



## Dy-Mark Grey Primer

Dy-Mark

Chemwatch: 26-2780

Version No: 11.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 4

Issue Date: 01/11/2019

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L.GHS.AUS.EN

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

#### Product Identifier

Product name	Dy-Mark Grey Primer
Synonyms	Not Available
Proper shipping name	AEROSOLS
Other means of identification	Not Available

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

Relevant identified uses	Application is by spray atomisation from a hand held aerosol pack Use according to manufacturer's directions.
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#### Details of the supplier of the safety data sheet

Registered company name	Dy-Mark
Address	89 Formation Street Wacol QLD 4076 Australia
Telephone	+61 7 3327 3004
Fax	+61 7 3327 3009
Website	<a href="http://www.dymark.com.au">http://www.dymark.com.au</a>
Email	<a href="mailto:info@dymark.com.au">info@dymark.com.au</a>

#### Emergency telephone number

Association / Organisation	Dy-Mark
Emergency telephone numbers	+61 7 3327 3099
Other emergency telephone numbers	Not Available

### SECTION 2 HAZARDS IDENTIFICATION

#### Classification of the substance or mixture

**HAZARDOUS CHEMICAL. DANGEROUS GOODS.** According to the WHS Regulations and the ADG Code.

#### CHEMWATCH HAZARD RATINGS

	Min	Max	
Flammability	4		0 = Minimum
Toxicity	2		1 = Low
Body Contact	2		2 = Moderate
Reactivity	1		3 = High
Chronic	2		4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Flammable Aerosols Category 1, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Germ cell mutagenicity Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

#### Label elements

Hazard pictogram(s)	
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Continued...

Dy-Mark Grey Primer

SIGNAL WORD	<b>DANGER</b>
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**Hazard statement(s)**

<b>H222</b>	Extremely flammable aerosol.
<b>H315</b>	Causes skin irritation.
<b>H319</b>	Causes serious eye irritation.
<b>H341</b>	Suspected of causing genetic defects.
<b>H336</b>	May cause drowsiness or dizziness.
<b>H411</b>	Toxic to aquatic life with long lasting effects.
<b>AUH044</b>	Risk of explosion if heated under confinement.

**Precautionary statement(s) Prevention**

<b>P201</b>	Obtain special instructions before use.
<b>P210</b>	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
<b>P211</b>	Do not spray on an open flame or other ignition source.
<b>P251</b>	Pressurized container: Do not pierce or burn, even after use.
<b>P271</b>	Use only outdoors or in a well-ventilated area.
<b>P281</b>	Use personal protective equipment as required.
<b>P261</b>	Avoid breathing mist/vapours/spray.
<b>P273</b>	Avoid release to the environment.
<b>P280</b>	Wear protective gloves/protective clothing/eye protection/face protection.

**Precautionary statement(s) Response**

<b>P308+P313</b>	IF exposed or concerned: Get medical advice/attention.
<b>P321</b>	Specific treatment (see advice on this label).
<b>P362</b>	Take off contaminated clothing and wash before reuse.
<b>P305+P351+P338</b>	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
<b>P312</b>	Call a POISON CENTER or doctor/physician if you feel unwell.
<b>P337+P313</b>	If eye irritation persists: Get medical advice/attention.
<b>P391</b>	Collect spillage.
<b>P302+P352</b>	IF ON SKIN: Wash with plenty of water and soap.
<b>P304+P340</b>	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
<b>P332+P313</b>	If skin irritation occurs: Get medical advice/attention.

**Precautionary statement(s) Storage**

<b>P405</b>	Store locked up.
<b>P410+P412</b>	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
<b>P403+P233</b>	Store in a well-ventilated place. Keep container tightly closed.

**Precautionary statement(s) Disposal**

<b>P501</b>	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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**SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**

**Substances**

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
1330-20-7	10-20	<u>xylene</u>
Not Available	10-20	resin, proprietary
Not Available	10-15	filler
13463-67-7	5-10	<u>titanium dioxide</u>
64742-95-6.	2-5	<u>naphtha petroleum, light aromatic solvent</u>
64-17-5	1-5	<u>ethanol</u>
7779-90-0	1-5	<u>zinc phosphate</u>
1333-86-4	0.1-0.5	<u>carbon black</u>
115-10-6	20-40	<u>dimethyl ether</u>

