



838 Total Ground Carbon Conductive Coating (Aerosol)

MG Chemicals (Head Office)

Chemwatch Hazard Alert Code: 3

Version No: 5.14

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

Issue Date: 26/09/2015

Print Date: 26/09/2015

Initial Date: 01/01/0001

L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	838 Total Ground Carbon Conductive Coating (Aerosol)
Synonyms	SDS Code 838-Aerosol; Related Part # 838-340G
Proper shipping name	AEROSOLS
Other means of identification	Not Available

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

Relevant identified uses	Electrically conductive coating
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]	Eye Irritation Category 2, STOT - SE (Resp. Irr.) Category 3, STOT - SE (Narcosis) Category 3, Carcinogen Category 2, STOT - RE Category 2, Chronic Aquatic Hazard Category 3, Aerosol Category 3, Flammable Aerosol Category 2, Reproductive Toxicity 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 **WARNING**

Hazard statement(s)

Continued...

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H319	Causes serious eye irritation
H335	May cause respiratory irritation
H336	May cause drowsiness or dizziness
H351	Suspected of causing cancer
H373	May cause damage to organs through prolonged or repeated exposure
H412	Harmful to aquatic life with long lasting effects
H229	Pressurised container: May burst if heated.
H223	Flammable aerosol
H361	Suspected of damaging fertility or the unborn child

Supplementary statement(s)

EUH066	Repeated exposure may cause skin dryness or cracking
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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.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
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.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
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.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
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 respiratory system and skin*.
	Inhalation may produce health damage*.

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 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.811-97-2 2.212-377-0 3.Not Available 4.01-2119459374-33-XXXX	50-70	<u>tetrafluoroethane</u>	Gas under Pressure (Compressed gas); H280, EUH018, EUH044 ^[1]
1.67-64-1 2.200-662-2 3.606-001-00-8 4.01-2119498062-37-XXXX,	10-30	<u>acetone</u>	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3; H225, H319, H336, EUH066 ^[3]

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01-2119471330-49-XXXX			
1.108-88-3 2.203-625-9 3.601-021-00-3 4.01-2119471310-51-XXXX	3-6	<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]
1.108-65-6 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]
1.64-17-5 2.200-578-6 3.603-002-00-5 4.01-2119457610-43-XXXX	1-5	<u>ethanol</u>	Flam. Liq. 2; H225 [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	1-5	<u>carbon black</u>	Carcinogen Category 2; H351 [1]
1.110-19-0 2.203-745-1 3.607-026-00-7 4.01-2119488971-22-XXXX	1-5	<u>isobutyl acetate</u>	Flam. Liq. 2; H225, EUH066 [3]
1.110-43-0 2.203-767-1 3.606-024-00-3 4.01-2119902391-49-XXXX	1-5	<u>amyl methyl ketone</u>	Flam. Liq. 3, Acute Tox. 4 *, Acute Tox. 4 *; H226, H332, H302 [3]
1.141-78-6 2.205-500-4 3.607-022-00-5 4.01-2119475103-46-XXXX	0.1-1	<u>ethyl acetate</u>	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3; H225, H319, H336, EUH066 [3]

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"> ▶ Not considered a normal route of entry. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus. <p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> ▶ Remove to fresh air. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor. <p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. <p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> ▶ Flush skin and hair with running water (and soap if available). ▶ Remove any adhering solids with industrial skin cleansing cream. ▶ DO NOT use solvents. ▶ Seek medical attention in the event of irritation.
Eye Contact	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> ▶ Flush skin and hair with running water (and soap if available). ▶ Remove any adhering solids with industrial skin cleansing cream. ▶ DO NOT use solvents. ▶ Seek medical attention in the event of irritation.
Inhalation	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> ▶ Remove to fresh air. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ Not considered a normal route of entry. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

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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

for intoxication due to Freons/ Halons;

A: Emergency and Supportive Measures

- ▶ Maintain an open airway and assist ventilation if necessary
- ▶ Treat coma and arrhythmias if they occur. Avoid (adrenaline) epinephrine or other sympathomimetic amines that may precipitate ventricular arrhythmias. Tachyarrhythmias caused by increased myocardial sensitisation may be treated with propranolol, 1-2 mg IV or esmolol 25-100 microg/kg/min IV.
- ▶ Monitor the ECG for 4-6 hours

B: Specific drugs and antidotes:

- ▶ There is no specific antidote

C: Decontamination

- ▶ Inhalation; remove victim from exposure, and give supplemental oxygen if available.
- ▶ Ingestion; (a) Prehospital: Administer activated charcoal, if available. **DO NOT** induce vomiting because of rapid absorption and the risk of abrupt onset CNS depression. (b) Hospital: Administer activated charcoal, although the efficacy of charcoal is unknown. Perform gastric lavage only if the ingestion was very large and recent (less than 30 minutes)

D: Enhanced elimination:

- ▶ There is no documented efficacy for diuresis, haemodialysis, haemoperfusion, or repeat-dose charcoal.

