



839 Super Shield Graphite Conductive Coating

MG Chemicals (Head Office)

Chemwatch Hazard Alert Code: 3

Version No: 3.5

Safety Data Sheet (Conforms to Regulations (EC) No 2015/830)

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L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	839 Super Shield Graphite Conductive Coating
Synonyms	SDS Code 839-Liquid; Related Part # 839-5ML, 839-900ML, 839-1G
Proper shipping name	PAINT or PAINT RELATED MATERIAL
Other means of identification	Not Available

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

Relevant identified uses	A graphite filled coating that makes surfaces electrically conductive.
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals (Head Office)	MG Chemicals UK Limited
Address	9347-193 Street, Surrey V4N 4E7 British Columbia Canada	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom
Telephone	+1-604-888-3084	+44 1663 362888
Fax	+1-604-888-7754	Not Available
Website	www.mgchemicals.com	Not Available
Email	info@mgchemicals.com	sales@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	Not Available	CHEMTREC
Emergency telephone numbers	Not Available	+(44)-870-8200418
Other emergency telephone numbers	Not Available	+(1) 703-527-3887

SECTION 2 HAZARDS IDENTIFICATION

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, STOT - SE (Narcosis) Category 3, Carcinogen Category 2, Reproductive Toxicity Category 2, Reproductive Toxicity Category 2, STOT - RE Category 2, Flammable Liquid Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

2.2. Label elements

CLP label elements	  
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SIGNAL WORD **DANGER**

Hazard statement(s)

Continued...

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H304	May be fatal if swallowed and enters airways
H315	Causes skin irritation
H319	Causes serious eye irritation
H336	May cause drowsiness or dizziness
H351	Suspected of causing cancer
H361	Suspected of damaging fertility or the unborn child
H361d	Suspected of damaging the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure
H225	Highly flammable liquid and vapour

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.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.

Precautionary statement(s) Response

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

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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2.3. Other hazards

	May produce discomfort of the eyes and respiratory tract*.
	Cumulative effects may result following exposure*.

REACH - Art.57-59: The mixture does not contain Substances of Very High Concern (SVHC) at the SDS print date.

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	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]

Continued...

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3.Index No 4.REACH No			
1.108-88-3 2.203-625-9 3.601-021-00-3 4.01-2119471310-51-XXXX	10-30	<u>toluene</u>	Flam. Liq. 2, Repr. 2, Asp. Tox. 1, STOT RE 2 *, Skin Irrit. 2, STOT SE 3; H225, H361d ***, H304, H373 **, H315, H336 [3]
1.7782-42-5 2.231-955-3 3.Not Available 4.01-2119486977-12-XXXX, 01-2119875125-36-XXXX	10-30	<u>graphite, natural</u>	STOT - RE Category 2; H373 [1]
1.110-43-0 2.203-767-1 3.606-024-00-3 4.01-2119902391-49-XXXX	5-10	<u>amyl methyl ketone</u>	Flam. Liq. 3, Acute Tox. 4 *, Acute Tox. 4 *; H226, H332, H302 [3]
1.67-64-1 2.200-662-2 3.606-001-00-8 4.01-2119498062-37-XXXX, 01-2119471330-49-XXXX	5-10	<u>acetone</u>	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3; H225, H319, H336, EUH066 [3]
1.64-17-5 2.200-578-6 3.603-002-00-5 4.01-2119457610-43-XXXX	5-10	<u>ethanol</u>	Flam. Liq. 2; H225 [3]
1.110-19-0 2.203-745-1 3.607-026-00-7 4.01-2119488971-22-XXXX	5-10	<u>isobutyl acetate</u>	Flam. Liq. 2; H225, EUH066 [3]
1.108-65-6 2.203-603-9, 283-152-2 3.607-195-00-7 4.01-2119475791-29-XXXX	1-5	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u>	Flam. Liq. 3; H226 [3]
1.141-78-6 2.205-500-4 3.607-022-00-5 4.01-2119475103-46-XXXX	1-5	<u>ethyl acetate</u>	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3; H225, H319, H336, EUH066 [3]
1.1333-86-4 2.215-609-9 3.Not Available 4.01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX	0.5-1.5	<u>carbon black</u>	Carcinogen Category 2; H351 [1]

Legend: 1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I; 3. Classification drawn from EC Directive 1272/2008 - Annex VI
4. Classification drawn from C&L

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

General	<ul style="list-style-type: none"> ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus. ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary. <p>If this product comes in contact with eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with water. ▶ If irritation continues, seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. <p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ 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.
Eye Contact	<p>If this product comes in contact with eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with water. ▶ If irritation continues, seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.

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Ingestion

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

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.

Treat symptomatically.

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*

Following acute or short term repeated exposures to toluene:

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

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

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

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

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

- ▶ Sand, dry powder extinguishers or other inerts should be used to smother dust fires.

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At temperatures above 1500 C, carbon, graphite or graphene reacts with substances containing oxygen, including water and carbon dioxide. In case of intensely hot fires sand should be used to cover and isolate these materials.

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

- ▶ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles.
- ▶ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse.
- ▶ 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.
- ▶ **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

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

Combustion products include: carbon dioxide (CO₂) hydrogen fluoride, silicon dioxide (SiO₂) other pyrolysis products typical of burning organic material

Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions. A fire in bulk finely divided carbon may not be obviously visible unless the material is disturbed and sparks appear. A straw broom may be useful to produce the disturbance. Explosion and Ignition Behaviour of Carbon Black with Air

Lower Limit for Explosion:	50 g/m ³ (carbon black in air)
Maximum Explosion Pressure:	10 bar
Maximum Rate of Pressure Rise:	30-100 bar/sec
Minimum Ignition Temperature:	315 deg. C.
Ignition Energy:	>1 kJ
Glow Temperature:	500 deg. C. (approx.)

Notes on Test Methods: Tests 1, 2 and 3 were conducted by Bergwerkeschaftliche Versuchsstrecke, Dortmunde-Derne, using a 1 m³ vessel with two chemical igniters having an intensity of 5000 W.S. Tests 1 and 2 results are confirmed by information in the Handbook of Powder Technology, Vol. 4 (P. Field) In Test 4, a modified Godbert-Greenwald furnace was used. See U.S. Bureau of Mines, Report 5624, 1960, p.5, "Lab Equipment and Test Procedures". Test 5 used a 1 m³ vessel with chemical igniters of variable intensity. Test 6 was conducted in a laboratory oven. Active glowing appeared after 3 minutes exposure. (European Committee for Biological Effects of Carbon Black) (2/84)

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

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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

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

- ▶ 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.

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

Graphite:

- ▶ is a good conductor of electricity; avoid contact with electrical circuitry.
- ▶ is a highly lubricious material and may present a slip hazard if spilled on pedestrian surfaces.

NOTE:

- ▶ Wet, activated carbon removes oxygen from the air thus producing a severe hazard to workers inside carbon vessels and in enclosed or confined spaces where activated carbons might accumulate.
- ▶ Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply.
- ▶ Containers, even those that have been emptied, may contain explosive vapours.
- ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.

