment dw (Sediment (Marine)) 0.29 mg/kg soil dw (Soil) 100 mg/L (STP)

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Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
methyl isobutyl ketone	Dermal 11.8 mg/kg bw/day (Systemic, Chronic) Inhalation 83 mg/m³ (Systemic, Chronic) Inhalation 83 mg/m³ (Local, Chronic) Inhalation 208 mg/m³ (Systemic, Acute) Inhalation 208 mg/m³ (Local, Acute) *Dermal 4.2 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 14.7 mg/m³ (Systemic, Chronic) ** *Oral 4.2 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 14.7 mg/m³ (Local, Chronic) ** *Inhalation 155.2 mg/m³ (Systemic, Acute) ** *Inhalation 155.2 mg/m³ (Local, Acute) **	0.6 mg/L (Water (Fresh)) 0.06 mg/L (Water - Intermittent release) 1.5 mg/L (Water (Marine)) 8.27 mg/kg sediment dw (Sediment (Fresh Water)) 0.83 mg/kg sediment dw (Sediment (Marine)) 1.3 mg/kg soil dw (Soil) 27.5 mg/L (STP)
hexamethylene diisocyanate homopolymer, MEK-oxime blocked	Inhalation 0.502 mg/m³ (Local, Chronic) Inhalation 1.5 mg/m³ (Local, Acute)	100 mg/L (STP)
naphtha petroleum, light aromatic solvent	Inhalation 837.5 mg/m³ (Local, Chronic) Inhalation 1 286.4 mg/m³ (Systemic, Acute) Inhalation 1 066.67 mg/m³ (Local, Acute) *Inhalation 178.57 mg/m³ (Local, Chronic) ** *Inhalation 1 152 mg/m³ (Systemic, Acute) ** *Inhalation 640 mg/m³ (Local, Acute) **	Not Available
1,2,4-trimethyl benzene	Dermal 16 171 mg/kg bw/day (Systemic, Chronic) Inhalation 100 mg/m³ (Systemic, Chronic) Inhalation 100 mg/m³ (Local, Chronic) Inhalation 100 mg/m³ (Systemic, Acute) Inhalation 100 mg/m³ (Local, Acute) *Dermal 9 512 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 29.4 mg/m³ (Systemic, Chronic) ** *Oral 15 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 29.4 mg/m³ (Local, Chronic) ** *Inhalation 29.4 mg/m³ (Systemic, Acute) ** *Inhalation 29.4 mg/m³ (Local, Acute) **	0.12 mg/L (Water (Fresh)) 0.12 mg/L (Water - Intermittent release) 0.12 mg/L (Water (Marine)) 13.56 mg/kg sediment dw (Sediment (Fresh Water)) 13.56 mg/kg sediment dw (Sediment (Marine)) 2.34 mg/kg soil dw (Soil) 2.41 mg/L (STP)
cumene	Dermal 15.4 mg/kg bw/day (Systemic, Chronic) Inhalation 100 mg/m³ (Systemic, Chronic) Inhalation 250 mg/m³ (Local, Acute) *Dermal 1.2 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 16.6 mg/m³ (Systemic, Chronic) ** *Oral 5 mg/kg bw/day (Systemic, Chronic) **	0.035 mg/L (Water (Fresh)) 0.004 mg/L (Water - Intermittent release) 0.012 mg/L (Water (Marine)) 3.22 mg/kg sediment dw (Sediment (Fresh Water)) 0.322 mg/kg sediment dw (Sediment (Marine)) 0.624 mg/kg soil dw (Soil) 200 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	silver	Silver, metallic	0.1 mg/m3	Not Available	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	acetone	Acetone	500 ppm / 1210 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	acetone	Acetone	500 ppm / 1210 mg/m3	3620 mg/m3 / 1500 ppm	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl-2-acetate	50 ppm / 275 mg/m3	550 mg/m3 / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Sk
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	methyl isobutyl ketone	4-Methylpentan-2-one	20 ppm / 83 mg/m3	208 mg/m3 / 50 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	methyl isobutyl ketone	4-Methylpentan-2-one	50 ppm / 208 mg/m3	416 mg/m3 / 100 ppm	Not Available	Sk, BMGV
Europe ECHA Occupational exposure limits - Activity list	hexamethylene diisocyanate homopolymer, MEK-oxime blocked	Not Available	Not Available	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	hexamethylene diisocyanate homopolymer, MEK-oxime blocked	Isocyanates, all (as -NCO) Except methyl isocyanate	0.02 mg/m3	0.07 mg/m3	Not Available	Sen
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	1,2,4-trimethyl benzene	1,2,4-Trimethylbenzene	20 ppm / 100 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	1,2,4-trimethyl benzene	Trimethylbenzenes, all isomers or mixtures	25 ppm / 125 mg/m3	Not Available	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	cumene	2-Phenylpropane (Cumene)	10 ppm / 50 mg/m3	250 mg/m3 / 50 ppm	Not Available	skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	cumene	Cumene	20 ppm / 100 mg/m3	250 mg/m3 / 50 ppm	Not Available	Skin

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	cumene	Cumene	25 ppm / 125 mg/m3	250 mg/m3 / 50 ppm	Not Available	Sk

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
silver	0.3 mg/m3	170 mg/m3	990 mg/m3
dimethyl carbonate	11 ppm	120 ppm	700 ppm
acetone	Not Available	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available	Not Available
methyl isobutyl ketone	75 ppm	500 ppm	3000* ppm
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
1,2,4-trimethyl benzene	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Not Available	Not Available	480 ppm
cumene	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
silver	10 mg/m3	Not Available
dimethyl carbonate	Not Available	Not Available
acetone	2,500 ppm	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
methyl isobutyl ketone	500 ppm	Not Available
hexamethylene diisocyanate homopolymer, MEK-oxime blocked	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
cumene	900 ppm	Not Available

MATERIAL DATA

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)

The adopted TLV-TWA for silver dust and fumes is 0.1 mg/m3 and for the more toxic soluble silver compounds the adopted value is 0.01 mg/m3. Cases of argyria (a slate to blue-grey discolouration of epithelial tissues) have been recorded when workers were exposed to silver nitrate at concentrations of 0.1 mg/m3 (as silver). Exposure to very high concentrations of silver fume has caused diffuse pulmonary fibrosis. Percutaneous absorption of silver compounds is reported to have resulted in allergy. Based on a 25% retention upon inhalation and a 10 m3/day respiratory volume, exposure to 0.1 mg/m3 (TWA) would result in total deposition of no more than 1.5 gms in 25 years.
for propylene glycol monomethyl ether acetate (PGMEA)
Saturated vapour concentration: 4868 ppm at 20 C.
A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternabral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

For trimethyl benzene as mixed isomers (of unstated proportions)
Odour Threshold Value: 2.4 ppm (detection)
Use care in interpreting effects as a single isomer or other isomer mix. Trimethylbenzene is an eye, nose and respiratory irritant. High concentrations cause central nervous system depression. Exposed workers show CNS changes, asthmatic bronchitis and blood dyscrasias at 60 ppm. The TLV-TWA is thought to be protective against the significant risk of CNS excitation, asthmatic bronchitis and blood dyscrasias associated with exposures above the limit.
Odour Safety Factor (OSF)
OSF=10 (1,2,4-TRIMETHYLBENZENE)

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:

Continued...