**SECTION 4 FIRST AID MEASURES**

**Description of first aid measures**

<b>Eye Contact</b>	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water.</li> <li>▶ 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.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Remove any adhering solids with industrial skin cleansing cream.</li> <li>▶ <b>DO NOT use solvents.</b></li> <li>▶ Seek medical attention in the event of irritation.</li> </ul>
<b>Inhalation</b>	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> <li>▶ Remove to fresh air.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ Avoid giving milk or oils.</li> <li>▶ Avoid giving alcohol.</li> <li>▶ Not considered a normal route of entry.</li> </ul>

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

Treat symptomatically.  
for lower alkyl ethers:

#### BASIC TREATMENT

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

#### ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension without signs of hypovolaemia may require vasopressors.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

#### EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Ethers may produce anion gap acidosis. Hyperventilation and bicarbonate therapy might be indicated.
- ▶ Haemodialysis might be considered in patients with impaired renal function.
- ▶ Consult a toxicologist as necessary.

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

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

For acute or short term repeated exposures to xylene:

- ▶ Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- ▶ Pulmonary absorption is rapid with about 60-65% retained at rest.
- ▶ Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO<sub>2</sub> < 50 mm Hg or pCO<sub>2</sub> > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

#### BIOLOGICAL EXPOSURE INDEX - BEI

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

Determinant	Index	Sampling Time	Comments
Methylhippuric acids in urine	1.5 gm/gm creatinine 2 mg/min	End of shift Last 4 hrs of shift	

## SECTION 5 FIREFIGHTING MEASURES

### Extinguishing media

#### SMALL FIRE:

- ▶ Water spray, dry chemical or CO<sub>2</sub>

#### LARGE FIRE:

- ▶ Water spray or fog.

## Dy-Mark Grey Primer

## Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> <li>▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result</li> </ul>
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## Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul>
Fire/Explosion Hazard	<ul style="list-style-type: none"> <li>▶ Liquid and vapour are highly flammable.</li> <li>▶ Severe fire hazard when exposed to heat or flame.</li> <li>▶ Vapour forms an explosive mixture with air.</li> <li>▶ Severe explosion hazard, in the form of vapour, when exposed to flame or spark.</li> <li>▶ Vapour may travel a considerable distance to source of ignition.</li> <li>▶ Heating may cause expansion or decomposition with violent container rupture.</li> <li>▶ Aerosol cans may explode on exposure to naked flames.</li> <li>▶ Rupturing containers may rocket and scatter burning materials.</li> <li>▶ Hazards may not be restricted to pressure effects.</li> <li>▶ May emit acrid, poisonous or corrosive fumes.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> </ul> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) other pyrolysis products typical of burning organic material.</p> <p><b>Contains low boiling substance:</b> Closed containers may rupture due to pressure buildup under fire conditions.</p>
HAZCHEM	Not Applicable

## SECTION 6 ACCIDENTAL RELEASE MEASURES

## Personal precautions, protective equipment and emergency procedures

See section 8

## Environmental precautions

See section 12

## Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Wear protective clothing, impervious gloves and safety glasses.</li> <li>▶ Shut off all possible sources of ignition and increase ventilation.</li> <li>▶ Wipe up.</li> <li>▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.</li> <li>▶ Undamaged cans should be gathered and stowed safely.</li> </ul>
Major Spills	<ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water courses</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Water spray or fog may be used to disperse / absorb vapour.</li> <li>▶ Absorb or cover spill with sand, earth, inert materials or vermiculite.</li> <li>▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.</li> <li>▶ Undamaged cans should be gathered and stowed safely.</li> <li>▶ Collect residues and seal in labelled drums for disposal.</li> </ul>

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

## SECTION 7 HANDLING AND STORAGE

## Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> <li>▶ <b>DO NOT</b> allow clothing wet with material to stay in contact with skin</li> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT</b> enter confined spaces until atmosphere has been checked.</li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT</b> eat, drink or smoke.</li> <li>▶ <b>DO NOT</b> incinerate or puncture aerosol cans.</li> <li>▶ <b>DO NOT</b> spray directly on humans, exposed food or food utensils.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> </ul>
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Dy-Mark Grey Primer

	<ul style="list-style-type: none"> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store below 38 deg. C.</li> <li>▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can</li> <li>▶ Store in original containers in approved flammable liquid storage area.</li> <li>▶ <b>DO NOT store in pits, depressions, basements or areas where vapours may be trapped.</b></li> <li>▶ No smoking, naked lights, heat or ignition sources.</li> <li>▶ Keep containers securely sealed. Contents under pressure.</li> <li>▶ Store away from incompatible materials.</li> <li>▶ Store in a cool, dry, well ventilated area.</li> <li>▶ Avoid storage at temperatures higher than 40 deg C.</li> <li>▶ Store in an upright position.</li> <li>▶ Protect containers against physical damage.</li> <li>▶ Check regularly for spills and leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>