Contains low boiling substance:

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

- ▶ Check for bulging containers.
- ▶ Vent periodically
- ▶ Always release caps or seals slowly to ensure slow dissipation of vapours
- ▶ **DO NOT allow clothing wet with material to stay in contact with skin**
- ▶ Electrostatic discharge may be generated during pumping - this may result in fire.
- ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment.
- ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (≤ 1 m/sec until fill pipe submerged to twice its diameter, then ≤ 7 m/sec).
- ▶ Avoid splash filling.
- ▶ Do NOT use compressed air for filling discharging or handling operations.
- ▶ Avoid all personal contact, including inhalation.
- ▶ Wear protective clothing when risk of exposure occurs.
- ▶ Use in a well-ventilated area.
- ▶ Prevent concentration in hollows and sumps.
- ▶ **DO NOT enter confined spaces until atmosphere has been checked.**
- ▶ Avoid smoking, naked lights, heat or ignition sources.
- ▶ When handling, **DO NOT eat, drink or smoke.**
- ▶ Vapour may ignite on pumping or pouring due to static electricity.
- ▶ **DO NOT use plastic buckets.**
- ▶ Earth and secure metal containers when dispensing or pouring product.
- ▶ Use spark-free tools when handling.
- ▶ Avoid contact with incompatible materials.
- ▶ Keep containers securely sealed.

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	<ul style="list-style-type: none"> ▶ 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 MSDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Fire and explosion protection	See section 5
Other information	<p>Carbon and charcoal may be stabilised for storage and transport, without moistening, by treatment with hot air at 50 deg. C.. Use of oxygen-impermeable bags to limit oxygen and moisture uptake has been proposed. Surface contamination with oxygenated volatiles may generate a heat of reaction (spontaneous heating). Should stored product reach 110 deg. C., stacked bags should be pulled apart with each bag separated by an air space to permit cooling away from other combustible materials.</p> <ul style="list-style-type: none"> ▶ 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 MSDS.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ 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	<p>Heptanones:</p> <ul style="list-style-type: none"> ▶ react violently with strong oxidisers, aldehydes, nitric acid, perchloric acid ▶ form a variety of unstable peroxides following reaction with hydrogen peroxide ▶ are incompatible with aliphatic amines, aldehydes, strong bases <p>Toluene:</p> <ul style="list-style-type: none"> ▶ reacts violently with strong oxidisers, bromine, bromine trifluoride, chlorine, hydrochloric acid/ sulfuric acid mixture, 1,3-dichloro-5,5-dimethyl-2,4-imidazolidindione, dinitrogen tetroxide, fluorine, concentrated nitric acid, nitrogen dioxide, silver chloride, sulfur dichloride, uranium fluoride, vinyl acetate ▶ forms explosive mixtures with strong acids, strong oxidisers, silver perchlorate, tetranitromethane ▶ is incompatible with bis-toluenediazo oxide ▶ attacks some plastics, rubber and coatings ▶ may generate electrostatic charges, due to low conductivity, on flow or agitation. ▶ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates. <p>For alkyl aromatics:</p> <p>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.</p> <ul style="list-style-type: none"> ▶ 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 NO_x - these may be components of photochemical smogs. <p>Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007</p> <ul style="list-style-type: none"> ▶ 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. ▶ 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. <p>Ketones in this group:</p> <ul style="list-style-type: none"> ▶ 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. <p>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).</p> <p>Propylene glycol monomethyl ether acetate:</p> <ul style="list-style-type: none"> ▶ 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 sodium peroxide, uranium fluoride ▶ is incompatible with sulfuric acid, nitric acid, caustics, aliphatic amines, isocyanates, boranes

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For carbon powders:

- ▶ Avoid oxidising agents, reducing agents.
- ▶ Reaction with finely divided metals, bromates, chlorates, chloramine monoxide, dichlorine oxide, iodates, metal nitrates, oxygen difluoride, peroxyformic acid, peroxyfuroic acid and trioxigen difluoride may result in an exotherm with ignition or explosion. Less active forms of carbon will ignite or explode on suitably intimate contact with oxygen, oxides, peroxides, oxosalts, halogens, interhalogens and other oxidising species.
- ▶ Explosive reaction with ammonium nitrate, ammonium perchlorate, calcium hypochlorite and iodine pentoxide may occur following heating. Carbon may react violently with nitric acid and may be explosively reactive with nitrogen trifluoride at reduced temperatures. In the presence of nitrogen oxide, incandescence and ignition may occur. Finely divided or highly porous forms of carbon, exhibiting a high surface area to mass (up to 2000 m²/g) may function as unusually active fuels possessing both adsorptive and catalytic properties which accelerate the release of energy in the presence of oxidising substances. Dry metal-impregnated charcoal catalysts may generate sufficient static, during handling, to cause ignition.
- ▶ Graphite in contact with liquid potassium, rubidium or caesium at 300 deg. C. produces intercalation compounds (C8M) which ignite in air and may react explosively with water. The fusion of powdered diamond and potassium hydroxide may produce explosive decomposition.
- ▶ Activated carbon, when exposed to air, represents a potential fire hazard due to a high surface area and adsorptive capacity. Freshly prepared material may ignite spontaneously in the presence of air especially at high humidity. Spontaneous combustion in air may occur at 90-100 deg. C. The presence of moisture in air facilitates the ignition. Drying oils and oxidising oils promote spontaneous heating and ignition; contamination with these must be avoided. Unsaturated drying oils (linseed oil etc.) may ignite following adsorption owing to an enormous increase in the surface area of oil exposed to air; the rate of oxidation may also be catalysed by metallic impurities in the carbon. A similar, but slower effect occurs on fibrous materials such as cotton waste. Spontaneous heating of activated carbon is related to the composition and method of preparation of the activated carbon. Free radicals, present in charcoal, are responsible for autoignition. Self-heating and autoignition may also result from adsorption of various vapours and gases (especially oxygen). For example, activated carbon auto-ignites in flowing air at 452-518 deg. C.; when the base, triethylenediamine, is adsorbed on the carbon (5%) the autoignition temperature is reduced to 230-260 deg. C. An exotherm is produced at 230-260 deg. C., at high flow rates of air, although ignition did not occur until 500 deg. C.. Mixtures of sodium borohydride with activated carbons, in air, promote the oxidation of sodium borohydride, producing a self-heating reaction that may result in the ignition of charcoal and in the production of hydrogen through thermal decomposition of the borohydride.

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	toluene	Toulene	191 mg/m ³ / 50 ppm	384 mg/m ³ / 100 ppm	Not Available	Sk
European Union (EU) Commission Directive 2006/15/EC establishing a second list of indicative occupational exposure limit values (IOELVs)	toluene	Toluene	192 mg/m ³ / 50 ppm	384 mg/m ³ / 100 ppm	Not Available	skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	toluene	Toluene	192 mg/m ³ / 50 ppm	384 mg/m ³ / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	amyl methyl ketone	Heptan-2-one	237 mg/m ³ / 50 ppm	475 mg/m ³ / 100 ppm	Not Available	Sk
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	amyl methyl ketone	Heptan-2-one	238 mg/m ³ / 50 ppm	475 mg/m ³ / 100 ppm	Not Available	Skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	amyl methyl ketone	Heptan-2-one	238 mg/m ³ / 50 ppm	475 mg/m ³ / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	acetone	Acetone	1210 mg/m ³ / 500 ppm	3620 mg/m ³ / 1500 ppm	Not Available	Not Available
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	acetone	Acetone	1 210 mg/m ³ / 500 ppm	Not Available	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	acetone	Acetone	1210 mg/m ³ / 500 ppm	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	ethanol	Ethanol	1920 mg/m ³ / 1000 ppm	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	isobutyl acetate	Isobutyl acetate	724 mg/m ³ / 150 ppm	903 mg/m ³ / 187 ppm	Not Available	Not Available

Continued...