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

for methyl isobutyl ketone (MIBK):
Unfatigued, odour recognition threshold (100% test panel) is 0.3 - 0.5 ppm.
Distinct odour at 15 ppm.
Odour is objectionable and vapours are irritating to eyes at 200 ppm.
NOTE: Detector tubes for methyl isobutyl ketone, measuring in excess of 50 ppm, are commercially available.
Exposure at or below the recommended TLV-TWA should provide sufficient protection against the potential irritant effects, headache and nausea, neurasthemic symptoms and other systemic toxicities (including liver and kidney damage) produced by MIBK.
The low odour threshold (1.64 mg/m3) and the irritant effects can provide warning of high concentrations. Exposure to levels of 10-410 mg/m3 (2.4-100 ppm) produced perceptible irritation of the eyes, nose, or throat, and 820 mg/m3 (200 ppm) produced discomfort. Symptoms, such as headache, nausea, or vertigo, also occurred at 10-410 mg/m3 (2.4-100 ppm). A 2-h exposure of up to 200 mg/m3 (50 ppm) did not produce any significant effects on a simple reaction-time task or a test of mental arithmetic.
Odour Safety Factor(OSF)
OSF=29 (METHYL ISOBUTYL KETONE)

For cumene:
Odour Threshold Value: 0.008-0.132 ppm (detection), 0.047 ppm (recognition)
Exposure at or below the TLV-TWA is thought to prevent induction of narcosis.
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

8.2. Exposure controls

8.2.1. 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:
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.
For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.
Spraying of material or material in admixture with other components must be carried out in conditions conforming to local state regulations. Local exhaust ventilation with full face air supplied breathing apparatus (hood or helmet type) is normally required. Unprotected personnel must vacate spraying area.
NOTE: Isocyanate vapours will not be adequately absorbed by organic vapour respirators. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

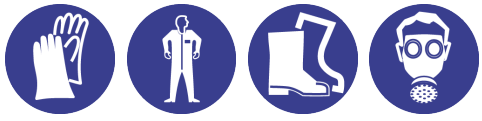
Type of Contaminant:	Air Speed:
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 4-10 m/s (800-2000 f/min.) for extraction of solvents generated by spraying at a point 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
· Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance.
· Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures.
· Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered.. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that te concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)

8.2.2. 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

Continued...

	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	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. For esters: ▸ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. ▸ Isocyanate resistant materials include Teflon, Viton, nitrile rubber and some PVA gloves. ▸ Protective gloves and overalls should be worn as specified in the appropriate national standard. ▸ Contaminated garments should be removed promptly and should not be re-used until they have been decontaminated. ▸ NOTE: Natural rubber, neoprene, PVC can be affected by isocyanates
Body protection	See Other protection below
Other protection	▸ 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 an 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.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
'Forsberg Clothing Performance Index'.
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
842UR Silver Conductive Coating

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as 'feel' or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the 'Exposure Standard' (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- In certain circumstances, personal protection of the individual employee is necessary. Personal protective devices should be regarded as being supplementary to substitution and engineering control and should not be used in preference to them as they do nothing to eliminate the hazard.
- However, in some situations, minimising exposure to isocyanates by enclosure and ventilation is not possible, and occupational exposure standards may be exceeded, particularly during on-site mixing of paints, spray-painting, foaming and maintenance of machine and ventilation systems. In these situations, air-line respirators or self-contained breathing apparatus complying with the appropriate nationals standard must be used.
- Organic vapour respirators with particulate pre- filters and powered, air-purifying respirators are NOT suitable.
- Personal protective equipment must be appropriately selected, individually fitted and workers trained in their correct use and maintenance. Personal protective equipment must be regularly checked and maintained to ensure that the worker is being protected.
- Air- line respirators or self-contained breathing apparatus complying with the appropriate national standard should be used during the clean-up of spills and the repair or clean-up of contaminated equipment and similar situations which cause emergency exposures to hazardous atmospheric concentrations of isocyanate.

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator

Continued...

up to 10	1000	AX-AUS / Class 1	-
up to 50	1000	-	AX-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	AX-2
up to 100	10000	-	AX-3
100+		-	Airline**

** - Continuous-flow or positive pressure demand.
A(All classes) = Organic vapours, B AUS or B1 = Acid gases, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 deg C)

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	Silver

Physical state	Liquid	**Relative density (Water = 1)**	1.33
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	3.02
Initial boiling point and boiling range (°C)	>56	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	-17	**Taste**	Not Available
Evaporation rate	<1 BuAC = 1	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	12	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	2.4	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	<2.01	**VOC g/L**	Not Available
Nanoform Solubility	Not Available	**Nanoform Particle Characteristics**	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2

Continued...

10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	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 main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression , headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures. The material has **NOT** been classified by EC Directives or other classification systems as 'harmful by inhalation'. This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols. Not normally a hazard due to non-volatile nature of product A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias. High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement. The vapour/mist may be highly irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning for several hours after exposure. Sensitized people can react to very low doses, and should not be allowed to work in situations allowing exposure to this material. Continued exposure of sensitised persons may lead to possible long term respiratory impairment. Inhalation hazard is increased at higher temperatures. Mice exposed at up to 3000 ppm PGMEA 6 hr/day for a total of 9 days during an 11-day period showed no pronounced effect on the weights of liver, kidneys, heart, spleen, thymus or testes. Histopathological examination revealed degeneration of the olfactory epithelium in mice exposed at 300 ppm for the same time. Rats, similarly failed to show changes in internal organs and did not show olfactory epithelium degeneration until 3000 ppm. The no-effect level in rats was 1000 ppm. 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. The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics. Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced. Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics. Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. 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. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.
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 has **NOT** been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant 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