POISONING and DRUG OVERDOSE, Californian Poison Control System Ed. Kent R Olson; 3rd Edition

- ▶ Do not administer sympathomimetic drugs unless absolutely necessary as material may increase myocardial irritability.
- ▶ No specific antidote.
- ▶ Because rapid absorption may occur through lungs if aspirated and cause systematic effects, the decision of whether to induce vomiting or not should be made by an attending physician.
- ▶ If lavage is performed, suggest endotracheal and/or esophageal control.
- ▶ Danger from lung aspiration must be weighed against toxicity when considering emptying the stomach.
- ▶ Treatment based on judgment of the physician in response to reactions of the patient

Treat symptomatically.

For acute or short term repeated exposures to acetone:

- ▶ Symptoms of acetone exposure approximate ethanol intoxication.
- ▶ About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.
- ▶ There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]

Management:

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

Inhalation Management:

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

Dermal Management:

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

Eye Management:

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

Oral Management:

- ▶ No **GASTRIC LAVAGE OR EMETIC**
- ▶ Encourage oral fluids.

Systemic Management:

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

The Chemical Incident Management Handbook:

Guy's and St. Thomas' Hospital Trust, 2000

BIOLOGICAL EXPOSURE INDEX

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

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

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

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

- ▶ Alcohol stable foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.
- ▶ Water spray or fog - Large fires only.

SMALL FIRE:

- ▶ Water spray, dry chemical or CO2

LARGE FIRE:

- ▶ Water spray or fog.

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

Continued...

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Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<p>Combustion products include; carbon dioxide (CO₂) WARNING: In use may form flammable/ explosive vapour-air mixtures.</p> <ul style="list-style-type: none"> ▶ Liquid and vapour are flammable. ▶ Moderate fire hazard when exposed to heat or flame. ▶ Vapour forms an explosive mixture with air. ▶ Moderate explosion hazard when exposed to heat or flame. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ Aerosol cans may explode on exposure to naked flame. ▶ Rupturing containers may rocket and scatter burning materials. ▶ Hazards may not be restricted to pressure effects. ▶ May emit acrid, poisonous or corrosive fumes. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>, carbon monoxide (CO) hydrogen fluoride, other pyrolysis products typical of burning organic material Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.</p>

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	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Wear protective clothing, impervious gloves and safety glasses. ▶ Shut off all possible sources of ignition and increase ventilation. ▶ Wipe up. ▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely. 																																																																											
Major Spills	<p>Chemical Class: aliphatics, halogenated For release onto land: recommended sorbents listed in order of priority.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 30%;">SORBENT TYPE</th> <th style="width: 10%;">RANK</th> <th style="width: 30%;">APPLICATION</th> <th style="width: 10%;">COLLECTION</th> <th style="width: 20%;">LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>shovel</td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>wood fiber - pillow</td> <td>2</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>treated wood fibre - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R, W, DGC</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>foamed glass - pillow</td> <td>3</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td colspan="5">LAND SPILL - MEDIUM</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R,W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>2</td> <td>throw</td> <td>skiploader</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>W, SS, DGC</td> </tr> <tr> <td>foamed glass - pillow</td> <td>3</td> <td>throw</td> <td>skiploader</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> </tbody> </table> <p>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</p>	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	wood fiber - pillow	2	throw	pitchfork	R, P, DGC, RT	treated wood fibre - particulate	2	shovel	shovel	R, W, DGC	sorbent clay - particulate	3	shovel	shovel	R, I, P	foamed glass - pillow	3	throw	pitchfork	R, P, 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	foamed glass - pillow	3	throw	skiploader	R, P, DGC, RT	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
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Continued...