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UK Workplace Exposure Limits (WELs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl acetate	274 mg/m ³ / 50 ppm	548 mg/m ³ / 100 ppm	Not Available	Sk
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	propylene glycol monomethyl ether acetate, alpha-isomer	2-Methoxy-1-methylethylacetate	275 mg/m ³ / 50 ppm	550 mg/m ³ / 100 ppm	Not Available	Skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl-2-acetate	275 mg/m ³ / 50 ppm	550 mg/m ³ / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	ethyl acetate	Ethyl acetate	200 ppm	400 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m ³	7 mg/m ³	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
toluene	Toluene	Not Available	Not Available	Not Available
graphite, natural	Graphite; (Mineral carbon)	2 mg/m ³	2 mg/m ³	95 mg/m ³
amyl methyl ketone	Methyl n-amyl ketone	50 ppm	50 ppm	4000 ppm
acetone	Acetone	Not Available	Not Available	Not Available
ethanol	Ethyl alcohol; (Ethanol)	Not Available	Not Available	Not Available
isobutyl acetate	Isobutyl acetate	450 ppm	1300 ppm	7500 ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
ethyl acetate	Ethyl acetate	400 ppm	400 ppm	10000 ppm
carbon black	Carbon black	9 mg/m ³	99 mg/m ³	590 mg/m ³

Ingredient	Original IDLH	Revised IDLH
toluene	2,000 ppm	500 ppm
graphite, natural	N.E. mg/m ³ / N.E. ppm	1,250 mg/m ³
amyl methyl ketone	4,000 ppm	800 ppm
acetone	20,000 ppm	2,500 [LEL] ppm
ethanol	15,000 ppm	3,300 [LEL] ppm
isobutyl acetate	7,500 ppm	1,300 [LEL] ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
ethyl acetate	10,000 ppm	2,000 [LEL] ppm
carbon black	N.E. mg/m ³ / N.E. ppm	1,750 mg/m ³

MATERIAL DATA

for isobutyl acetate:

Odour Threshold Value: 0.40-0.44 ppm (recognition)

The TLV-TWA is identical with that of n-butyl acetate and is thought to minimise the potential for ocular and upper respiratory tract irritation.

For ethanol:

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

Eye and respiratory tract irritation do not appear to occur at exposure levels of less than 5000 ppm and the TLV-TWA is thought to provide an adequate margin of safety against such effects.

Experiments in man show that inhalation of 1000 ppm caused slight symptoms of poisoning and 5000 ppm caused strong stupor and morbid sleepiness. Subjects exposed to 5000 ppm to 10000 ppm experienced smarting of the eyes and nose and coughing. Symptoms disappeared within minutes. Inhalation also causes local irritating effects to the eyes and upper respiratory tract, headaches, sensation of heat intraocular tension, stupor, fatigue and a need to sleep. At 15000 ppm there was continuous lachrymation and coughing.

For graphite:

Graphite pneumoconiosis resembles coal workers' pneumoconiosis. Data indicate that the higher the crystalline silica content of graphite the more likely the disease will increase in severity. The presence of anthracite coal in the production of some synthetic grades of graphite appears to make arbitrary the use of the term, "synthetic", "artificial" or "natural".

The TLV-TWA for carbon black is recommended to minimise complaints of excessive dirtiness and applies only to commercially produced carbon blacks or to soots derived from combustion sources containing absorbed polycyclic aromatic hydrocarbons (PAHs). When PAHs are present in carbon black (measured as the cyclohexane-extractable fraction) NIOSH has established a REL-TWA of 0.1 mg/m³ and considers the material to be an occupational carcinogen.

The NIOSH REL-TWA was "selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs".

This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.

For ethyl acetate:

Odour Threshold Value: 6.4-50 ppm (detection), 13.3-75 ppm (recognition)

The TLV-TWA provides a significant margin of safety from the standpoint of adverse health effects. Unacclimated subjects found the odour objectionably strong at 200 ppm. Mild nose, eye and throat irritation was experienced at 400 ppm. Workers exposed regularly at concentrations ranging from 375 ppm to 1500 ppm for several months showed no unusual signs or symptoms.

Odour Safety Factor(OSF)

OSF=51 (ETHYL ACETATE)

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.

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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 concentration of dust, for application of respirable dust limits, is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative log-normal function with a median aerodynamic diameter of 4.0 μm (+) 0.3 μm and with a geometric standard deviation of 1.5 μm (+) 0.1 μm , i.e., generally less than 5 μm .

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 sternbral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

Because the margin of safety of the quartz TLV is not known with certainty and given the associated link between silicosis and lung cancer it is recommended that quartz concentrations be maintained as far below the TLV as prudent practices will allow.

Exposure to respirable crystalline silicas (RCS) represents a significant hazard to workers, particularly those employed in the construction industry where respirable dusts of cement and concrete are common. Cutting, grinding and other high speed processes, involving their finished products, may further result in dusty atmospheres. Bricks are also a potential source of RCSs under such circumstances.

It is estimated that half of the occupations, involved in construction work, are exposed to levels of RCSs, higher than the current allowable limits. Beaudry et al: Journal of Occupational and Environmental Hygiene 10: 71-77; 2013

For toluene:

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

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

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

Odour Safety Factor(OSF)

OSF=17 (TOLUENE)

For amyl methyl ketone:

Odour Threshold Value: 0.18 ppm (detection)

The TLV-TWA is well below the highest level of vapour (1025 ppm) reported to be associated with adverse effects in animals including dermal irritation.

Odour Safety Factor (OSF)

OSF=1.4E2 (2-HEPTANONE)

8.2. Exposure controls

Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air.

Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde]

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.

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:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
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 (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to

8.2.1. Appropriate engineering controls

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	distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
8.2.2. Personal protection	
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> ▶ frequency and duration of contact, ▶ chemical resistance of glove material, ▶ glove thickness and ▶ dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> ▶ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ▶ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ▶ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. ▶ Contaminated gloves should be replaced. <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower. <p>Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.</p> <p>For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).</p> <p>Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot and shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.</p>
Thermal hazards	Not Available

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:

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Material	CPI
PE/EVAL/PE	A
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVA	C
PVC	C

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 / Class 1	-	AX-PAPR-AUS / Class 1
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	AX-3	-
100+ x ES	-	Air-line**	-

* - Continuous-flow; ** - Continuous-flow or positive pressure demand

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)

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PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/CHLOROBUTYL	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.