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	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. Repeated application of commercial grade PGMEA to the skin of rabbits for 2-weeks caused slight redness and very slight exfoliation. 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.
Eye	Evidence exists, or practical experience predicts, that the material may cause 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. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Undiluted propylene glycol monomethyl ether acetate (PGMEA) causes moderate discomfort, slight conjunctival redness and slight corneal injury in rabbits
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. 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. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats Which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer . beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). Silver is one of the most physically and physiologically cumulative of the elements. Chronic exposure to silver salts may cause argyria, a permanent ashen-grey discolouration of the skin, conjunctiva and internal organs (due to the deposit of an insoluble albuminate of silver). The respiratory tract may also be a site of local argyria (following chronic inhalation exposures) with a mild chronic bronchitis being the only obvious symptom. Sub-chronic exposure to a substance containing silver results in elevated alkaline phosphatase levels along with pigmentation of the tissues and organs. These effects are commonly observed in studies on silver. Organ and tissue pigmentation appears to be an intrinsic property of silver ions, constituting an early marker of silver toxicity. This effect is therefore taken into consideration for the derivation of toxicicological reference values. The lowest NOAELs for the medium- and long-term toxicity of silver ions were based respectively on the 90-day study of rats conducted with silver sodium hydrogen and zirconium phosphate and on the 105-week combined chronic study on rats conducted with silver-zinc zeolite. These NOAELs were recalculated to take account of the silver content of the substance tested and the rate of release of the silver ions. In order to derive the toxicological reference values, an oral absorption of 5% and a safety factor of 100 (10 for intra-species variability and 10 for inter-species variability) were used. In the absence of any observed acute toxicity effect, it is not possible to define a toxicity reference value for short-term exposure. The conservative approach set out in the European assessment is to use the medium-term acceptable exposure limit (AEL) as the short-term AEL. This value is based on the no observed effect level in rats exposed for 90 days. · Short/medium-term AEL = 0.3 mg/kg bw/d x 5% / 100 = 0.15 µg/kg bw/d (silver ion equivalent) · Long-term AEL = 0.09 mg/kg bw/d x 5% / 100 = 0.045 µg/kg bw/d (silver ion equivalent) In a 2015 opinion on the classification of silver-zinc zeolite, the ECHA Committee for Risk Assessment (RAC) concluded that there was a potential embryotoxic effect in rats at doses where the dams were not severely affected by the treatment. This was manifested primarily by a decrease in the viability of the foetuses/pups, observed to varying degrees in developmental toxicity studies conducted with silver chloride (post-implantation losses, mortality of all offspring, increased incidence of hydronephrosis and cryptorchidism) and silver acetate (slight increase in the percentage of litters with late foetal death) and in a two-generation study with silver-zinc zeolite (lower number of births (F19), higher stillbirth rate, lower live birth rate, reduced pup weight, lower thymus weight, increased incidence of hydronephrosis. A two-generation study of rats conducted with a different active substance containing silver also observed a lower number of births (F1), along with a smaller live litter size on day 1 (F210), and a lower thymus weight. Repeated exposure to higher concentrations of propylene glycol monomethyl ether acetate (PGMEA) (1000 ppm and above) causes mild liver and kidney damage in animals. A minor component, 2-methoxy-1-propyl acetate (the beta-isomer) produced birth defects on inhalation exposure of pregnant rabbits at 545 ppm, but not at 145 or 36 ppm; maternal and embryo/foetal toxicity on inhalation exposure of pregnant rats at 2710 ppm, but not at 545 or 110 ppm; and no adverse effects on dermal exposure of pregnant rabbits at applied dosages of 1000 and 2000 mg/kg of body weight per day during the

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critical period or embryo/foetal development. In a further study, no developmental effects were seen following exposure of pregnant rats at air concentrations of commercial propylene glycol monomethyl ether acetate (containing 3-5% of the minor component) up to 4000 ppm; slight maternal effects were seen at 5000 ppm and greater.
Exposure of pregnant rats and rabbits to the parent glycol ether, propylene glycol monomethyl ether which contained comparable amounts of the primary isomer, 2-methoxy-1-propanol, did not produce teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rat foetuses but not in rabbit foetuses at this concentration and maternal toxicity was noted in both species at this concentration
Persons with a history of asthma or other respiratory problems or are known to be sensitised, should not be engaged in any work involving the handling of isocyanates.
The chemistry of reaction of isocyanates, as evidenced by MDI, in biological milieu is such that in the event of a true exposure of small MDI doses to the mouth, reactions will commence at once with biological macromolecules in the buccal region and will continue along the digestive tract prior to reaching the stomach. Reaction products will be a variety of polyureas and macromolecular conjugates with for example mucus, proteins and cell components.
This is corroborated by the results from an MDI inhalation study. Following an inhalation exposure of rats to radiolabelled MDI, 79% of the dose was excreted in faeces. The faecal excretion in these animals was considered entirely due to ingestion of radioactivity from grooming and ingestion of deposited material from the nasopharangeal region via the mucociliary escalator, i.e. not following systemic absorption. The faecal radioactivity was tentatively identified as mixed molecular weight polyureas derived from MDI. Diamine was not present. Thus, for MDI and diisocyanates in general the oral gavage dosing route is inappropriate for toxicological studies and risk assessment.
It is expected that oral gavage dosing will result in a similar outcome to that produced by TDI or MDI, that is (1) reaction with stomach contents and (2) polymerization to solid polyureas.

- Reaction with stomach contents is very plausibly described in case reports of accidental ingestion of polymeric MDI based glue in domestic animals. Extensive polymerization and CO2 liberation resulting in an expansion of the gastric content is described in the stomach, without apparent acute chemical toxicity
- Polyurea formation in organic and aqueous phases has been described. In this generally accepted chemistry of hydrolysis of an isocyanate the initially produced carbamate decarboxylates to an amine which. The amine, as a reactive intermediate, then reacts very readily with the present isocyanate to produce a solid and inert polyurea. This urea formation acts as a pH buffer in the stomach, thus promoting transformation of the diisocyanate into polyurea, even under the acidic conditions.

At the resorbtive tissues in the small intestine, these high molecular reaction products are likely to be of very low bioavailability, which is substantiated by the absence of systemic toxicity in acute oral bioassays with rats at the OECD limit dose (LC50>2 g/kg bw).
The respiratory tract may be regarded as the main entry for systemically available isocyanates as evidenced following MDI.exposures.
A detailed summary on urinary, plasma and in vitro metabolite studies is provided below. Taken together, all available studies provide convincing evidence that MDI-protein adduct and MDI-metabolite formation proceeds:

- via formation of a labile isocyanate glutathione (GSH)-adduct,
- then transfer to a more stable adduct with larger proteins, and
- without formation of free MDA. MDA reported as a metabolite is actually formed by analytical workup procedures (strong acid or base hydrolysis) and is not an identified metabolite in urine or blood

Metallic dusts generated by the industrial process give rise to a number of potential health problems. The larger particles, above 5 micron, are nose and throat irritants. Smaller particles however, may cause lung deterioration. Particles of less than 1.5 micron can be trapped in the lungs and, dependent on the nature of the particle, may give rise to further serious health consequences.
Metals are widely distributed in the environment and are not biodegradable. Biologically, many metals are essential to living systems and are involved in a variety of cellular, physiological, and structural functions. They often are cofactors of enzymes, and play a role in transcriptional control, muscle contraction, nerve transmission, blood clotting, and oxygen transport and delivery. Although all metals are potentially toxic at some level, some are highly toxic at relatively low levels. Moreover, in some cases the same metal can be essential at low levels and toxic at higher levels, or it may be toxic via one route of entry but not another. Toxic effects of some metals are associated with disruption of functions of essential metals. Metals may have a range of effects, including cancer, neurotoxicity, immunotoxicity, cardiotoxicity, reproductive toxicity, teratogenicity, and genotoxicity. Biological half lives of metals vary greatly, from hours to years. Furthermore, the half life of a given metal varies in different tissues. Lead has a half life of 14 days in soft tissues and 20 years in bone.
In considering how to evaluate the toxicity of metals of potential concern, a number of aspects of metal toxicity should be kept in mind:
Different species vary in their responses to different metals; in some cases, humans are more sensitive than rodents. Thus, there is a need for broad-based testing of metals;

- The route of exposure may affect the dose and site where the metal concentrates, and thus the observed toxic effects;
- Metal-metal interactions can reduce or enhance toxicity; biotransformation can reduce or enhance toxicity;
- It is difficult to predict the toxicity of one metal based on the adverse effects of another; in trying to evaluate the toxicity of one particular metal compound, predictions based on similar compounds of the same metal may be valid.