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Chemical Class: ketones

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

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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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 - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
foamed glass - pillow	4	throw	pitchfork	R, P, DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skid loader	R,W, SS
cross-linked polymer - pillow	2	throw	skid loader	R, DGC, RT
sorbent clay - particulate	3	blower	skid loader	R, I, P
polypropylene - particulate	3	blower	skid loader	R, SS, DGC
expanded mineral - particulate	4	blower	skid loader	R, I, W, P, DGC
polypropylene - mat	4	throw	skid loader	DGC, RT

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

Chemical Class: aromatic hydrocarbons

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

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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LAND SPILL - SMALL

Feathers - pillow	1	throw	pitchfork	DGC, RT
cross-linked polymer - particulate	2	shovel	shovel	R,W,SS
cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	3	shovel	shovel	R, I, P,
treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I
wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT

LAND SPILL - MEDIUM

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

Legend

DGC: Not effective where ground cover is dense

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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 all unprotected personnel and move upwind.
- ▶ Alert Emergency Authority and advise them of the location and nature of hazard.
- ▶ May be violently or explosively reactive.
- ▶ Wear full body clothing with breathing apparatus.
- ▶ Prevent by any means available, spillage from entering drains and water-courses.
- ▶ Consider evacuation.
- ▶ Shut off all possible sources of ignition and increase ventilation.
- ▶ No smoking or naked lights within area.
- ▶ Use extreme caution to prevent violent reaction.
- ▶ Stop leak only if safe to do so.

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- ▶ Water spray or fog may be used to disperse vapour.
- ▶ **DO NOT enter confined space where gas may have collected.**
- ▶ Keep area clear until gas has dispersed.

- ▶ Remove leaking cylinders to a safe place.
- ▶ Fit vent pipes. Release pressure under safe, controlled conditions
- ▶ Burn issuing gas at vent pipes.
- ▶ **DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve.**
- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ May be violently or explosively reactive.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water courses
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse / absorb vapour.
- ▶ Absorb or cover spill with sand, earth, inert materials or vermiculite.
- ▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.
- ▶ Undamaged cans should be gathered and stowed safely.
- ▶ Collect residues and seal in labelled drums for disposal.

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	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ DO NOT incinerate or puncture aerosol cans. ▶ DO NOT spray directly on humans, exposed food or food utensils. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this MSDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Fire and explosion protection	See section 5
Other information	<ul style="list-style-type: none"> ▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can ▶ Store in original containers in approved flammable liquid storage area. ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped. ▶ No smoking, naked lights, heat or ignition sources. ▶ Keep containers securely sealed. Contents under pressure. ▶ Store away from incompatible materials. ▶ Store in a cool, dry, well ventilated area. ▶ Avoid storage at temperatures higher than 40 deg C. ▶ Store in an upright position. ▶ Protect containers against physical damage. ▶ Check regularly for spills and leaks. ▶ Observe manufacturer's 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"> ▶ DO NOT use aluminium or galvanised containers ▶ Aerosol dispenser. ▶ Check that containers are clearly labelled.
Storage incompatibility	<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.

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- ▶ 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.

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

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

As a general rule, hydrofluorocarbons tend to be flammable unless they contain more fluorine atoms than hydrogen atoms.

Acetone:

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

Haloalkanes:

- ▶ are highly reactive: some of the more lightly substituted lower members are highly flammable; the more highly substituted may be used as fire suppressants, not always with the anticipated results.
- ▶ may react with the lighter divalent metals to produce more reactive compounds analogous to Grignard reagents.
- ▶ may produce explosive compounds following prolonged contact with metallic or other azides
- ▶ may react on contact with potassium or its alloys - although apparently stable on contact with a wide range of halocarbons, reaction products may be shock-sensitive and may explode with great violence on light impact; severity generally increases with the degree of halocarbon substitution and potassium-sodium alloys give extremely sensitive mixtures.

BRETHERRICK L.: Handbook of Reactive Chemical Hazards

- ▶ react with metal halides and active metals, eg. sodium (Na), potassium (K), lithium (Li), calcium (Ca), zinc (Zn), powdered aluminium (Al) and aluminium alloys, magnesium (Mg) and magnesium alloys.
- ▶ may react with brass and steel.
- ▶ may react explosively with strong oxidisers
- ▶ may degrade rubber, and plastics such as methacrylate polymers, polyethylene and polystyrene, paint and coatings

Ketones in this group:

- ▶ are reactive with many acids and bases liberating heat and flammable gases (e.g., H₂).
- ▶ react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H₂) and heat.
- ▶ are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides.
- ▶ react violently with aldehydes, HNO₃ (nitric acid), HNO₃ + H₂O₂ (mixture of nitric acid and hydrogen peroxide), and HClO₄ (perchloric acid).
- ▶ may react with hydrogen peroxide to form unstable peroxides; many are heat- and shock-sensitive explosives.