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	Note that all of the monopropylene glycol ethers may exist in two isomeric forms, alpha or beta. The alpha form, which is thermodynamically favored during synthesis, consists of a secondary alcohol configuration. The beta form consists of a primary alcohol. The two isomeric forms are shown above. The di- and tripropylene glycol ethers may form up to 4 and 8 isomeric forms, respectively. Even so, all isomers exhibit either the "alpha" or "beta" configuration, existing as secondary or primary alcohols, respectively. The distribution of isomeric forms for the di- and tripropylene glycols, as with the mono-PGEs, also results in predominantly the alpha form (i.e., a secondary alcohol). It should be noted that only the alpha isomer and isomeric mixtures (consisting predominantly of the alpha isomer) are produced commercially; the purified beta isomer is not produced at this time.		
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	Dark Grey		

Physical state	Liquid	Relative density (Water = 1)	0.996
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>315
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	>34
Initial boiling point and boiling range (°C)	>56	Molecular weight (g/mol)	Not Available
Flash point (°C)	-18	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	10	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

9.2. Other information

	Not Available
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SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2.Chemical stability	<ul style="list-style-type: none"> ▶ 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

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10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 TOXICOLOGICAL INFORMATION

11.1. Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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</p> <p>The most common signs of inhalation overexposure to ethanol, in animals, include ataxia, incoordination and drowsiness for those surviving narcosis. The narcotic dose for rats, after 2 hours of exposure, is 19260 ppm.</p> <p>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.</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p> <p>Although carbon itself has no toxic action, associated impurities may be toxic. Iodine is often found as an impurity and air-borne carbon dusts, as a result, may produce irritation of the mucous membranes, the eyes, and skin. Symptoms of exposure may include coughing, irritation of the nose and throat and burning of the eyes.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Effects on lungs are significantly enhanced in the presence of respirable particles. Overexposure to respirable dust may produce wheezing, coughing and breathing difficulties leading to or symptomatic of impaired respiratory function.</p>								
Ingestion	<p>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.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Ingestion of ethanol may produce nausea, vomiting, gastrointestinal bleeding, abdominal pain and diarrhoea. Systemic effects:</p> <table border="1"> <thead> <tr> <th>Blood concentration:</th> <th>Effects:</th> </tr> </thead> <tbody> <tr> <td><1.5 g/l</td> <td>Mild: Impaired visual acuity, coordination and reaction time, emotional lability</td> </tr> <tr> <td>1.5-3.0 g/l</td> <td>Moderate: Slurred speech, confusion, ataxia, emotional lability, perceptual and sensation disturbances possible blackout spells, and incoordination with impaired objective performance in standardised tests. Possible diplopia, flushing, tachycardia, sweating and incontinence. Bradypnoea may occur early and tachypnoea may develop in cases of metabolic acidosis, hypoglycaemia and hypokalaemia. CNS depression may progress to coma.</td> </tr> <tr> <td>3-5 g/l</td> <td>Severe: Cold clammy skin, hypothermia and hypotension. Atrial fibrillation and atrioventricular block have been reported. Respiratory depression may occur, respiratory failure may follow serious intoxication, aspiration of vomitus may result in pneumonitis and pulmonary oedema. Convulsions due to severe hypoglycaemia may also occur Acute hepatitis may develop.</td> </tr> </tbody> </table> <p>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.</p> <p>Ingestion of finely divided carbon may produce gagging and constipation. Aspiration does not appear to be a concern as the material is generally regarded as inert and is often used as a food additive. Ingestion may produce a black stool.</p>	Blood concentration:	Effects:	<1.5 g/l	Mild: Impaired visual acuity, coordination and reaction time, emotional lability	1.5-3.0 g/l	Moderate: Slurred speech, confusion, ataxia, emotional lability, perceptual and sensation disturbances possible blackout spells, and incoordination with impaired objective performance in standardised tests. Possible diplopia, flushing, tachycardia, sweating and incontinence. Bradypnoea may occur early and tachypnoea may develop in cases of metabolic acidosis, hypoglycaemia and hypokalaemia. CNS depression may progress to coma.	3-5 g/l	Severe: Cold clammy skin, hypothermia and hypotension. Atrial fibrillation and atrioventricular block have been reported. Respiratory depression may occur, respiratory failure may follow serious intoxication, aspiration of vomitus may result in pneumonitis and pulmonary oedema. Convulsions due to severe hypoglycaemia may also occur Acute hepatitis may develop.
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Skin Contact	<p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry</p>								

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	<p>through wounds, lesions or abrasions. Toxic effects may result from skin absorption 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.</p>
Eye	<p>Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn). Direct contact of the eye with ethanol may cause immediate stinging and burning with reflex closure of the lid and tearing, transient injury of the corneal epithelium and hyperaemia of the conjunctiva. Foreign-body type discomfort may persist for up to 2 days but healing is usually spontaneous and complete.</p> <p>Symptoms of exposure by the eye to carbon particulates include irritation and a burning sensation. Following an industrial explosion, fine particles become embedded in the cornea and conjunctiva resulting in an inflammation which persisted for 2-3 weeks. Some particles remained permanently producing a punctate purplish-black discolouration. Undiluted propylene glycol monomethyl ether acetate (PGMEA) causes moderate discomfort, slight conjunctival redness and slight corneal injury in rabbits</p>
Chronic	<p>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. Harmful: danger of serious damage to health by prolonged exposure through inhalation. 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.</p> <p>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. Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents. Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size. Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1). (1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996</p> <p>Chronic symptoms produced by crystalline silicas included decreased vital lung capacity and chest infections. Lengthy exposure may cause silicosis a disabling form of pneumoconiosis which may lead to fibrosis, a scarring of the lining of the air sacs in the lung. Symptoms may appear 8 to 18 months after initial exposure. Smoking increases this risk. Classic silicosis is a chronic disease characterised by the formation of scattered, rounded or stellate silica-containing nodules of scar tissue in the lungs ranging from microscopic to 1.0 cm or more. The nodules isolate the inhaled silica particles and protect the surrounding normal and functioning tissue from continuing injury. Simple silicosis (in which the nodules are less than 1.0 cm in diameter) is generally asymptomatic but may be slowly progressive even in the absence of continuing exposure. Simple silicosis can develop in complicated silicoses (in which nodules are greater than 1.0 cm in diameter) and can produce disabilities including an associated tuberculous infection (which 50 years ago accounted for 75% of the deaths among silicotic workers). Crystalline silica deposited in the lungs causes epithelial and macrophage injury and activation. Crystalline silica translocates to the interstitium and the regional lymph nodes and cause the recruitment of inflammatory cells in a dose dependent manner. In humans, a large fraction of crystalline silica persists in the lungs. The question of potential carcinogenicity associated with chronic inhalation of crystalline silica remains equivocal with some studies supporting the proposition and others finding no significant association. The results of recent epidemiological studies suggest that lung cancer risk is elevated only in those patients with overt silicosis. A relatively large number of epidemiological studies have been undertaken and in some, increased risk gradients have been observed in relation to dose surrogates - cumulative exposure, duration of exposure, the presence of radiographically defined silicosis, and peak intensity exposure. Chronic inhalation in rats by single or repeated intratracheal instillation produced a significant increase in the incidences of adenocarcinomas and squamous cell carcinomas of the lung. Lifetime inhalation of crystalline silica (87% alpha-quartz) at 1 mg/m³ (74% respirable) by rats, produced an increase in animals with keratinising cystic squamous cell tumours, adenomas, adenocarcinomas, adenosquamous cell carcinomas, squamous cell carcinoma and nodular bronchiolar alveolar hyperplasia accompanied by extensive subpleural and peribronchiolar fibrosis, increased pulmonary collagen content, focal lipoproteinosis and macrophage infiltration. Thoracic and abdominal malignant lymphomas developed in rats after single intrapleural and intraperitoneal injection of suspensions of several types of quartz.</p> <p>Some studies show excess numbers of cases of scleroderma, connective tissue disorders, lupus, rheumatoid arthritis chronic kidney diseases, and end-stage kidney disease in workers</p> <p>NOTE: Some jurisdictions require health surveillance be conducted on workers occupationally exposed to silica, crystalline. Such surveillance should emphasise</p> <ul style="list-style-type: none"> ▶ demography, occupational and medical history and health advice ▶ standardised respiratory function tests such as FEV₁, FVC and FEV₁/FVC ▶ standardised respiratory function tests such as FV₁, FVC and FEV₁/FVC ▶ chest X-ray, full size PA view ▶ records of personal exposure <p>Prolonged or repeated inhalation of dust may result in pneumoconiosis (lung disease caused by inhalation dust). Graphite workers have reported symptoms of headaches, coughing, depression, low appetite, dyspnoea (difficult breathing) and black sputum. A number of studies indicate that graphitosis is a progressive and disabling disease and that the presence of crystalline silica and some silicates as graphite impurities have a pronounced synergistic effect. Workers suffering from graphite pneumoconiosis have generally worked in the industry for long periods, i.e. 10 years or more, although some cases have been reported after as little as four years. Data indicate the higher the crystalline silica content of graphite the greater is the severity of the pneumoconiosis. Pre-employment and periodic examinations should be directed towards detecting significant respiratory disease through chest X-rays and pulmonary function tests Chronic inhalation exposure of production workers has caused decreased pulmonary function and myocardial dystrophy. There is suggestive but inconclusive evidence that carbon black containing polyaromatic hydrocarbons (PAHs) has been responsible for induction of skin cancers in exposed workers. Long term inhalation of carbon black can cause cough, phlegm, tiredness, chest pain and headache. Dermal, mucosal, or inhalation exposure can cause irritation. Inhalation of carbon black by mice, rats and monkeys caused thickened alveolar walls, increased pulmonary collagen, right atrial and ventricular strain, hypertrophy of the right atrial and ventricular septum and increased heart weights. Although carbon black itself did not cause cancer in treated animals, carbon black containing polyaromatic hydrocarbons (PAHs) did cause cancer following chronic administration by all routes tested. Epidemiological studies of workers in the carbon black producing industries of North America and Western Europe show no significant health effect due to occupational exposure to carbon black. Several other studies provide conflicting evidence. Early studies in the former USSR and Eastern Europe report</p>