842UR Silver Conductive Coating

TOXICITY	IRRITATION
Not Available	Not Available

silver

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; >5.16 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50; >2000 mg/kg[2]	

dimethyl carbonate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; >5.36 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50; >5000 mg/kg[1]	

acetone

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 20000 mg/kg[2]	Eye (human): 500 ppm - irritant
Inhalation(Mouse) LC50; 44 mg/L4h[2]	Eye (rabbit): 20mg/24hr -moderate
Oral (Rat) LD50; 5800 mg/kg[2]	Eye (rabbit): 3.95 mg - SEVERE
	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit): 500 mg/24hr - mild

Continued...

	Skin (rabbit):395mg (open) - mild
	Skin: no adverse effect observed (not irritating)[1]

propylene glycol monomethyl ether acetate, alpha-isomer

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50; 3739 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

methyl isobutyl ketone

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >16000 mg/kg[1]	Eye (human): 200 ppm/15m
Inhalation(Rat) LC50; ~8.2-16.4 mg/l4h[2]	Eye (rabbit): 40 mg - SEVERE
Oral (Rat) LD50; 2080 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
	Skin (rabbit): 500 mg/24h - mild

hexamethylene diisocyanate homopolymer, MEK-oxime blocked

TOXICITY	IRRITATION
dermal (rat) LD50: >2667 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; >2.757 mg/L4h[1]	Skin: adverse effect observed (irritating)[1]
Oral (Rat) LD50; >2000 mg/kg[1]	

naphtha petroleum, light aromatic solvent

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

1,2,4-trimethyl benzene

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >3160 mg/kg[2]	Not Available
Inhalation(Rat) LC50; 18 mg/L4h[2]	
Oral (Rat) LD50; 6000 mg/kg[1]	

cumene

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 2000 mg/kg[2]	Eye (rabbit): 500 mg/24h mild
Inhalation(Rat) LC50; 39 mg/L4h[2]	Eye (rabbit): 86 mg mild
Oral (Rat) LD50; 1400 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Skin (rabbit): 10 mg/24h mild
	Skin (rabbit):100 mg/24h moderate
	Skin: no adverse effect observed (not irritating)[1]

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*

842UR Silver Conductive Coating

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.
Generally,linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized
Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw
Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic.
The JEFCA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of

Continued...

	aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods **Internationl Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)** **Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998**
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/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals. The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I] *Shin-Etsu SDS
METHYL ISOBUTYL KETONE	For methyl isobutyl ketone (MIBK): MIBK is primarily absorbed by the lungs in animals and humans; it can however be absorbed by the gastrointestinal system and through skin. In two cases involving individuals exposed to the vapour MIBK was found in the brain, liver, lung, vitreous fluid, kidney and blood. Experiments in guinea pigs show that MIBK is metabolised to 4-hydroxy-4-methyl-2-pentanone and 4-methyl-2-pentanol. Ketones are generally excreted rapidly in expired air. Small amounts of MIBK are also excreted in the urine. Humans excreted less than 0.1% of the dose as unmetabolised MIBK in the urine within the first 3 hours post exposure. Serum half-life in guinea pigs is about 55 minutes with a clearance time of 6 hours In animal studies, the acute systemic toxicity of MIBK, via the oral and inhalation routes of exposure, is low. In a 90-day gavage study on rats, a no-observed-effect level (NOEL) of 50 mg/kg per day was found. In 90-day inhalation studies on rats and mice, concentrations of up to 4100 mg/m3 (1000 ppm) did not result in significant toxicity, though compound-related reversible morphological changes were reported in the liver and kidney. Evidence of central nervous system depression was seen in animals exposed to a level of 4100 mg/m3 (1000 ppm). In a number of studies, exposure to MIBK concentrations as low as 1025 mg/m3 (250 ppm) resulted in an increase in liver size and induced hepatic microsomal metabolism. This may be responsible for the exacerbation of haloalkane toxicity and for the potentiation of the neurotoxicity of *n*-hexane. MIBK was also found to potentiate the cholestatic effects of manganese given with, or without, bilirubin. In 90-day studies on mice, rats, dogs, and monkeys, only male rats developed hyaline droplets in the proximal tubules of the kidney. Effects on behaviour were reported in baboons exposed for 7 days to 205 mg/m3 (50 ppm). At a concentration of 4100 mg/m3 (1000 ppm), MIBK was not embryotoxic, foetotoxic, or teratogenic in rats or mice. Foetotoxicity was only observed at concentrations of MIBK that caused maternal toxicity. MIBK did not induce gene mutations in *in vitro* bacterial test systems with, or without, metabolic activation. Negative results were also obtained *in vitro* with, or without, metabolic activation, in tests for mitotic gene conversion in yeast, and for gene mutation in cultured mammalian cells. The results of *in vitro* assays for unscheduled DNA synthesis in primary rat hepatocytes and for structural chromosome damage in cultured rat liver cells were negative. An *in vivo* micronucleus test on mice was negative. These data indicate that MIBK is not genotoxic. No long-term or carcinogenicity studies are available. The toxicity of MIBK for aquatic organisms and microorganisms is low.
HEXAMETHYLENE DIISOCYANATE HOMOPOLYMER, MEK-OXIME BLOCKED	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. No significant acute toxicological data identified in literature search. In a subacute 14-day inhalation study in Wistar rats, aersosol exposure caused concentration dependent pulmonary toxicity as indicated by the increased lung weights and the histological changes of lung and mediastinal lymph nodes. The lowest tested concentration of 30.5 mg/m3 was the No Observed Effect Concentration (NOEC) under the test condition. In a subchronic 90-day inhalation study in Wistar rats, aersosol exposure caused concentration dependent pulmonary lesions as indicated by the increased weights of lung and corresponding histological findings in lungs and mediastinal lymph nodes. These effects may have become systemically effective and caused increased total white blood cell counts, which is triggered by the increased neutrophil counts. Overall, at the high concentration of 150 mg/m3 (measured concentration 149 +/-7 mg/m3) the histological changes in lungs and mediastinal lymph nodes, the changes in differential white blood cell count were not reversible within 4 weeks recovery period. Moreover, less severe, histopathological changes were also observed in nasal cavity (likely adaptive) and trachea (full reversible). No histopathological effects were seen in any other organ outside the respiratory tract. The lowest tested concentration of 5.2 mg/m3 is the No Observed Effect Concentration (NOEC) under the current test conditions.
NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe] 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 LBPN 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.