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

Propylene glycol monomethyl ether acetate:

- ▶ may polymerise unless properly inhibited due to peroxide formation
- ▶ should be isolated from UV light, high temperatures, free radical initiators
- ▶ may react with strong oxidisers to produce fire and/ or explosion
- ▶ reacts violently with sodium peroxide, uranium fluoride
- ▶ is incompatible with sulfuric acid, nitric acid, caustics, aliphatic amines, isocyanates, boranes
- ▶ Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances

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)	tetrafluoroethane	1,1,1,2-Tetrafluoroethane (HFC 134a)	4240 mg/m ³ / 1000 ppm	Not Available	Not Available	Not Available
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

Continued...

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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)	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)	ethanol	Ethanol	1920 mg/m ³ / 1000 ppm	Not Available	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
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
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)	ethyl acetate	Ethyl acetate	200 ppm	400 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
tetrafluoroethane	Tetrafluoroethane, 1,1,1,2-; (HFC 134a)	Not Available	Not Available	Not Available
acetone	Acetone	Not Available	Not Available	Not Available
toluene	Toluene	Not Available	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
ethanol	Ethyl alcohol; (Ethanol)	Not Available	Not Available	Not Available
carbon black	Carbon black	9 mg/m ³	99 mg/m ³	590 mg/m ³
isobutyl acetate	Isobutyl acetate	450 ppm	1300 ppm	7500 ppm
amyl methyl ketone	Methyl n-amyl ketone	50 ppm	50 ppm	4000 ppm
ethyl acetate	Ethyl acetate	400 ppm	400 ppm	10000 ppm

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

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:

Continued...

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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 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. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.

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

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

Odour Safety Factor(OSF)

OSF=38 (ACETONE)

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

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

8.2.1. Appropriate engineering controls

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

The basic types of engineering controls are:

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

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

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

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

Provide adequate ventilation in warehouse or closed storage areas.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.


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

Within each range the appropriate value depends on:

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

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	of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
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] ▶ Close fitting gas tight goggles <p>DO NOT wear contact lenses.</p> <ul style="list-style-type: none"> ▶ 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"> ▶ No special equipment needed when handling small quantities. ▶ OTHERWISE: ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton. ▶ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. <p>BRETHERRICK: Handbook of Reactive Chemical Hazards.</p> <p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces.
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
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	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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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	<p>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.</p>		
	<p>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.</p>		
	<p>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.</p>		
	<p>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.</p>		
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Physical state	Liquid	Relative density (Water = 1)	0.89
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	465
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

Continued...

838 Total Ground Carbon Conductive Coating (Aerosol)

Flash point (°C)	-18	Taste	Not Available
Evaporation rate	>1 BuAC = 1	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"> ▶ Elevated temperatures. ▶ Presence of open flame. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
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>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</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>Exposure to high concentrations of fluorocarbons may produce cardiac arrhythmias or cardiac arrest due sensitisation of the heart to adrenalin or noradrenalin. Deaths associated with exposures to fluorocarbons (specifically halogenated aliphatics) have occurred in occupational settings and in inhalation of bronchodilator drugs.</p> <p>Bronchospasm consistently occurs in human subjects inhaling fluorocarbons. At a measured concentration of 1700 ppm of one of the commercially available aerosols there is a biphasic change in ventilatory capacity, the first reduction occurring within a few minutes and the second delayed up to 30 minutes. Most subjects developed bradycardia (reduced pulse rate).</p> <p>Bradycardia is encountered in dogs when administration is limited to upper respiratory tract (oropharyngeal and nasal areas). Cardiac arrhythmias can be experimentally induced in animals (species dependency is pronounced with dogs and monkeys requiring lesser amounts of fluorocarbon FC-11 than rats or mice). Sensitivity is increased by injection of adrenalin or cardiac ischaemia/necrosis or pulmonary thrombosis/bronchitis. The cardiotoxic effects of the fluorocarbons originate from irritation of the respiratory tract which in turn reflexively influences the heart rate (even prior to absorption of the fluorocarbon) followed by direct depression of the heart after absorption.</p> <p>Exposure to fluorocarbon thermal decomposition products may produce flu-like symptoms including chills, fever, weakness, muscular aches, headache, chest discomfort, sore throat and dry cough. Complete recovery usually occurs within 24 hours of exposure.</p> <p>Common, generalised symptoms associated with toxic gas inhalation include:</p> <ul style="list-style-type: none"> ▶ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures; ▶ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest; ▶ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest; ▶ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain. <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>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</p>
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838 Total Ground Carbon Conductive Coating (Aerosol)