**Conditions for safe storage, including any incompatibilities**

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Aerosol dispenser.</li> <li>▶ Check that containers are clearly labelled.</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid reaction with oxidising agents</li> </ul>

**SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION**

**Control parameters**

**OCCUPATIONAL EXPOSURE LIMITS (OEL)**

**INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ethanol	Ethyl alcohol	1000 ppm / 1880 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dimethyl ether	Dimethyl ether	400 ppm / 760 mg/m3	950 mg/m3 / 500 ppm	Not Available	Not Available

**EMERGENCY LIMITS**

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
xylene	Xylenes	Not Available	Not Available	Not Available
titanium dioxide	Titanium oxide; (Titanium dioxide)	30 mg/m3	330 mg/m3	2,000 mg/m3
naphtha petroleum, light aromatic solvent	Naphtha (coal tar); includes solvent naphtha, petroleum (64742-88-7), naphtha (petroleum) light aliphatic, rubber solvent (64742-89-8), heavy catalytic cracked (64741-54-4), light straight run (64741-46-4), heavy aliphatic solvent (64742-96-7), high flash aromatic and aromatic solvent naphtha (64742-95-6)	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
ethanol	Ethanol: (Ethyl alcohol)	Not Available	Not Available	15000* ppm
zinc phosphate	Zinc phosphate (3:2)	12 mg/m3	36 mg/m3	220 mg/m3
carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3
dimethyl ether	Methyl ether; (Dimethyl ether)	3,000 ppm	3800* ppm	7200* ppm

Ingredient	Original IDLH	Revised IDLH
xylene	900 ppm	Not Available
titanium dioxide	5,000 mg/m3	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
ethanol	3,300 ppm	Not Available
zinc phosphate	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
dimethyl ether	Not Available	Not Available

**MATERIAL DATA**

For ethanol:

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

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

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

for dimethyl ether:

The no-effect-level for dimethyl ether is somewhere between 2000 ppm (rabbits) and 50,000 ppm (humans) with possible cardiac sensitisation occurring around 200,000 ppm (dogs). The AIHA has adopted a safety factor of 100 in respect to the 50,000 ppm level in its recommendation for a workplace environmental exposure level (WEEL) which is thought to protect against both narcotic and sensitising effects. This level is consistent with the TLV-TWA of 400 ppm for diethyl ether and should be easily achievable using current technologies. The use of the traditionally allowable excursion of 1.25 to the level of 6.25 ppm is felt to be more than adequate as an upper safe limit of exposure.

Human data:

50,000 ppm (12 mins): Feelings of mild intoxication.

75,000 ppm (12 mins): As above plus slight lack of attenuation.

82,000 ppm (12 mins): Some incoordination, slight blurring of vision

(30 mins): As above plus analgesia of the face and rushing of blood to the face.

100,000 ppm (10-20 mins): Narcotic symptoms; (64 mins): Sickness (assumed to be nausea)

144,000 ppm (36 mins): Unconsciousness

For trimethyl benzene as mixed isomers (of unstated proportions)

Odour Threshold Value: 2.4 ppm (detection)

Use care in interpreting effects as a single isomer or other isomer mix. Trimethylbenzene is an eye, nose and respiratory irritant. High concentrations cause central nervous system depression. Exposed workers show CNS changes, asthmatic bronchitis and blood dyscrasias at 60 ppm. The TLV-TWA is thought to be protective against the significant risk of CNS excitation, asthmatic bronchitis and blood dyscrasias associated with exposures above the limit.

Odour Safety Factor (OSF)

OSF=10 (1,2,4-TRIMETHYLBENZENE)

Animals exposed by inhalation to 10 mg/m<sup>3</sup> titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

for xylenes:

IDLH Level: 900 ppm

Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)

NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).

Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.

Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially contribute to the dose received by inhalation.

Odour Safety Factor(OSF)

OSF=4 (XYLENE)

NOTE H: Special requirements exist in relation to classification and labelling of this substance. This note applies to certain coal- and oil -derived substances and to certain entries for groups of substances in Annex VI. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

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

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

## Exposure controls

<b>Appropriate engineering controls</b>	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.										
	<table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 70%;">Type of Contaminant:</th> <th style="width: 30%;">Speed:</th> </tr> </thead> <tbody> <tr> <td>aerosols, (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </tbody> </table>		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.)			
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4: Large hood or large air mass in motion	4: Small hood-local control only										
Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.											