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respiratory diseases amongst workers exposed to carbon black, including bronchitis, pneumonia, emphysema and rhinitis. These studies are of questionable validity due to inadequate study design and methodology, lack of appropriate controls for cigarette smoking and other confounding factors such as concurrent exposure to carbon dioxide, coal oil and petroleum vapours. Moreover, review of these studies indicates that the concentrations of carbon black were greater than current occupational standards.

Carbon black may cause adverse pulmonary changes following prolonged or repeated inhalation of the dust; these include oral mucosal lesions, bronchitis and pneumoconiosis which may lead to lung tumours.

The body of evidence of carcinogenicity in animal studies comes from two chronic inhalation studies and two intratracheal instillation studies in rats, which showed significantly elevated rates of lung cancer in exposed animals. An inhalation study was tested on mice, but did not show significantly elevated rates of lung cancer in exposed animals. Epidemiologic data comes from three different cohort studies of carbon black production workers. Two studies, from the United Kingdom and Germany, with over 1,000 workers in each study group, showed elevated mortality from lung cancer in the carbon black workers. Another study of over 5,000 workers in the United States did not show elevated mortality from lung cancer in the carbon black workers. Newer findings of increased lung cancer mortality in an update from the UK study may suggest that carbon black could be a late-stage carcinogen. However, a more recent and larger study from Germany did not confirm this hypothesis that carbon black acts as a late-stage carcinogen.

In studies employing channel and furnace black, hamsters, mice, guinea pigs, rabbits and monkeys exposed to dusts for 7 hours/day, 5 days/week, at concentrations of 87.4 mg/m³ for channel black and 56.5 mg/m³ for furnace black, no malignancies were observed in any of the animals. Channel black had little if any absorbed polyaromatic hydrocarbons (PAHs) (as benzene extractables) whilst furnace black had 0.28%.

Several findings have strengthened the association between inflammation and cancer and between the particle surface area dose of carbon black and other poorly soluble low toxicity (PSLT) particles and the pulmonary inflammation response in mice and the proinflammatory effects in lung cells in vitro. Other evidence suggests that in addition to a cancer mechanism involving indirect genotoxicity through inflammation and oxidative stress, nanoparticles may act as direct carcinogens.

Carbon black appears to act like PSLT particles, which can elicit lung tumours in rats following prolonged exposure to sufficiently high concentrations of particles. Particle surface area dose was found to be most predictive of pulmonary inflammation and tumour response in rats when comparing the dose-response relationships for various types and sizes of PSLT including carbon black. Compared to fine PSLT, much lower concentrations of ultrafine PSLT (e.g. 2.5, 6.5 or 11.5 mg/m³ carbon black and ~10 mg/m³ ultrafine titanium dioxide) were associated with impaired clearance, persistent inflammation, and malignant lung tumours in chronic inhalation studies in rats. Most evidence suggests that carbon black and other PSLT-elicited lung tumours occurs through a secondary genotoxic mechanism, involving chronic inflammation and oxidative stress. Experimental studies have shown that when the particle lung dose reaches a sufficiently high concentration (e.g., mass dose of ~0.5 mg fine-sized PSLT/g lung in rats), the alveolar macrophage-mediated clearance process begins to be impaired (complete impairment occurs at ~10 mg/g lung). Overloading of lung clearance is accompanied by pulmonary inflammation, leading to increased production of reactive oxygen and nitrogen species, depletion of antioxidants and/or impairment of other defense mechanisms, cell injury, cell proliferation, fibrosis, and as seen in rats, induction of mutations and eventually cancer. Rats appear to be more sensitive to carbon black and other PSLT than other rodent species. Although studies in humans have not shown a direct link between inhaled PSLT and lung cancer, many of the steps in the mechanism observed in rats have also been observed in humans who work in dusty jobs, including increased particle lung retention and pulmonary inflammation in workers exposed to coal dust or crystalline silica and elevated lung cancer has been observed in some studies of workers exposed to carbon black, crystalline silica, and diesel exhaust particles.

Monkeys exposed to channel black for 1000-1500 hours showed evidence of electrocardiac changes indicative of right atrial and right ventricular strain. These changes increased progressively until after 10,000 hours of exposure, when the changes were marked. The authors of this study concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of non-toxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours' exposure and marked atrial and right ventricular strain after 10,000 hours' exposure. The authors concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of nontoxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours exposure and marked atrial and right ventricular strain after 10,000 hours exposure.

Chromatographic fractions of oily material extracted from carbon black have been shown to be carcinogenic whilst the unfractionated extracts are not. The activity of some carcinogens appear to be inhibited by carbon black itself.

Chronic toluene habituation occurs following intentional abuse (glue sniffing) or from occupational exposure. Ataxia, incoordination and tremors of the hands and feet (as a consequence of diffuse cerebral atrophy), headache, abnormal speech, transient memory loss, convulsions, coma, drowsiness, reduced colour perception, frank blindness, nystagmus (rapid, involuntary eye-movements), hearing loss leading to deafness and mild dementia have all been associated with chronic abuse. Peripheral nerve damage, encephalopathy, giant axonopathy electrolyte disturbances in the cerebrospinal fluid and abnormal computer tomographic (CT scans) are common amongst toluene addicts. Although toluene abuse has been linked with kidney disease, this does not commonly appear in cases of occupational toluene exposures. Cardiac and haematological toxicity are however associated with chronic toluene exposures. Cardiac arrhythmia, multifocal and premature ventricular contractions and supraventricular tachycardia are present in 20% of patients who abused toluene-containing paints.