	<p>1000 ppm. WARNING: Intentional misuse by concentrating/inhaling contents may be lethal. Acute intoxication by halogenated aliphatic hydrocarbons appears to take place over two stages. Signs of a reversible narcosis are evident in the first stage and in the second stage signs of injury to organs may become evident, a single organ alone is (almost) never involved.</p>								
Ingestion	<p>Overexposure is unlikely in this form.</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>Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments Accidental ingestion of the material may be damaging to the health of the individual.</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>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either:</p> <ul style="list-style-type: none"> ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but mild, 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 (non allergic). 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. Spray mist may produce discomfort</p> <p>In common with other halogenated aliphatics, fluorocarbons may cause dermal problems due to a tendency to remove natural oils from the skin causing irritation and the development of dry, sensitive skin. They do not appear to be appreciably absorbed. 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. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p>								
Eye	<p>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. Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p> <p>Undiluted propylene glycol monomethyl ether acetate (PGMEA) causes moderate discomfort, slight conjunctival redness and slight corneal injury in rabbits Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration</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. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</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. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. 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 Principal route of occupational exposure to the gas is by inhalation.</p>								

838 Total Ground Carbon Conductive Coating (Aerosol)

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".

It is generally accepted that the fluorocarbons are less toxic than the corresponding halogenated aliphatic based on chlorine. Repeated inhalation exposure to the fluorocarbon FC-11 does not produce pathologic lesions of the liver and other visceral organs in experimental animals. There has been conjecture in non-scientific publications that fluorocarbons may cause leukemia, cancer, sterility and birth defects; these have not been verified by current research. The high incidence of cancer, spontaneous abortion and congenital anomalies amongst hospital personnel, repeatedly exposed to fluorine-containing general anaesthetics, has caused some scientists to call for a lowering of the fluorocarbon exposure standard to 5 ppm since some are mutagens.

Workers exposed to 700 ppm acetone for 3 hours/day for 7-15 years showed inflammation of the respiratory tract, stomach and duodenum, attacks of giddiness and loss of strength. Exposure to acetone may enhance liver toxicity of chlorinated solvents.

838 Total Ground Carbon Conductive Coating (Aerosol)	TOXICITY	IRRITATION
	Not Available	Not Available
tetrafluoroethane	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 1500 mg/L/4h ^[2]	Not Available
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	
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/kgE ^[2]	Skin (rabbit):20 mg/24h-moderate
	Skin (rabbit):500 mg - moderate	
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]	
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	
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2]	Not Available
	Oral (rat) LD50: >8000 mg/kg ^[1]	

838 Total Ground Carbon Conductive Coating (Aerosol)

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
amyl methyl ketone	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12600 mg/kg ^[2]	Skin (rabbit): 14 mg/24h Mild
	Inhalation (rat) LC50: 4000 ppm/4h ^[2]	Skin (rabbit): Primary Irritant
ethyl acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >18000 mg/kg ^[2]	Eye (human): 400 ppm
	Inhalation (mouse) LC50: >18 mg/4 h ^[1]	
	Inhalation (mouse) LC50: 33.5 mg/2 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]		
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	