## Dy-Mark Grey Primer

<b>Personal protection</b>	
<b>Eye and face protection</b>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ 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]</li> </ul>
<b>Skin protection</b>	See Hand protection below
<b>Hands/feet protection</b>	<ul style="list-style-type: none"> <li>▶ No special equipment needed when handling small quantities.</li> <li>▶ <b>OTHERWISE:</b></li> <li>▶ For potentially moderate exposures:</li> <li>▶ Wear general protective gloves, eg. light weight rubber gloves.</li> <li>▶ For potentially heavy exposures:</li> <li>▶ Wear chemical protective gloves, eg. PVC. and safety footwear.</li> </ul>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<p>No special equipment needed when handling small quantities.</p> <p><b>OTHERWISE:</b></p> <ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eyewash unit.</li> <li>▶ Do not spray on hot surfaces.</li> <li>▶ 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.</li> <li>▶ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost.</li> </ul> <p>BREThERICK: Handbook of Reactive Chemical Hazards.</p>

## 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:

Dy-Mark Grey Primer

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
TEFLON	C
VITON	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

**NOTE:** As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

\* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

## Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

## Information on basic physical and chemical properties

## Dy-Mark Grey Primer

<b>Appearance</b>	Grey coloured flammable aerosol; not miscible with water. Supplied as an aerosol pack. Contents under <b>PRESSURE</b> . Contains highly flammable hydrocarbon propellant.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	Not Available
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	-81 (propellant)	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	HIGHLY FLAMMABLE.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	>50
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Applicable
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available

## SECTION 10 STABILITY AND REACTIVITY

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>▶ Elevated temperatures.</li> <li>▶ Presence of open flame.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 TOXICOLOGICAL INFORMATION

## Information on toxicological effects

<b>Inhaled</b>	<p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>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>Common, generalised symptoms associated with toxic gas inhalation include:</p> <ul style="list-style-type: none"> <li>▶ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures;</li> <li>▶ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest;</li> <li>▶ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest;</li> <li>▶ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain.</li> </ul> <p>A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias. High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement.</p> <p>Ethers produce narcosis following inhalation.</p> <p>Inhalation of lower alkyl ethers may result in central nervous system depression or stimulation, intoxication, headache, dizziness, weakness,</p>
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## Dy-Mark Grey Primer

	<p>blurred vision, seizures and possible coma. Cardiovascular involvement may produce hypotension, bradycardia and cardiovascular collapse, whilst respiratory symptoms might include irritation of nose and throat, cough, laryngeal spasm, pharyngitis, irregular respiration, depression, pulmonary oedema and respiratory arrest. Nausea, vomiting and salivation might also indicate overexposure.</p> <p>Convulsions, respiratory distress or paralysis, asphyxia, pneumonitis, and unconsciousness are all serious manifestations of poisoning. Fatalities have been reported. Kidney and liver damage with interstitial cystitis may result from massive exposures.</p> <p>Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p> <p><b>WARNING: intentional misuse by concentrating/inhaling contents may be lethal.</b></p> <p>Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue.</p> <p>Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>The primary physiological effect which follows exposure to diethyl ether is acute narcosis.</p> <p>Inhalation at about 7.5%, in air, produces mild intoxication in about 12 minutes. Longer exposures and exposure to higher concentrations produces incoordination, blurring of vision, headache, dizziness and unconsciousness (20% produces unconsciousness in about 20 minutes). Heavy exposures may be lethal and deaths occur due to depression of the respiratory system. Dimethyl ether is a weak cardiac sensitiser in dogs.</p>								
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Ingestion of ethanol may produce nausea, vomiting, gastrointestinal bleeding, abdominal pain and diarrhoea. Systemic effects:</p> <table border="1" data-bbox="384 904 1489 1279"> <thead> <tr> <th>Blood concentration:</th> <th>Effects:</th> </tr> </thead> <tbody> <tr> <td>&lt;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</p> <p>Ingestion of alkyl ethers may produce symptoms similar to those produced following inhalation.</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 with the material may be harmful; systemic effects may result following absorption.</p> <p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Spray mist may produce discomfort</p> <p>Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p>								
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <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.</p> <p>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>Eye contact with alkyl ethers (vapours or liquid) may produce irritation, redness and lachrymation.</p>								
Chronic	<p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</p> <p>Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents.</p> <p>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</p>								