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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 LBPN 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.
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 LBPN 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 LBPN 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/m3 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 LBPN group.
Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPN 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 LBPN, 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 LBPN 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/m3 (CAS RN 8052-41-3) to 27 687 mg/m3 (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m3 to 27 059 mg/m3 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/m3, 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/m3 delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

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Low Boiling Point Naphthas [Site-Restricted]
Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins.
The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the 'hydrocarbon continuum hypothesis', and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.
For C9 aromatics (typically trimethylbenzenes - TMBs)
Acute Toxicity
Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50 s range from 6,000 to 10,000 mg/m 3 for C9 aromatic naphtha and 18,000 to 24,000 mg/m3 for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.
Irritation and Sensitization
Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.
Repeated Dose Toxicity
Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m3). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m3, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m3) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.
The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m3. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m3). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m3 (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m3 for respiratory irritation and 250 ppm or 1230 mg/m3 for systemic effects.
Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.
Mutagenicity
In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m3) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category
Reproductive and Developmental Toxicity
Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m3 , respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex /group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex /group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.
Systemic Effects on Parental Generations:
The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m3).
Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m3). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation,, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m3), which excludes analysis of the highest concentration due to excessive mortality.

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	Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m3) based on the body weights reductions observed in the F3 offspring. Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity. for petroleum: Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic. This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents **Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans. **Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays. **Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed. **Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.
1,2,4-TRIMETHYL BENZENE	Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene CHEMWATCH 2325 1,3,5-trimethylbenzene
CUMENE	Cumene is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals. Cumene caused tumours at several tissue sites, including lung and liver in mice and kidney in male rats. Several proposed mechanisms of carcinogenesis support the relevance to humans of lung and liver tumours in experimental animals. Specifically, there is evidence that humans and experimental animals metabolise cumene through similar metabolic pathways. There is also evidence that cumene is genotoxic in some tissues, based on findings of DNA damage in rodent lung and liver. Furthermore, mutations of the K-ras oncogene and p53 tumor-suppressor gene observed in cumene-induced lung tumours in mice, along with altered expression of many other genes, resemble molecular alterations found in human lung and other cancers. The relevance of the kidney tumors to cancer in humans is uncertain; there is evidence that a species-specific mechanism not relevant to humans contributes to their induction, but it is possible that other mechanisms relevant to humans, such as genotoxicity, may also contribute to kidney-tumour formation in male rats. 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. For aromatic terpenes: **Acute toxicity:** Mammalian LD50 for p-cymene have shown it to have low toxic potential. Similar studies with cumene have concurred with these results In general, the studies indicate that p-cymene (p-methylisopropylbenzene) or cumene (isopropylbenzene) is rapidly absorbed by oral or inhalation routes. They undergo oxidation (hydroxylation) of the side chain isopropyl substituent and, in the case of p-cymene, the methyl substituent to yield polar oxygenated metabolites. These metabolites are either excreted unchanged in the urine or undergo Phase II conjugation with glucuronic acid and/or glycine followed by excretion in the urine. Unchanged p-cymene or cumene were not detected in the urine or faeces. Humans (5 males and 5 females/group) exposed to an atmosphere containing 49, 98, or 147 ppm cumene for 7 hours showed 64% absorption at 0.5 hours and 45% at 7 hours. Maximum excretion is observed at 6 to 8 hours and is essentially complete at 48 hours. Approximately 35% of the dose inhaled was excreted as 2-phenyl-2-propanol **Repeat Dose Toxicity:** Subacute Studies: Groups of 7 to 12 male rats were exposed to 0, 50, or 250 ppm of p-cymene for 6 hours/day, 5 days/week for 4 weeks with an 8-week recovery period. there was no overt toxicity in the treated rats and no effect on body weight or terminal weight of the brain, cerebellum or whole brain. There was also no effect on regional enzyme activities, regional protein synthesis or regional neurotransmitter concentrations. Cumene has been tested by the National Toxicology Program (NTP) in both rats and mice. Animals were exposed to up to 4,000 ppm cumene by whole-body inhalation for 12-13 days over a period of 16-17 days. In rats, all animals died at 4,000 ppm, and about half the animals died at the next exposure concentration (2,000 ppm). Varying degrees of ataxia were reported in surviving rats exposed to 500 to 2,000 ppm cumene. Increased relative liver and kidney weights were reported in rats exposed to cumene. In exposed male rats, hyaline droplets in the renal cortical tubules were reported. At 2,000 ppm, superlative inflammation of the lung was reported in 40% of the rats. In mice, all animals died at the 2 highest exposures (2,000 and 4,000 ppm). At 1,000 ppm, 80% of the female mice died and male mice showed varying degrees of ataxia. Increased relative liver and kidney weights were reported in mice exposed to cumene. Decreased thymus weight was reported in male mice exposed to 1,000 ppm of cumene. No histopathological findings accompanied the organ weight changes. A NOAEL of 1,000 ppm was determined for female rats and male mice and a NOAEL of 500 ppm was determined for female mice based on mortality and histopathological findings. **Chronic toxicity:** The US EPA concluded that there is some evidence that suggests that cumene is not likely to produce a carcinogenic response (i.e., numerous genotoxic tests, including gene mutation, chromosomal aberration, and primary DNA damage tests, all but one of which were negative or not reproducible) In addition, EPA noted that cumene does not appear to metabolise to highly reactive chemical species and in terms of metabolism, cumene is analogous to methyl benzene for which a 2-year inhalation study was conducted by NTP and no evidence of carcinogenic activity was reported in either rats or mice. Given that the only structural difference between p-cymene and cumene is the presence of a second alkyl substituent (isopropylbenzene versus p-methylisopropylbenzene), similar conclusions can be drawn for p-cymene, particularly since the pharmacokinetic, metabolic and toxicologic data that are available support this conclusion. **Reproductive toxicity:** Taking into consideration the rapid metabolism and excretion of cumene, the US EPA concluded, “cumene has low potential for reproductive toxicity.” **Developmental toxicity:** Even at maternally toxic concentrations exposure to cumene vapor did not produce developmental toxicity in rats. However the US EPA determined that the changes in gestational parameters of the rabbits, though not significant, were consistent in indicating possible developmental effects and therefore set the NOAEL in rabbits for both developmental and maternal effects at 1,206 ppm and the LOAEL at 2,297 ppm, respectively (as reported in EPA, 1997). Since both cumene and p-cymene exhibit such similar pharmacokinetic and metabolic profiles, and show no evidence of toxicity at levels of exposure similar to those experienced by humans, further teratogenic or developmental testing is not recommended **Genotoxicity:** The genotoxicity database on p-cymene and cumene shows no mutagenic potential in the Ames assay. In cytogenetic assays,