838 Total Ground Carbon Conductive Coating (Aerosol)	<p>No significant acute toxicological data identified in literature search.</p> <p>Disinfection by products (DBPs) re formed when disinfectants such as chlorine, chloramine, and ozone react with organic and inorganic matter in water. The observations that some DBPs such as trihalomethanes (THMs), di-/trichloroacetic acids, and 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) are carcinogenic in animal studies have raised public concern over the possible adverse health effects of DBPs. To date, several hundred DBPs have been identified.</p> <p>Numerous haloalkanes and haloalkenes have been tested for carcinogenic and mutagenic activities. In general, the genotoxic potential is dependent on the nature, number, and position of halogen(s) and the molecular size of the compound. Short-chain monohalogenated (excluding fluorine) alkanes and alkenes are potential direct-acting alkylating agents, particularly if the halogen is at the terminal end of the carbon chain or at an allylic position. Dihalogenated alkanes are also potential alkylating or cross-linking agents (either directly or after GSH conjugation), particularly if they are vicinally substituted (e.g., 1,2-dihaloalkane) or substituted at the two terminal ends of a short to medium-size (e.g., 2-7) alkyl moiety (i.e., alpha, omega-dihaloalkane). Fully halogenated haloalkanes tend to act by free radical or nongenotoxic mechanisms (such as generating peroxisome-proliferative intermediates) or undergo reductive dehalogenation to yield haloalkenes that in turn could be activated to epoxides.</p> <p>Haloalkenes are of concern because of potential to generate genotoxic intermediates after epoxidation. The concern for haloalkenes may be diminished if the double bond is internal or sterically hindered.</p> <p>The cancer concern levels of the 14 haloalkanes and haloalkenes, have been rated based on available screening cancer bioassay (pulmonary adenoma assay) and genotoxicity data. Five brominated and iodinated methane and ethane derivatives are given a moderate rating. Beyond the fact that bromine and iodine are better leaving groups than chlorine, there is also evidence that brominated THMs may be preferentially activated by a theta-class glutathione S-transferase (GSTT1-1) to mutagens in Salmonella even at low substrate concentrations. Furthermore, there are human carcinogenicity implications because of polymorphism in GSTT1-1. Human subpopulations with expressed GSTT1-1 may be at a greater risk to brominate THMs than humans who lack the gene.</p> <p>Six, two, and one haloalkanes/ haloalkene(s) are given low-moderate, marginal, and low concern, respectively.</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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <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,</p>
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838 Total Ground Carbon Conductive Coating (Aerosol)

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.I]

for acetone:

The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitizer but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage.

Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m³ and in rats at 26,100 mg/m³. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m³ for both rats and mice.

Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m³, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.

The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m³ have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m³ were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m³ or greater.

TETRAFLUOROETHANE

* with added oxygen - ZhongHao New Chemical Materials MSDS Excessive concentration can have a narcotic effect; inhalation of high concentrations of decomposition products can cause lung oedema.

ACETONE

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.

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TOLUENE

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 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

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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, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day).

Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developing human fetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.

Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy.

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

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

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

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

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

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

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

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

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

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

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

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

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER

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	<p>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 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.</p> <p>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.</p> <p>The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. <i>In vitro</i>, 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 <i>in vivo</i>. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.</p> <p>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.</p> <p>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]</p> <p>A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I] *Shin-Etsu SDS</p>
ETHANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
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/m ³ /6h/90D-I Nil reported
ISOBUTYL ACETATE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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. Inhalation (rat): 8000ppm/4h Skin(rabbit): 500 mg/24hr moderate
AMYL METHYL KETONE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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
tetrafluoroethane	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
acetone	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
toluene	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
ethanol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
carbon black	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
isobutyl acetate	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
amyl methyl ketone	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...

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ethyl acetate	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
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Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

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

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

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 5×10^{-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 5×10^{-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 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/molecule-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

In addition to carbon dioxide (CO₂), methane (CH₄) and nitrous oxide (N₂O), the greenhouse gases mentioned in the Kyoto Protocol include synthetic substances that share the common feature of being highly persistent in the atmosphere and exhibiting very high specific radiative forcing (radiative forcing is the change in the balance between radiation coming into the atmosphere and radiation out; a positive radiative forcing tends on average to warm the surface of the earth). These synthetic substances include hydrocarbons that are partially fluorinated (HFCs) or totally fluorinated (PFCs) as well as sulfur hexafluoride (SF₆).

The greenhouse potential of these substances, expressed as multiples of that of CO₂, are within the range of 140 to 11,700 for HFCs, from 6500 to 9,200 for PFCs and 23,900 for SF₆. Once emitted into the atmosphere, these substances have an impact on the environment for decades, centuries, or in certain instances, for thousands of years.

Many of these substances have only been commercialised for a few years, and still only contribute only a small percentage of those gases released to the atmosphere by humans (anthropogenic) which increase the greenhouse effect. However, a rapid increase can be seen in their consumption and emission, and therefore in their contribution to the anthropogenic increase in the greenhouse effect.