Previous suggestions that chronic toluene inhalation produced human peripheral neuropathy have been discounted. However central nervous system (CNS) depression is well documented where blood toluene exceeds 2.2 mg%. Toluene abusers can achieve transient circulating concentrations of 6.5%. Amongst workers exposed for a median time of 29 years, to toluene, no subacute effects on neuroathenic complaints and psychometric test results could be established.

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

839 Super Shield Graphite Conductive Coating	TOXICITY	IRRITATION
	Not Available	Not Available
toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation (rat) LC50: >26700 ppm/1hd ^[2]	Eye (rabbit): 0.87 mg - mild
	Inhalation (rat) LC50: 49 mg/L/4H ^[2]	Eye (rabbit): 100 mg/30sec - mild
	Oral (rat) LD50: 636 mg/kg ^[2]	Skin (rabbit): 20 mg/24h - moderate
		Skin (rabbit): 500 mg - moderate
graphite, natural	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2 mg/L4 h ^[1]	Eye (rabbit): non-irritant *
	Oral (rat) LD50: >2000 mg/kg ^{+[2]}	Eye : Not irritating
		Skin (rabbit): 4 h non-irritant *
		Skin : Not irritating
amyl methyl ketone	TOXICITY	IRRITATION

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	Dermal (rabbit) LD50: 12600 mg/kg ^[2]	Skin (rabbit): 14 mg/24h Mild
	Inhalation (rat) LC50: 4000 ppm/4h ^[2]	Skin (rabbit): Primary Irritant
	Oral (rat) LD50: 1670 mg/kgd ^[2]	
acetone	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 20000 mg/kg ^[2]	Eye (human): 500 ppm - irritant
	Inhalation (rat) LC50: 50.1 mg/L/8 hr ^[2]	Eye (rabbit): 20mg/24hr -moderate
	Oral (rat) LD50: 5800 mg/kgE ^[2]	Eye (rabbit): 3.95 mg - SEVERE
		Skin (rabbit): 500 mg/24hr - mild
		Skin (rabbit):395mg (open) - mild
ethanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 17100 mg/kg ^[1]	Eye (rabbit): 500 mg SEVERE
	Inhalation (rat) LC50: 64000 ppm/4h ^[2]	Eye (rabbit):100mg/24hr-moderate
	Oral (rat) LD50: >11872769 mg/kg ^[1]	Skin (rabbit):20 mg/24hr-moderate
		Skin (rabbit):400 mg (open)-mild
isobutyl acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[1]	moderate
	Oral (rat) LD50: 13400 mg/kgd ^[2]	Skin(rabbit): 500 mg open mild
propylene glycol monomethyl ether acetate, alpha-isomer	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	* [CCINFO]
	dermal (rat) LD50: >2000 mg/kg ^[1]	Nil reported
	Inhalation (rat) LC50: 4345 ppm/6h ^[2]	
	Inhalation (rat) LC50: 4345 ppm/6h ^[2]	
	Oral (rat) LD50: >14.1 ml ^[1]	
	Oral (rat) LD50: 8532 mg/kgd ^[2]	
ethyl acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >18000 mg/kg ^[2]	Eye (human): 400 ppm
	Inhalation (mouse) LC50: >18 mg/14 h ^[1]	
	Inhalation (mouse) LC50: 33.5 mg/12 h ^[1]	
	Inhalation (mouse) LC50: 45 mg/L/2H ^[2]	
	Inhalation (rat) LC50: >6000 ppm/6H ^[2]	
	Inhalation (rat) LC50: 1600 ppm/8h ^[2]	
	Inhalation (rat) LC50: 200 mg/11 h ^[1]	
Oral (rat) LD50: 10170 mg/kg ^[1]		
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2]	Not Available
	Oral (rat) LD50: >8000 mg/kg ^[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	

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No significant acute toxicological data identified in literature search.

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 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

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commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

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

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

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

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

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m³ for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m³. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m³), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating.

None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m³ (600 ppm) for PnB and 2,010 mg/m³ (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m³ (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m³ (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m³) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m³). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m³), with decreased body weights occurring at 3000 ppm (11058 mg/m³). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

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.]

For toluene:

Acute Toxicity

Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.

Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.

Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.

Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.

Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea. Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death.

Toluene can also strip the skin of lipids causing dermatitis.

Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days.

Subchronic/Chronic Effects:

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

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

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

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

Developmental/Reproductive Toxicity

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	<p>Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.</p> <p>Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy</p> <p>Animals - Sterebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m³. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.</p> <p>Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor. Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .</p> <p>Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .</p> <p>Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites</p> <p>Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.</p>
<p style="text-align: center;">TOLUENE</p>	<p>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.</p> <p>For toluene:</p> <p>Acute Toxicity</p> <p>Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.</p> <p>Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy. Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days. Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death. Toluene can also strip the skin of lipids causing dermatitis</p> <p>Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days</p> <p>Subchronic/Chronic Effects:</p> <p>Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.</p> <p>Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitizer and fatal cardiotoxin. Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L</p> <p>Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .</p> <p>Developmental/Reproductive Toxicity</p> <p>Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.</p> <p>Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy</p> <p>Animals - Sterebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m³. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.</p> <p>Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor. Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .</p> <p>Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .</p> <p>Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites</p> <p>Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.</p>
<p style="text-align: center;">GRAPHITE, NATURAL</p>	<p>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</p>

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	<p>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.</p> <p>* Timcal MSDS</p>
AMYL METHYL KETONE	<p>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.</p>
ACETONE	<p>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.</p> <p>for acetone:</p> <p>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.</p> <p>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.</p> <p>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.</p>
ETHANOL	<p>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.</p>
ISOBUTYL ACETATE	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>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.</p> <p>Inhalation (rat): 8000ppm/4h Skin(rabbit): 500 mg/24hr moderate</p>
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	<p>for propylene glycol ethers (PGEs):</p> <p>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).</p> <p>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.</p> <p>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).</p> <p>This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.</p> <p>Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.</p> <p>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.</p> <p>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</p> <p>None are skin sensitizers.</p> <p>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.</p> <p>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</p>

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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]

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CARBON BLACK

No significant acute toxicological data identified in literature search.

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
Inhalation (rat) TCLo: 50 mg/m³/6h/90D-I Nil reported

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 required to make classification available
✗ – Data available but does not fill the criteria for classification
☐ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
toluene	Not Available					
graphite, natural	Not Available					
amyl methyl ketone	Not Available					
acetone	Not Available					
ethanol	Not Available					
isobutyl acetate	Not Available					
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available					
ethyl acetate	Not Available					
carbon black	Not Available					

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.

When ethanol is released into the soil it readily and quickly biodegrades but may leach into ground water; most is lost by evaporation. When released into water the material readily evaporates and is biodegradable.

Ethanol does not bioaccumulate to an appreciable extent.

The material is readily degraded by reaction with photochemically produced hydroxy radicals; release into air will result in photodegradation and wet deposition.

Environmental Fate:

TERRESTRIAL FATE: An estimated Koc value of 1 indicates that ethanol is expected to have very high mobility in soil. Volatilisation of ethanol from moist soil surfaces is expected to be an important fate process given a Henry's Law constant of 5X10⁻⁶ atm-m³/mole. The potential for volatilisation of ethanol from dry soil surfaces may exist based upon an extrapolated vapor pressure of 59.3 mmHg. Biodegradation is expected to be an important fate process for ethanol based on half-lives on the order of a few days for ethanol in sandy soil/groundwater microcosms.