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	there is no evidence of a genotoxic potential in vitro. In whole animals, the genotoxicity results for cumene are mixed showing weakly positive results in micronuclei induction in rats, but no evidence of genotoxicity in mice. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
842UR Silver Conductive Coating & METHYL ISOBUTYL KETONE & 1,2,4-TRIMETHYL BENZENE & CUMENE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
842UR Silver Conductive Coating & HEXAMETHYLENE DIISOCYANATE HOMOPOLYMER, MEK-OXIME BLOCKED	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
842UR Silver Conductive Coating & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & 1,2,4-TRIMETHYL BENZENE	For trimethylbenzenes: Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion . After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates . The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid . The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates. **Acute Toxicity** Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis . High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness . The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg) . Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels . No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days **Neurotoxicity** 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested. **Subchronic/Chronic Toxicity** Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia . **Genotoxicity:** Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella tymphimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation. **Developmental/Reproductive Toxicity:** A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established Developmental toxicity, including possible develop- mental neurotoxicity, was evident in rats in a 3-generation reproductive study No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation
842UR Silver Conductive Coating & PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the

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	alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I]
ACETONE & METHYL ISOBUTYL KETONE	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.
METHYL ISOBUTYL KETONE & CUMENE	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

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*

11.2.1. Endocrine Disruption Properties

Many chemicals may mimic or interfere with the body s hormones, known as the endocrine system. Endocrine disruptors are chemicals that can interfere with endocrine (or hormonal) systems. Endocrine disruptors interfere with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body. Any system in the body controlled by hormones can be derailed by hormone disruptors. Specifically, endocrine disruptors may be associated with the development of learning disabilities, deformations of the body various cancers and sexual development problems. Endocrine disrupting chemicals cause adverse effects in animals. But limited scientific information exists on potential health problems in humans. Because people are typically exposed to multiple endocrine disruptors at the same time, assessing public health effects is difficult.

SECTION 12 Ecological information

12.1. Toxicity

842UR Silver Conductive Coating

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

silver

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	120h	Fish	<0.001mg/L	4
LC50	96h	Fish	0.006mg/l	2
EC50	72h	Algae or other aquatic plants	11.89mg/l	2

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	EC50	48h	Crustacea	0.001mg/l	2
	EC50	96h	Algae or other aquatic plants	0.002mg/L	4

dimethyl carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	25mg/l	2
LC50	96h	Fish	>=100mg/l	2
EC50	72h	Algae or other aquatic plants	>57.29mg/l	2
EC50	48h	Crustacea	>74.16mg/l	2
EC50	96h	Algae or other aquatic plants	166.6-211mg/l	2

acetone

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

propylene glycol monomethyl ether acetate, alpha-isomer

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	336h	Fish	47.5mg/l	2
LC50	96h	Fish	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>1000mg/l	2
EC50	48h	Crustacea	373mg/l	2
EC50	96h	Algae or other aquatic plants	>1000mg/l	2

methyl isobutyl ketone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48h	Crustacea	170mg/l	1
LC50	96h	Fish	>179mg/l	2
EC50	48h	Crustacea	170mg/l	1
EC50	96h	Algae or other aquatic plants	400mg/l	1

hexamethylene diisocyanate homopolymer, MEK-oxime blocked

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48h	Crustacea	>1.61mg/l	2
LC50	96h	Fish	141.4mg/l	2
EC50	72h	Algae or other aquatic plants	>8.1mg/l	2
EC50	48h	Crustacea	>1.61mg/l	2

naphtha petroleum, light aromatic solvent

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
EC50	72h	Algae or other aquatic plants	19mg/l	1
EC50	48h	Crustacea	6.14mg/l	1
EC50	96h	Algae or other aquatic plants	64mg/l	2

1,2,4-trimethyl benzene

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	31-207	7
EC50(ECx)	96h	Algae or other aquatic plants	2.356mg/l	2
LC50	96h	Fish	3.41mg/l	2
EC50	48h	Crustacea	ca.6.14mg/l	1
EC50	96h	Algae or other aquatic plants	2.356mg/l	2

cumene

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	96h	Crustacea	0.4mg/l	1
LC50	96h	Fish	2.7mg/l	2
EC50	72h	Algae or other aquatic plants	1.29mg/l	2
EC50	48h	Crustacea	4mg/l	1

Legend: *Extracted from 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*

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Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the
oxygen transfer between the air and the water
Oils of any kind can cause:

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

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.
for propylene glycol ethers:
Environmental fate:
Most are liquids at room temperature and all are water-soluble.
Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM)
Environmental fate: Log octanol-water partition coefficients (log Kow's) range from 0.309 for TPM to 1.523 for DPnB. Calculated BCFs range from 1.47 for DPnB to 3.16 for DPMA and TPM, indicating low bioaccumulation. Henry's Law Constants, which indicate propensity to partition from water to air, are low for all category members, ranging from 5.7 x 10-9 atm-m3/mole for TPM to 2.7 x10-9 atm-m3/mole for PnB. Fugacity modeling indicates that most propylene glycol ethers are likely to partition roughly equally into the soil and water compartments in the environment with small to negligible amounts remaining in other environmental compartments (air, sediment, and aquatic biota). Propylene glycol ethers are unlikely to persist in the environment. Once in air, the half-life of the category members due to direct reactions with photochemically generated hydroxyl radicals, range from 2.0 hours for TPM to 4.6 hours for PnB. In water, most members of this family are 'readily biodegradable' under aerobic conditions. (DPMA degraded within 28 days (and within the specified 10-day window) but only using pre-adapted or 'acclimated' inoculum.). In soil, biodegradation is rapid for PM and PMA.
Ecotoxicity:
Acute aquatic toxicity testing indicates low toxicity for both ethers and acetates. For ethers, effect concentrations are > 500 mg/L. For acetates, effect concentrations are > 151 mg/L.
For 1,2,4-trimethylbenzene:
Half-life (hr) air : 0.48-16
Half-life (hr) H2O surface water : 0.24-672
Half-life (hr) H2O ground : 336-1344
Half-life (hr) soil : 168-672
Henry's Pa m3 /mol: 385-627
Bioaccumulation : not significant
1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance. As a VOC, 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs.
Environmental fate:
Transport: ,1,2,4-Trimethylbenzene volatilises rapidly from surface waters as predicted by a Henry's law constant of 5.18 x 10-3 (vapor pressure, 2.03 mm Hg). The volatilisation half-life from a model river is calculated to be 3.4 hours. The chemical also volatilises from soils, however, based on an estimated Koc of 472, moderate adsorption to soils and sediments may occur
Transformation/Persistence
Air - Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals Reaction also occurs with ozone but very slowly (half life, 8820 days) In the atmosphere, two estimates of the half-life are approximately 6 hours and, in the presence of hydroxyl radicals, 0.5 days
Soil - Volatilisation is the major route of removal of 1,2,4- trimethylbenzene from soils; although, biodegradation may also occur Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations
Water - Because of 1,2,4-trimethylbenzene's water solubility and its vapor pressure of 2.03 mm Hg, the chemical will rapidly volatilise from surface waters Biodegradation of 1,2,4-trimethylbenzene occurred with inoculums from both seawater and ground water Various strains of Pseudomonas can biodegrade 1,2,4-trimethylbenzene.
Biota - The estimated bioconcentration factor (439) and high volatility of 1,2,4-trimethylbenzene indicates that bioaccumulation of the chemical will not be significant
Ecotoxicity:
Fish LC50 (96 h): fathead minnow 7.72 mg/l
No stress was observed in Oncorhynchus mykiss (rainbow trout, fingerling) or Petromyzon marinus (sea lamprey, larvae) at 5 mg/L for 24 hours
Daphnia magna EC50 (48 h): 3.61 mg/l
Cancer magister (dungeness crab) LC50 996 h): 5.1 mg/l
1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms; acute toxicity values fall within the range of greater than 1 mg/L and 100 mg/L. LC50 values for specific aquatic organisms range from approximately 5 to 8 mg/L which is orders of magnitude greater than any measured concentration in seawater (0.002 - 0.54 microgram/L) The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.
Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms.
When released to dry soil most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. Environmental processes may also be important in changing solubilities.
Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects.
A metal ion is considered infinitely persistent because it cannot degrade further.
The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.
The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.
Environmental processes may enhance bioavailability.
For aromatic hydrocarbons:
Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (Palaemonetes pugio) and brown shrimp (Penaeus aztecus) was dimethylnaphthalenes > methylnaphthalenes >naphthalenes.
Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene. The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.
Anthracene is a phototoxic PAH . UV light greatly increases the toxicity of anthracene to bluegill sunfish. . Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.
Volatile furandiones and aldehydes are significant atmospheric oxidation products of aromatic compounds. Highly acidic dicarboxylic acids produced by the reactions between furandiones and water were shown to rapidly acidify an aqueous phase
For silver and its compounds:
Environmental fate:
Silver is a rare but naturally occurring metal, often found deposited as a mineral ore in association with other elements. Emissions from smelting operations, manufacture and disposal of certain photographic and electrical supplies, coal combustion, and cloud seeding are some of the anthropogenic sources of silver in the biosphere. The global biogeochemical movements of silver are characterized by releases to the atmosphere, water, and land by natural and anthropogenic sources, long-range transport of fine particles in the atmosphere, wet and dry deposition, and sorption to soils and sediments.
In general, accumulation of silver by terrestrial plants from soils is low, even if the soil is amended with silver-containing sewage sludge or the plants are grown on tailings from silver