Since the adoption of the Kyoto Protocol, new fluorinated substances have appeared on the market, which are stable in air and have a high greenhouse potential; these include nitrogen trifluoride (NF₃) and fluoroethers.

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%

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COD : 0.7-2.52,21-27%

ThOD : 3.13

BCF : 1.67-380

log BCF : 0.22-3.28

Environmental fate:

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

Transformation/Persistence:

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

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

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

Biota - Bioaccumulation in most organisms is limited by the metabolism of toluene into more polar compounds 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)

DO NOT discharge into sewer or waterways.

for acetone:

log Kow: -0.24

Half-life (hr) air: 312-1896

Half-life (hr) H₂O surface water: 20

Henry's atm m³/mol: 3.67E-05

BOD 5: 0.31-1.76,46-55%

COD: 1.12-2.07

ThOD: 2.2

BCF: 0.69

Environmental fate:

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

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

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

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

Acetone does not concentrate in the food chain.

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

Drinking Water Standard: none available.

Soil Guidelines: none available.

Air Quality Standards: none available.

Ecotoxicity:

Testing shows that acetone exhibits a low order of toxicity

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

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

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

Aquatic invertebrate 2100 - 16700 mg/l

Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*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.

12.2. Persistence and degradability

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

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12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
tetrafluoroethane	LOW (LogKOW = 1.68)
acetone	LOW (BCF = 69)
toluene	LOW (BCF = 90)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
ethanol	LOW (LogKOW = -0.31)
isobutyl acetate	LOW (LogKOW = 1.78)
amyl methyl ketone	LOW (LogKOW = 1.98)
ethyl acetate	HIGH (BCF = 3300)

12.4. Mobility in soil

Ingredient	Mobility
tetrafluoroethane	LOW (KOC = 96.63)
acetone	HIGH (KOC = 1.981)
toluene	LOW (KOC = 268)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
ethanol	HIGH (KOC = 1)
isobutyl acetate	LOW (KOC = 17.48)
amyl methyl ketone	LOW (KOC = 24.01)
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

12.6. Other adverse effects

No data available


SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	<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. ▶ Consult State Land Waste Management Authority for disposal. ▶ Discharge contents of damaged aerosol cans at an approved site. ▶ Allow small quantities to evaporate. ▶ DO NOT incinerate or puncture aerosol cans. ▶ Bury residues and emptied aerosol cans at an approved site.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
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Land transport (ADR)

14.1. UN number	1950				
14.2. Packing group	Not Applicable				
14.3. UN proper shipping name	Aerosols, flammable				
14.4. Environmental hazard	No relevant data				
14.5. Transport hazard class(es)	<table border="1"> <tr> <td>Class</td> <td>2.1</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	2.1	Subrisk	Not Applicable
Class	2.1				
Subrisk	Not Applicable				
14.6. Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>Not Applicable</td> </tr> <tr> <td>Limited quantity</td> <td>Not Applicable</td> </tr> </table>	Special provisions	Not Applicable	Limited quantity	Not Applicable
Special provisions	Not Applicable				
Limited quantity	Not Applicable				

Air transport (ICAO-IATA / DGR)

14.1. UN number	1950														
14.2. Packing group	Not Applicable														
14.3. UN proper shipping name	Aerosols, flammable														
14.4. Environmental hazard	No relevant data														
14.5. Transport hazard class(es)	<table border="1"> <tr> <td>ICAO/IATA Class</td> <td>2.1</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>10L</td> </tr> </table>	ICAO/IATA Class	2.1	ICAO / IATA Subrisk	Not Applicable	ERG Code	10L								
ICAO/IATA Class	2.1														
ICAO / IATA Subrisk	Not Applicable														
ERG Code	10L														
14.6. Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>A145A167A802; A1A145A167A802</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>203</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>150 kg</td> </tr> <tr> <td>Passenger and Cargo Packing Instructions</td> <td>203; Forbidden</td> </tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td> <td>75 kg; Forbidden</td> </tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td> <td>Y203; Forbidden</td> </tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td> <td>30 kg G; Forbidden</td> </tr> </table>	Special provisions	A145A167A802; A1A145A167A802	Cargo Only Packing Instructions	203	Cargo Only Maximum Qty / Pack	150 kg	Passenger and Cargo Packing Instructions	203; Forbidden	Passenger and Cargo Maximum Qty / Pack	75 kg; Forbidden	Passenger and Cargo Limited Quantity Packing Instructions	Y203; Forbidden	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G; Forbidden
Special provisions	A145A167A802; A1A145A167A802														
Cargo Only Packing Instructions	203														
Cargo Only Maximum Qty / Pack	150 kg														
Passenger and Cargo Packing Instructions	203; Forbidden														
Passenger and Cargo Maximum Qty / Pack	75 kg; Forbidden														
Passenger and Cargo Limited Quantity Packing Instructions	Y203; Forbidden														
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G; Forbidden														