	<p>deficiency, behavioural disorders and reduced head size.</p> <p>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).</p> <p>(1) Boehncke W.H., &amp; H.Gall, Clinical &amp; Experimental Allergy, 26, 1089-1091, 1996</p> <p>Principal route of occupational exposure to the gas is by inhalation.</p> <p>Chronic exposure to alkyl ethers may result in loss of appetite, excessive thirst, fatigue, and weight loss</p> <p>Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.</p> <p>Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.</p> <p>Xylene has been classed as a developmental toxin in some jurisdictions.</p> <p>Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.</p> <p>Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).</p> <p>Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]</p>
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	TOXICITY	IRRITATION
<b>Dy-Mark Grey Primer</b>	Not Available	Not Available
<b>xylene</b>	Dermal (rabbit) LD50: >1700 mg/kg <sup>[2]</sup>	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 4994.295 mg/l/4h <sup>[2]</sup>	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 3523-8700 mg/kg <sup>[2]</sup>	Eye (rabbit): 87 mg mild
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit):500 mg/24h moderate
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
<b>titanium dioxide</b>	dermal (hamster) LD50: >=10000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Skin (human): 0.3 mg /3D (int)-mild *
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>naphtha petroleum, light aromatic solvent</b>	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation (rat) LC50: >7331.62506 mg/l/8h <sup>[2]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (rat) LD50: >4500 mg/kg <sup>[1]</sup>	
<b>ethanol</b>	Inhalation (rat) LC50: 124.7 mg/l/4H <sup>[2]</sup>	Eye (rabbit): 500 mg SEVERE
	Oral (rat) LD50: =1501 mg/kg <sup>[2]</sup>	Eye (rabbit):100mg/24hr-moderate
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit):20 mg/24hr-moderate
		Skin (rabbit):400 mg (open)-mild
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>zinc phosphate</b>	Oral (rat) LD50: >5000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>carbon black</b>	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (rat) LD50: >15400 mg/kg <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>dimethyl ether</b>	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 309 mg/l/4H <sup>[2]</sup>	Not Available

**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

**XYLENE** Reproductive effector in rats

Continued...

	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The substance is classified by IARC as Group 3: <b>NOT</b> classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
<p style="text-align: center;"><b>TITANIUM DIOXIDE</b></p>	<p>* IUCLID</p> <p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p> <p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>For titanium dioxide:</p> <p>Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.</p> <p>Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.</p> <p>Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.</p> <p>Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.</p> <p><b>Animal carcinogenicity data</b></p> <p>Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.</p> <p>In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.</p> <p>Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.</p> <p>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<p style="text-align: center;"><b>NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT</b></p>	<p>Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe]</p> <p>For Low Boiling Point Naphthas (LBPNS):</p> <p><b>Acute toxicity:</b></p> <p>LBPNS generally have low acute toxicity by the oral (median lethal dose [LD50] in rats &gt; 2000 mg/kg-bw), inhalation (LD50 in rats &gt; 5000 mg/m3) and dermal (LD50 in rabbits &gt; 2000 mg/kg-bw) routes of exposure</p> <p>Most LBPNS are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices.</p> <p><b>Sensitisation:</b></p> <p>LBPNS do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies</p> <p><b>Repeat dose toxicity:</b></p> <p>The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPNS substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNS in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNS, were considered species- and sex-specific. These effects were determined to be due to a</p>

## Dy-Mark Grey Primer

mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.

Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNS. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m<sup>3</sup>, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m<sup>3</sup>

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

No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNS and very few non-cancer chronic toxicity studies were identified for other LBPNS. An LOAEC of 200 mg/m<sup>3</sup> was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m<sup>3</sup>, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m<sup>3</sup>). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m<sup>3</sup>

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

**Genotoxicity:**

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

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

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

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

**Carcinogenicity:**

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

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

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

Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNS. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals' lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light

straight-run naphtha and naphtha. Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha

or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.

**Reproductive/ Developmental toxicity:**

No reproductive or developmental toxicity was observed for the majority of LBPNS substances evaluated. Most of these studies were carried out by inhalation exposure in rodents.

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

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

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

Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of

n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.

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

For trimethylbenzenes:

Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption. 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid. The major routes of excretion of 1,2,4-trimethylbenzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.

**Acute Toxicity** Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis. High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness. The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes. Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg). Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels. No effects were reported for rats exposed to a mixture of trimethylbenzenes at 1700 ppm for 10 to 21 days.

**Neurotoxicity** 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes.

Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.

**Subchronic/Chronic Toxicity** Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene.

Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia.

**Genotoxicity:** Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella typhimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation.

**Developmental/Reproductive Toxicity:** A three-generation reproductive study on the C9 fraction was conducted. CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established. Developmental toxicity, including possible developmental neurotoxicity, was evident in rats in a 3-generation reproductive study.

No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethylbenzenes, 4-6 hours/day, 5 days/week over one generation.