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mines, where silver accumulates mainly in the root systems.
The ability to accumulate dissolved silver varies widely between species. Some reported bioconcentration factors for marine organisms (calculated as milligrams of silver per kilogram fresh weight organism divided by milligrams of silver per litre of medium) are 210 in diatoms, 240 in brown algae, 330 in mussels, 2300 in scallops, and 18 700 in oysters, whereas bioconcentration factors for freshwater organisms have been reported to range from negligible in bluegills (*Lepomis macrochirus*) to 60 in daphnids; these values represent uptake of bioavailable silver in laboratory experiments. Laboratory studies with the less toxic silver compounds, such as silver sulfide and silver chloride, reveal that accumulation of silver does not necessarily lead to adverse effects. At concentrations normally encountered in the environment, food-chain biomagnification of silver in aquatic systems is unlikely. Elevated silver concentrations in biota occur in the vicinities of sewage outfalls, electroplating plants, mine waste sites, and silver iodide-seeded areas. Maximum concentrations recorded in field collections, in milligrams total silver per kilogram dry weight (tissue), were 1.5 in marine mammals (liver) (except Alaskan beluga whales *Delphinapterus leucas*, which had concentrations 2 orders of magnitude higher than those of other marine mammals), 6 in fish (bone), 14 in plants (whole), 30 in annelid worms (whole), 44 in birds (liver), 110 in mushrooms (whole), 185 in bivalve molluscs (soft parts), and 320 in gastropods (whole).
Ecotoxicity:
In general, silver ion was less toxic to freshwater aquatic organisms under conditions of low dissolved silver ion concentration and increasing water pH, hardness, sulfides, and dissolved and particulate organic loadings; under static test conditions, compared with flow-through regimens; and when animals were adequately nourished instead of being starved. Silver ions are very toxic to microorganisms. However, there is generally no strong inhibitory effect on microbial activity in sewage treatment plants because of reduced bioavailability due to rapid complexation and adsorption. Free silver ion was lethal to representative species of sensitive aquatic plants, invertebrates, and teleosts at nominal water concentrations of 1-5 ug/litre. Adverse effects occur on development of trout at concentrations as low as 0.17 ug/litre and on phytoplankton species composition and succession at 0.3-0.6 ug/litre.

A knowledge of the speciation of silver and its consequent bioavailability is crucial to understanding the potential risk of the metal. Measurement of free ionic silver is the only direct method that can be used to assess the likely effects of the metal on organisms. Speciation models can be used to assess the likely proportion of the total silver measured that is bioavailable to organisms. Unlike some other metals, background freshwater concentrations in pristine and most urban areas are well below concentrations causing toxic effects. Levels in most industrialized areas border on the effect concentration, assuming that conditions favour bioavailability. On the basis of available toxicity test results, it is unlikely that bioavailable free silver ions would ever be at sufficiently high concentrations to cause toxicity in marine environments.
No data were found on effects of silver on wild birds or mammals. Silver was harmful to poultry (tested as silver nitrate) at concentrations as low as 100 mg total silver/litre in drinking-water or 200 mg total silver/kg in diets. Sensitive laboratory mammals were adversely affected at total silver concentrations (added as silver nitrate) as low as 250 ug/litre in drinking-water (brain histopathology), 6 mg/kg in diet (high accumulations in kidneys and liver), or 13.9 mg/kg body weight (lethality).

Silver and Silver Compounds; Concise International Chemical Assessment Document (CICAD) 44 IPCS InChem (WHO)
'The transport of silver through estuarine and coastal marine systems is dependent on biological uptake and incorporation. Uptake by phytoplankton is rapid, in proportion to silver concentration and inversely proportional to salinity. In contrast to studies performed with other toxic metals, sliver availability appears to be controlled by both the free silver ion concentration and the concentration of other silver complexes. Silver incorporated by phytoplankton is not lost as salinity increase; as a result silver associated with cellular material is largely retained within the estuary. Phytoplankton exhibit a variable sensitivity to silver. Sensitive species exhibit a marked delay in the onset of growth in response to silver at low concentrations, even though maximum growth rates are similar to controls. A delay in the onset of growth reduces the ability of a population to respond to short-term favourable conditions and to succeed within th community.
James G. Saunders and George R Abbe: Aquatic Toxicology and Environmental Fate; ASTM STP 1007, 1989, pp 5-18
For glycol ethers:
Environmental fate:
Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).
Ecotoxicity:
Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are 'practically non-toxic' to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.
Glycols exert a high oxygen demand for decomposition and once released to the environments cause the death of aquatic organisms if dissolved oxygen is depleted.
For ketones:
Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds
Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrateThe higher molecular weight ketones do no form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions
Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (*e.g.,* ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable.
Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.
for acetone:
log Kow: -0.24
Half-life (hr) air: 312-1896
Half-life (hr) H2O surface water: 20
Henry's atm m3 /mol: 3.67E-05
BOD 5: 0.31-1.76,46-55%
COD: 1.12-2.07
ThOD: 2.2
BCF: 0.69
Environmental fate:
Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water
In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.
Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.
Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.
Acetone does not concentrate in the food chain.
Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period
Drinking Water Standard: none available.
Soil Guidelines: none available.
Air Quality Standards: none available.
Ecotoxicity:
Testing shows that acetone exhibits a low order of toxicity
Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l
Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l
Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l
Aquatic invertebrate 2100 - 16700 mg/l
Aquatic plant NOEC: 5400-7500 mg/l
Daphnia magna chronic NOEC 1660 mg/l
Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephestia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m3. The LT50 values for the eggs were 30-50% lower