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1950						
14.2. Packing group	Not Applicable						
14.3. UN proper shipping name	Aerosols, flammable						
14.4. Environmental hazard	Not Applicable						
14.5. Transport hazard class(es)	<table border="1"> <tr> <td>IMDG Class</td> <td>2.1</td> </tr> <tr> <td>IMDG Subrisk</td> <td>Not Applicable</td> </tr> </table>	IMDG Class	2.1	IMDG Subrisk	Not Applicable		
IMDG Class	2.1						
IMDG Subrisk	Not Applicable						
14.6. Special precautions for user	<table border="1"> <tr> <td>EMS Number</td> <td>F-D , S-U</td> </tr> <tr> <td>Special provisions</td> <td>63 190 277 327 344 959</td> </tr> <tr> <td>Limited Quantities</td> <td>See SP277</td> </tr> </table>	EMS Number	F-D , S-U	Special provisions	63 190 277 327 344 959	Limited Quantities	See SP277
EMS Number	F-D , S-U						
Special provisions	63 190 277 327 344 959						
Limited Quantities	See SP277						

Inland waterways transport (ADN)

14.1. UN number	1950		
14.2. Packing group	Not Applicable		
14.3. UN proper shipping name	Aerosols, flammable		
14.4. Environmental hazard	No relevant data		
14.5. Transport hazard class(es)	<table border="1"> <tr> <td>2.1</td> <td>Not Applicable</td> </tr> </table>	2.1	Not Applicable
2.1	Not Applicable		

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14.6. Special precautions for user

Classification code	Not Applicable
Limited quantity	Not Applicable
Equipment required	Not Applicable
Fire cones number	Not Applicable

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	propylene glycol monomethyl ether acetate, alpha-isomer	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	amyl methyl ketone	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

TETRAFLUOROETHANE(811-97-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Customs Inventory of Chemical Substances ECICS (English)	UK Workplace Exposure Limits (WELs)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	

ACETONE(67-64-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)
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 Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)
European Customs Inventory of Chemical Substances ECICS (English)	European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation	European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)
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) (Maltese)
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) (Polish)
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) (Portuguese)
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) (Romanian)
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) (Slovak)
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) (Slovenian)
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) (Spanish)
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) (Swedish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)	UK Workplace Exposure Limits (WELs)

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

838 Total Ground Carbon Conductive Coating (Aerosol)

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

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)

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)

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European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)

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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)

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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)

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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)

ETHANOL(64-17-5) 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)

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)

AMYL METHYL KETONE(110-43-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

838 Total Ground Carbon Conductive Coating (Aerosol)

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)
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 Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)
European Customs Inventory of Chemical Substances ECICS (English)	European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)
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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) (Romanian)
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) (Slovak)
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) (Slovenian)
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) (Spanish)
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) (Swedish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)	UK Workplace Exposure Limits (WELs)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)	

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 Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Customs Inventory of Chemical Substances ECICS (English)	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	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
tetrafluoroethane	811-97-2	Not Available	01-2119459374-33-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Press. Gas.	GHS04, Wng	H280
2	Press. Gas., Liq. Gas, STOT SE 1	GHS04, Wng, GHS08, Dgr	H280, H370

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
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
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838 Total Ground Carbon Conductive Coating (Aerosol)

propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6	607-195-00-7	01-2119475791-29-XXXX
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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
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
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.

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
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
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.

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; tetrafluoroethane; carbon black; amyl methyl ketone)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y

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Japan - ENCS	Y
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
H225	Highly flammable liquid and vapour
H226	Flammable liquid and vapour
H228	Flammable solid
H251	Self-heating; may catch fire
H280	Contains gas under pressure; may explode if heated
H301	Toxic if swallowed
H302	Harmful if swallowed
H304	May be fatal if swallowed and enters airways
H311	Toxic in contact with skin
H315	Causes skin irritation
H331	Toxic if inhaled
H332	Harmful if inhaled
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

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

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