For C9 aromatics (typically trimethylbenzenes - TMBs)

Acute Toxicity

Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50's range from 6,000 to 10,000 mg/m<sup>3</sup> for C9 aromatic naphtha and 18,000 to 24,000 mg/m<sup>3</sup> for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 mL/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.

Irritation and Sensitization

Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.

Repeated Dose Toxicity

**Inhalation:** The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m<sup>3</sup>). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neurobehavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m<sup>3</sup>, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m<sup>3</sup>) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.

The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m<sup>3</sup>. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4- and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m<sup>3</sup>). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m<sup>3</sup> (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m<sup>3</sup> for respiratory irritation and 250 ppm or 1230 mg/m<sup>3</sup> for systemic effects.

**Oral:** The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated

with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg-bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

#### Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with *Salmonella typhimurium* and *Escherichia coli* bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m<sup>3</sup>) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic

#### Hydrocarbon Solvents Category

##### Reproductive and Developmental Toxicity

Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m<sup>3</sup>, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex/group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex/group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.

##### Systemic Effects on Parental Generations:

The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m<sup>3</sup>).

**Reproductive Toxicity-Effects on Parental Generations:** There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m<sup>3</sup>). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m<sup>3</sup>), which excludes analysis of the highest concentration due to excessive mortality.

**Developmental Toxicity - Effects on Pups:** Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m<sup>3</sup>) based on the body weights reductions observed in the F3 offspring.

**Conclusion:** No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.

for petroleum:

Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline

This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.

This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.

This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents

**Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.

**Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.

**Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.

**Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.

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

#### CARBON BLACK

Inhalation (rat) TCLo: 50 mg/m<sup>3</sup>/6h/90D-I Nil reported

#### XYLENE & 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.

#### TITANIUM DIOXIDE & 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.

Dy-Mark Grey Primer

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

Legend: ✗ – Data either not available or does not fill the criteria for classification  
✓ – Data available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Dy-Mark Grey Primer	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

  

xylene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	2.6mg/L	2
	EC50	48	Crustacea	1.8mg/L	2
	EC50	72	Algae or other aquatic plants	3.2mg/L	2
	NOEC	73	Algae or other aquatic plants	0.44mg/L	2

  

titanium dioxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>1-mg/L	2
	EC50	48	Crustacea	>1-mg/L	2
	EC50	72	Algae or other aquatic plants	5.83mg/L	4
NOEC	336	Fish	0.089mg/L	4	

  

naphtha petroleum, light aromatic solvent	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	4.1mg/L	2
	EC50	48	Crustacea	3.2mg/L	2
	EC50	72	Algae or other aquatic plants	>1-mg/L	2
NOEC	72	Algae or other aquatic plants	=1mg/L	1	

  

ethanol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	11-mg/L	2
	EC50	48	Crustacea	2mg/L	4
	EC50	96	Algae or other aquatic plants	17.921mg/L	4
NOEC	2016	Fish	0.000375mg/L	4	

  

zinc phosphate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.58mg/L	2
	EC50	48	Crustacea	0.001-0.833mg/L	2
NOEC	72	Algae or other aquatic plants	0.00038608mg/L	2	

  

carbon black	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>10-mg/L	2
	EC10	72	Algae or other aquatic plants	>10-mg/L	2
NOEC	96	Fish	>=1-mg/L	2	

  

dimethyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-783.04mg/L	2
	EC50	48	Crustacea	>4400.0mg/L	2
	EC50	96	Algae or other aquatic plants	154.917mg/L	2
NOEC	48	Crustacea	>4000mg/L	1	

Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

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

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

Continued...

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

For aromatic hydrocarbons:

Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (*Palaemonetes pugio*) and brown shrimp (*Penaeus aztecus*) was dimethylnaphthalenes > methylnaphthalenes > naphthalenes.

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

Volatile furandiones and aldehydes are significant atmospheric oxidation products of aromatic compounds. Highly acidic dicarboxylic acids produced by the reactions between furandiones and water were shown to rapidly acidify an aqueous phase

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

Ethanol does not bioaccumulate to an appreciable extent.

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

#### Environmental Fate:

**TERRESTRIAL FATE:** An estimated Koc value of 1 indicates that ethanol is expected to have very high mobility in soil. Volatilisation of ethanol from moist soil surfaces is expected to be an important fate process given a Henry's Law constant of 5X10<sup>-6</sup> atm-m<sup>3</sup>/mole. The potential for volatilisation of ethanol from dry soil surfaces may exist based upon an extrapolated vapor pressure of 59.3 mmHg. Biodegradation is expected to be an important fate process for ethanol based on half-lives on the order of a few days for ethanol in sandy soil/groundwater microcosms.