AQUATIC FATE: An estimated Koc value of 1 indicates that ethanol is not expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon a Henry's Law constant of 5X10⁻⁶ atm-m³/mole. Using this Henry's Law constant and an estimation method, volatilisation half-lives for a model river and model lake are 3 and 39 days, respectively. An estimated BCF= 3, from a log Kow of -0.31 suggests bioconcentration in aquatic organisms is low. Hydrolysis and photolysis in sunlight surface waters is not expected to be an important environmental fate process for ethanol since this compound lacks functional groups that hydrolyse or absorb light under environmentally relevant conditions. Ethanol was degraded with half-lives on

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the order of a few days in aquatic studies conducted using microcosms constructed with a low organic sandy soil and groundwater, indicating it is unlikely to be persistent in aquatic environments(8).

ATMOSPHERIC FATE: Ethanol, which has an extrapolated vapor pressure of 59.3 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapour-phase ethanol is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 5 days, calculated from its rate constant of 3.3×10^{-12} m³/mole-sec at 25 deg C.

Ecotoxicity:

log Kow: -0.31 - -0.32

Half-life (hr) air: 144

Half-life (hr) H₂O surface water: 144

Henry's atm m³/mol: 6.29E-06

BOD 5 if unstated: 0.93-1.67,63%

COD: 1.99-2.11,97%

ThOD: 2.1

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 substrate. The higher molecular weight ketones do not 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 propylene glycol ethers:

Environmental fate:

Most are liquids at room temperature and all are water-soluble.

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

Environmental fate: Log octanol-water partition coefficients (log Kow's) range from 0.309 for TPM to 1.523 for DPnB. Calculated BCFs range from 1.47 for DPnB to 3.16 for DPMA and TPM, indicating low bioaccumulation. Henry's Law Constants, which indicate propensity to partition from water to air, are low for all category members, ranging from 5.7×10^{-9} atm-m³/mole for TPM to 2.7×10^{-9} atm-m³/mole for PnB. Fugacity modeling indicates that most propylene glycol ethers are likely to partition roughly equally into the soil and water compartments in the environment with small to negligible amounts remaining in other environmental compartments (air, sediment, and aquatic biota). Propylene glycol ethers are unlikely to persist in the environment. Once in air, the half-life of the category members due to direct reactions with photochemically generated hydroxyl radicals, range from 2.0 hours for TPM to 4.6 hours for PnB. In water, most 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 toluene:

log Kow : 2.1-3

log Koc : 1.12-2.85

Koc : 37-260

log Kom : 1.39-2.89

Half-life (hr) air : 2.4-104

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

Half-life (hr) H₂O ground : 168-2628

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

Henry's Pa m³/mol: 518-694

Henry's atm m³/mol: 5.94E-03

BOD 5 0.86-2.12, 5%

COD : 0.7-2.52,21-27%

ThOD : 3.13

BCF : 1.67-380

log BCF : 0.22-3.28

Environmental fate:

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

Transformation/Persistence:

Air - The main degradation pathway for toluene in the atmosphere is reaction with photochemically produced hydroxyl radicals. The estimated atmospheric half life for toluene is about 13 hours.

Toluene is also oxidized by reactions with atmospheric nitrogen dioxide, oxygen, and ozone, but these are minor degradation pathways. Photolysis is not considered a significant degradative pathway for toluene

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

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

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

Ecotoxicity:

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

Fish LC50 (96 h): fathead minnow (*Pimephales promelas*) 12.6-72 mg/l; *Lepomis macrochirus* 13-24 mg/l;

guppy (*Poecilia reticulata*) 28.2-59.3 mg/l; channel catfish (*Ictalurus punctatus*) 240 mg/l; goldfish (*Carassius auratus*): 22.8-57.68 mg/l

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

shrimp (*Palaemonetes pugio*) 9.5 mg/l

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

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:

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

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

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

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

Acetone does not concentrate in the food chain.

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

Drinking Water Standard: none available.

Soil Guidelines: none available.

Air Quality Standards: none available.

Ecotoxicity:

Testing shows that acetone exhibits a low order of toxicity

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

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

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

Aquatic invertebrate 2100 - 16700 mg/l

Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephesia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m³. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone.

The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
amyl methyl ketone	LOW	LOW
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
isobutyl acetate	LOW	LOW
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
ethyl acetate	LOW (Half-life = 14 days)	LOW (Half-life = 14.71 days)

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
toluene	LOW (BCF = 90)
amyl methyl ketone	LOW (LogKOW = 1.98)
acetone	LOW (BCF = 69)
ethanol	LOW (LogKOW = -0.31)
isobutyl acetate	LOW (LogKOW = 1.78)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
ethyl acetate	HIGH (BCF = 3300)

12.4. Mobility in soil

Ingredient	Mobility
toluene	LOW (KOC = 268)
amyl methyl ketone	LOW (KOC = 24.01)
acetone	HIGH (KOC = 1.981)
ethanol	HIGH (KOC = 1)
isobutyl acetate	LOW (KOC = 17.48)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
ethyl acetate	LOW (KOC = 6.131)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT Criteria fulfilled?	Not Available	Not Available	Not Available

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12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ 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 MSDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ 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 licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced 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: 839-5ML, 839-900ML, 839-1G
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Land transport (ADR)

14.1. UN number	1263				
14.2. Packing group	II				
14.3. UN proper shipping name	PAINT or PAINT RELATED MATERIAL				
14.4. Environmental hazard	No relevant data				
14.5. Transport hazard class(es)	<table border="1"> <tr> <td>Class</td> <td>3</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	3	Subrisk	Not Applicable
Class	3				
Subrisk	Not Applicable				
14.6. Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>163 640C 640D 650</td> </tr> <tr> <td>Limited quantity</td> <td>5 L</td> </tr> </table>	Special provisions	163 640C 640D 650	Limited quantity	5 L
Special provisions	163 640C 640D 650				
Limited quantity	5 L				

Air transport (ICAO-IATA / DGR)

14.1. UN number	1263				
14.2. Packing group	II				
14.3. UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)				
14.4. Environmental hazard	No relevant data				
14.5. Transport hazard class(es)	<table border="1"> <tr> <td>ICAO/IATA Class</td> <td>3</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> </table>	ICAO/IATA Class	3	ICAO / IATA Subrisk	Not Applicable
ICAO/IATA Class	3				
ICAO / IATA Subrisk	Not Applicable				

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	ERG Code	3L
14.6. Special precautions for user	Special provisions	A3 A72 A192
	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	1263
14.2. Packing group	II
14.3. UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
14.4. Environmental hazard	Not Applicable
14.5. Transport hazard class(es)	IMDG Class 3
	IMDG Subrisk Not Applicable
14.6. Special precautions for user	EMS Number F-E , S-E
	Special provisions 163
	Limited Quantities 5 L

Inland waterways transport (ADN)

14.1. UN number	1263
14.2. Packing group	II
14.3. UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning and reducing compound)
14.4. Environmental hazard	No relevant data
14.5. Transport hazard class(es)	3 Not Applicable
14.6. Special precautions for user	Classification code F1
	Limited quantity 5 L
	Equipment required PP, EX, A
	Fire cones number 1

Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	toluene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	arnyl methyl ketone	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	isobutyl acetate	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	propylene glycol monomethyl ether acetate, alpha-isomer	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	ethyl acetate	Z

SECTION 15 REGULATORY INFORMATION

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

TOLUENE(108-88-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

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EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 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
 European Customs Inventory of Chemical Substances ECICS (English)
 European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
 European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

GRAPHITE, NATURAL(7782-42-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 European Customs Inventory of Chemical Substances ECICS (English)

AMYL METHYL KETONE(110-43-0) 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
 European Customs Inventory of Chemical Substances ECICS (English)
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
 European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Bulgarian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Czech)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Danish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Dutch)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Estonian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Finnish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)

ACETONE(67-64-1) 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
 European Customs Inventory of Chemical Substances ECICS (English)
 European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
 European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Bulgarian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Czech)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Danish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Dutch)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Estonian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Finnish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)

ETHANOL(64-17-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Reprotoxic Substances
 European Union (EU) Commission Directive 2006/15/EC establishing a second list of indicative occupational exposure limit values (IOELVs)
 European Union (EU) Commission Directive 2006/15/EC establishing a second list of indicative occupational exposure limit values (IOELVs) (Spanish)
 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
 UK Workplace Exposure Limits (WELs)

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

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Maltese)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Polish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Portuguese)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Romanian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovak)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovenian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Spanish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Swedish)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
 UK Workplace Exposure Limits (WELs)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Maltese)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Polish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Portuguese)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Romanian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovak)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovenian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Spanish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Swedish)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
 UK Workplace Exposure Limits (WELs)

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

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
UK Workplace Exposure Limits (WELs)

ISOBUTYL ACETATE(110-19-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
UK Workplace Exposure Limits (WELs)

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER(108-65-6) 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
European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Bulgarian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Czech)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Danish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Dutch)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Estonian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Finnish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Maltese)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Polish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Portuguese)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Romanian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovak)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovenian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Spanish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Swedish)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
UK Workplace Exposure Limits (WELs)

ETHYL ACETATE(141-78-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
UK Workplace Exposure Limits (WELs)

CARBON BLACK(1333-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Customs Inventory of Chemical Substances ECICS (English)
European List of Notified Chemical Substances (ELINCS)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
UK Workplace Exposure Limits (WELs)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
toluene	108-88-3	601-021-00-3	01-2119471310-51-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2, Asp. Tox. 1, Skin Irrit. 2, STOT SE 3, Repr. 2, STOT RE 2	GHS02, GHS08, Dgr	H225, H304, H315, H336, H361, H373
2	Flam. Liq. 2, Asp. Tox. 1, Skin Irrit. 2, Eye Irrit. 2, Aquatic Chronic 2, STOT RE 1, Aquatic Chronic 3, Repr. 1A, Acute Tox. 4, Muta. 1B, Carc. 1A, STOT SE 1, Skin Sens. 1	GHS08, Dgr, GHS09, GHS01, GHS06	H225, H304, H315, H319, H411, H372, H362, H301, H332, H228, H360, H340, H350, H370

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
graphite, natural	7782-42-5	Not Available	01-2119486977-12-XXXX, 01-2119875125-36-XXXX

839 Super Shield Graphite Conductive Coating

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Eye Irrit. 2, STOT SE 3, Skin Irrit. 2, STOT RE 1, Acute Tox. 4, Aquatic Chronic 3	Wng, GHS08, Dgr, GHS02	H335, H315, H318, H372, H228, H302, H412

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
amyl methyl ketone	110-43-0	606-024-00-3	01-2119902391-49-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 3, Acute Tox. 4	GHS07, GHS02, Wng	H226, H302, H332
2	Flam. Liq. 3, Acute Tox. 4, STOT SE 3	GHS07, Wng, GHS01	H226, H302, H332, H336

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
acetone	67-64-1	606-001-00-8	01-2119498062-37-XXXX, 01-2119471330-49-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3	GHS07, GHS02, Dgr	H225, H319, H336
2	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3, Eye Irrit. 2A	Dgr, GHS01, GHS08, Wng, GHS06	H225, H319, H400, H371, H228, H315, H340, H332, H302

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
ethanol	64-17-5	603-002-00-5	01-2119457610-43-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2	GHS02, Dgr	H225
2	Flam. Liq. 2, Eye Irrit. 2, STOT RE 1, Muta. 1B, Repr. 1A, Acute Tox. 3, STOT SE 1, Met. Corr. 1, Skin Corr. 1B, Aquatic Acute 1, Aquatic Chronic 1	Dgr, GHS01, GHS08, Wng, GHS06, GHS05	H225, H319, H340, H304, H372, H315, H220, H360, H301, H311, H331, H370
1	Carc. 2	GHS08, Wng	H351
2	Carc. 2	GHS08, Wng	H351

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
isobutyl acetate	110-19-0	607-026-00-7	01-2119488971-22-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2	GHS02, Dgr	H225
2	Flam. Liq. 2, STOT SE 3, Eye Irrit. 2	Dgr, GHS07, GHS01	H225, H336, H319

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6	607-195-00-7	01-2119475791-29-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 3, Eye Irrit. 2, Repr. 1B	GHS02, Wng, GHS08, Dgr	H226, H319, H360
2	Flam. Liq. 3, Eye Dam. 1, Eye Irrit. 2, Repr. 1B	GHS02, Wng, GHS05, Dgr, GHS03, GHS08	H226, H319, H360

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
ethyl acetate	141-78-6	607-022-00-5	01-2119475103-46-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3	GHS07, GHS02, Dgr	H225, H319, H336
2	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3, Aquatic Chronic 1, Eye Irrit. 2A, Acute Tox. 4, Asp. Tox. 1, Skin Sens. 1	GHS07, Dgr, GHS01, Wng	H225, H319, H336, H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
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839 Super Shield Graphite Conductive Coating

carbon black	1333-86-4	Not Available	01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX
Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Carc. 2, Eye Irrit. 2, STOT RE 1, Self-heat. 1, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Acute Tox. 4, Flam. Sol. 2	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H319, H372, H251, H228, H315, H370, H410, H332

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (toluene; propylene glycol monomethyl ether acetate, alpha-isomer; acetone; ethyl acetate; ethanol; isobutyl acetate; carbon black; graphite, natural; amyl methyl ketone)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (graphite, natural)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Full text Risk and Hazard codes

H220	Extremely flammable gas
H226	Flammable liquid and vapour
H228	Flammable solid
H251	Self-heating; may catch fire
H301	Toxic if swallowed
H302	Harmful if swallowed
H311	Toxic in contact with skin
H318	Causes serious eye damage
H331	Toxic if inhaled
H332	Harmful if inhaled
H335	May cause respiratory irritation
H340	May cause genetic defects
H350	May cause cancer
H360	May damage fertility or the unborn child
H361d ***	H361d ***
H362	May cause harm to breast-fed children
H370	Causes damage to organs
H371	May cause damage to organs
H372	Causes damage to organs through prolonged or repeated exposure
H373 **	H373 **
H400	Very toxic to aquatic life
H410	Very toxic to aquatic life with long lasting effects
H411	Toxic to aquatic life with long lasting effects
H412	Harmful to aquatic life with long lasting effects

Other information

Ingredients with multiple cas numbers

Name	CAS No
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 142300-82-1, 84540-57-8

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

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

www.chemwatch.net

839 Super Shield Graphite Conductive Coating

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

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