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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.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
dimethyl carbonate	HIGH	HIGH
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
methyl isobutyl ketone	HIGH (Half-life = 7001 days)	LOW (Half-life = 1.9 days)
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
cumene	HIGH	HIGH

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
dimethyl carbonate	LOW (LogKOW = 0.2336)
acetone	LOW (BCF = 0.69)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
methyl isobutyl ketone	LOW (LogKOW = 1.31)
1,2,4-trimethyl benzene	LOW (BCF = 275)
cumene	LOW (BCF = 35.5)

12.4. Mobility in soil

Ingredient	Mobility
dimethyl carbonate	LOW (KOC = 8.254)
acetone	HIGH (KOC = 1.981)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
methyl isobutyl ketone	LOW (KOC = 10.91)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
cumene	LOW (KOC = 817.2)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗

PBT Criteria fulfilled?	No
vPvB	No

12.6. Endocrine Disruption Properties

The evidence linking adverse effects to endocrine disruptors is more compelling in the environment than it is in humans. Endocrine distruptors profoundly alter reproductive physiology of ecosystems and ultimately impact entire populations. Some endocrine-disrupting chemicals are slow to break-down in the environment. That characteristic makes them potentially hazardous over long periods of time. Some well established adverse effects of endocrine disruptors in various wildlife species include; eggshell-thinning, displayed of characteristics of the opposite sex and impaired reproductive development. Other adverse changes in wildlife species that have been suggested, but not proven include; reproductive abnormalities, immune dysfunction and skeletal deformaties.

12.7. Other adverse effects

Not Available

SECTION 13 Disposal considerations

13.1. 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.

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	▸ **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.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 Transport information

Labels Required

	 Limited quantity: 842UR-12ML, 842UR-150ML, 842UR-850ML

Land transport (ADR-RID)

14.1. **UN number**	1993
14.2. **UN proper shipping name**	FLAMMABLE LIQUID, N.O.S. (vapour pressure at 50 °C not more than 110 kPa) (contains dimethyl carbonate and acetone); FLAMMABLE LIQUID, N.O.S. (vapour pressure at 50 °C more than 110 kPa) (contains dimethyl carbonate and acetone)
14.3. **Transport hazard class(es)**	Class: 3 Subrisk: Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Hazard identification (Kemler): 33 Classification code: F1 Hazard Label: 3 Special provisions: 274 601 640C; 274 601 640D Limited quantity: 1 L Tunnel Restriction Code: 2 (D/E)

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	1993
14.2. **UN proper shipping name**	Flammable liquid, n.o.s. * (contains dimethyl carbonate and acetone)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3H
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A3 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L Passenger and Cargo Packing Instructions: 353 Passenger and Cargo Maximum Qty / Pack: 5 L Passenger and Cargo Limited Quantity Packing Instructions: Y341 Passenger and Cargo Limited Maximum Qty / Pack: 1 L

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	1993
14.2. **UN proper shipping name**	FLAMMABLE LIQUID, N.O.S. (contains dimethyl carbonate and acetone)
14.3. **Transport hazard class(es)**	IMDG Class: 3 IMDG Subrisk: Not Applicable
14.4. **Packing group**	II

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14.5. **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-E , S-E Special provisions: 274 Limited Quantities: 1 L

Inland waterways transport (ADN)

14.1. **UN number**	1993
14.2. **UN proper shipping name**	FLAMMABLE LIQUID, N.O.S. (vapour pressure at 50 °C more than 110 kPa) (contains dimethyl carbonate and acetone); FLAMMABLE LIQUID, N.O.S. (vapour pressure at 50 °C not more than 110 kPa) (contains dimethyl carbonate and acetone)
14.3. **Transport hazard class(es)**	3 Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Classification code: F1 Special provisions: 274; 601; 640C|274; 601; 640D Limited quantity: 1 L Equipment required: PP, EX, A Fire cones number: 1

14.7. Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

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

Product name	Group
silver	Not Available
dimethyl carbonate	Not Available
acetone	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
methyl isobutyl ketone	Not Available
hexamethylene diisocyanate homopolymer, MEK-oxime blocked	Not Available
naphtha petroleum, light aromatic solvent	Not Available
1,2,4-trimethyl benzene	Not Available
cumene	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
silver	Not Available
dimethyl carbonate	Not Available
acetone	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
methyl isobutyl ketone	Not Available
hexamethylene diisocyanate homopolymer, MEK-oxime blocked	Not Available
naphtha petroleum, light aromatic solvent	Not Available
1,2,4-trimethyl benzene	Not Available
cumene	Not Available

SECTION 15 Regulatory information

15.1. Safety, health and environmental regulations / legislation specific for the substance or mixture

silver is found on the following regulatory lists

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

Europe EC Inventory

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

dimethyl carbonate is found on the following regulatory lists

Continued...

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

acetone is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

propylene glycol monomethyl ether acetate, alpha-isomer is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

methyl isobutyl ketone is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

hexamethylene diisocyanate homopolymer, MEK-oxime blocked is found on the following regulatory lists

Europe EC Inventory
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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

Chemical Footprint Project - Chemicals of High Concern List
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 2) Carcinogens: Category 1 B
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 4) Germ cell mutagens: Category 1 B
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

1,2,4-trimethyl benzene is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

cumene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (silver; dimethyl carbonate; acetone; propylene glycol monomethyl ether acetate, alpha-isomer; methyl isobutyl ketone; hexamethylene diisocyanate homopolymer, MEK-oxime blocked; naphtha petroleum, light aromatic solvent; 1,2,4-trimethyl benzene; cumene)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (silver; hexamethylene diisocyanate homopolymer, MEK-oxime blocked)
Korea - KECI	Yes
New Zealand - NZIoC	Yes

Continued...

National Inventory	Status
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (hexamethylene diisocyanate homopolymer, MEK-oxime blocked)
Vietnam - NCI	Yes
Russia - FBEPH	No (hexamethylene diisocyanate homopolymer, MEK-oxime blocked)
Legend:	*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	24/02/2022
Initial Date	10/09/2018

Full text Risk and Hazard codes

H226	Flammable liquid and vapour.
H303	May be harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H305	May be harmful if swallowed and enters airways.
H315	Causes skin irritation.
H316	Causes mild skin irritation.
H320	Causes eye irritation.
H332	Harmful if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H411	Toxic to aquatic life with long lasting effects.

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.
For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:
EN 166 Personal eye-protection
EN 340 Protective clothing
EN 374 Protective gloves against chemicals and micro-organisms
EN 13832 Footwear protecting against chemicals
EN 133 Respiratory protective devices

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

Reason for Change

A-2.00 - Added UFI number and modifications to the safety data sheet

end of SDS