**AQUATIC FATE:** An estimated Koc value of 1 indicates that ethanol is not expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon a Henry's Law constant of 5X10<sup>-6</sup> atm-m<sup>3</sup>/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 sunlit 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 3.3X10<sup>-12</sup> m<sup>3</sup>/molecule-sec at 25 deg C.

#### Ecotoxicity:

log Kow: -0.31- -0.32

Half-life (hr) air: 144

Half-life (hr) H<sub>2</sub>O surface water: 144

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

BOD 5 if unstated: 0.93-1.67,63%

COD: 1.99-2.11,97%

ThOD: 2.1

For C9 aromatics (typically trimethylbenzene - TMBs)

Chemicals in this category possess properties indicating a hazard for the environment (acute toxicity for fish, invertebrates, and algae from 1 to 10 mg/L). Category members are readily biodegradable, except 1,3,5-trimethylbenzene (CAS RN 108-67-8). Category members are not expected to be bioaccumulative.

Environmental Fate:

In the air, category member constituents have the potential to rapidly degrade through indirect photolytic processes mediated primarily by hydroxyl radicals with calculated degradation half-lives ranging from 0.54 to 2.81 days (based on a 12-hour day and a hydroxyl radical concentration of 5x10<sup>+5</sup>). Aqueous photolysis and hydrolysis will not contribute to the transformation of category chemical constituents in aquatic environments because they are either poorly reactive or not susceptible to these reactions.

Results of the Mackay Level I environmental distribution model show that chemical constituents of C9 Aromatic Hydrocarbon Solvents Category members have the potential to partition to air (96.8 to 98.9 %), with a negligible amount partitioning to water (0.2 to 0.6%) and soil (0.9 to 2.7%). In comparison, Level III modeling indicates that category members partition primarily to soil (66.3 to 79.6%) and water (17.8 to 25.0%) compartments rather than air (2.4 to 8.4%) when an equal emission rate (1000 kg/hr) is assumed to each of the air, water, and soil compartments. When release (1000 kg/hr) is modeled only to either the air, water, or soil compartment, constituents are indicated in the modeling to partition primarily (>94%) to the compartment to which they are emitted as advection and degradation influence constituent concentration in compartments to which constituents are not released. Solvent naphtha, (pet.), light aromatic (CAS RN 64742-95-6), 1,2,4-trimethylbenzene (CAS RN 95-63-6), and 1-ethyl-3-methylbenzene (CAS RN 620-14-4) were determined to be readily biodegradable based on the studies that used the TG OECD 301F (the latter substance is used to characterize the potential biodegradability of the category member, ethylmethylbenzene (CAS RN 25550-14-5)). These three substances exceed 60%

biodegradation in 28 days and met the 10-day window criterion for ready biodegradation. In comparison 1,3,5-trimethylbenzene (CAS RN 108-67-8) was not readily biodegradable. It achieved 42% biodegradation after 28 days and 60% biodegradation after 39 days. The result for the multi-constituent substance (CAS RN 64742-95-6), a UVCB, characterizes the biodegradability of that substance as a whole, but it does not suggest that each constituent is equally biodegradable. As with all ready biodegradation test guidelines, the test system and study design used with these substances (OECD TG 301F) is not capable of distinguishing the relative contribution of the substances' constituents to the total biodegradation measured.

Based on Henry's Law constants (HLCs) representing a potential to volatilize from water that range from 590 to 1000 Pa-m<sup>3</sup>/mole, the potential to volatilize from surface waters for chemicals in the C9 Aromatic Hydrocarbon Solvents Category is expected to be high.

Based on the measured bioconcentration factors that range from 23 to 342 for 1,2,4-trimethylbenzene and 1,3,5-trimethylbenzene, the category members are not expected to be bioaccumulative.

Ecotoxicity

Acute toxicity values used to characterize this category for fish (LL50; LC50) and invertebrates (EL50; EC50) range from 3.5 to 9.2 mg/L, based on measured data. For algae, one study for a category member (CAS RN 64742-95-6) resulted in a 72-hr EC50 of 2.4 mg/L (biomass) and 2.7 mg/L (growth rate) based on measured concentrations.

The algal 72-hour NOEC (no observed effect concentration) for biomass and growth rate is 1.3 mg/L, based on mean measured concentrations. A 21-day *Daphnia magna* reproduction study with 1,3,5-trimethylbenzene (CAS RN 108-67-8) resulted in a NOEC value of 0.4 mg/L, based on a minimum measured value.

for Petroleum Hydrocarbon Gases:

**Environmental fate:**

The environmental fate characteristics of petroleum hydrocarbon gases are governed by these physical-chemical attributes. All components of these gases will partition to the air where interaction with hydroxyl radicals is an important fate process. Hydrocarbons having molecular weights represented in these streams are inherently biodegradable, but their tendency to partition to the atmosphere would prevent their biotic degradation in water and soils. However, if higher molecular weight fractions of these streams enter the aquatic terrestrial environment, biodegradation may be an important fate mechanism.

The majority of components making up hydrocarbon gases typically have low melting and boiling points. They also have high vapor pressures and low octanol/water partition coefficients. The aqueous solubilities of these substances vary, and range from approximately 22 parts per million to several hundred parts per million. The environmental fate characteristics of refinery gases are governed by these physical-chemical attributes. Components of the hydrocarbon gas streams will partition to the air, and photodegradation reactions will be an important fate process for many of the hydrocarbon components. The hydrocarbons in these mixtures are inherently biodegradable, but due to their tendency to partition to the atmosphere, biodegradation is not anticipated to be an important fate mechanisms. However, if released to water or soil, some of the higher molecular weight fractions may become available for microbial attack. The inorganic gases are chemically stable and may be lost to the atmosphere or simply become involved in the environmental recycling of their atoms. Some show substantial water solubility, but their volatility eventually causes these gases to enter the atmosphere.

Substances in Refinery Gases that volatilise to air may undergo a gas-phase oxidation reaction with photochemically produced hydroxyl radicals (OH-). Atmospheric oxidation as a result of hydroxyl radical attack is not direct photochemical degradation, but rather indirect degradation. Indirect photodegradation of the hydrocarbon components in Refinery Gases can be an important fate process for these constituents. In general, half lives decrease with increasing carbon chain length. Half lives for this fraction of Refinery Gases ranged from 960 days (methane) to 0.16 days (butadiene). The constituents of the C5- C6 hydrocarbon fraction have photodegradation half-lives of approximately two days.

The hydrocarbon and non-hydrocarbon constituents in Refinery Gases do not contain the functional groups or chemical linkages known to undergo hydrolysis reactions. Therefore hydrolysis will not play an important role in the environmental fate for the components in Refinery Gas streams.

Biodegradation of the hydrocarbon components in refinery gases may occur in soil and water. Gaseous hydrocarbons are widespread in nature and numerous types of microbes have

evolved which are capable of oxidizing these substances as their sole energy source. Although volatilization is the predominant behavior for these gases, sufficient aqueous solubility and bioavailability is exhibited by these compounds. The use of gaseous carbon sources for cell growth is common among autotrophic organisms. Higher chain length hydrocarbons typical of naphtha streams also are known to inherently biodegrade in the environment

#### Ecotoxicity:

Acute LC/EC50 values for the hydrocarbon components of these gas streams ranged roughly from 1 to 100 mg/L.

Although the LC/EC50 data for the individual gases illustrate the potential toxicity to aquatic organisms, aqueous concentrations from releases of these gases would likely not persist in the aquatic environment for a sufficient duration to elicit toxicity. Based on a simple conceptual exposure model analysis, emissions of petroleum hydrocarbon gases to the atmosphere would not likely result in acutely toxic concentrations in adjacent water bodies because such emissions will tend to remain in the atmosphere.

Several of the constituents in refinery gases were shown to be highly hazardous to aquatic organisms in laboratory toxicity tests where exposure concentrations can be maintained over time. Hydrogen sulfide was shown to be the most toxic constituent to fish (LC50 ranged 0.007 to 0.2 mg/L) and invertebrates (EC50 ranged 0.022 to 1.07 mg/L), although several LC/EC50 values for ammonia also were below 1 mg/l for these organisms (0.083 to 4.6 mg/L and 0.53 to 22.8 mg/L, respectively).

Most ethers are very resistant to hydrolysis, and the rate of cleavage of the carbon-oxygen bond by abiotic processes is expected to be insignificant.

Direct photolysis will not be an important removal process since aliphatic ethers do not absorb light at wavelengths >290 nm

For xylenes:

log Koc : 2.05-3.08

Koc : 25.4-204

Half-life (hr) air : 0.24-42

Half-life (hr) H2O surface water : 24-672

Half-life (hr) H2O ground : 336-8640

Half-life (hr) soil : 52-672

Henry's Pa m<sup>3</sup>/mol: 637-879

Henry's atm m<sup>3</sup>/mol: 7.68E-03

BOD 5 if unstated: 1.4,1%

COD : 2.56,13%

ThOD : 3.125

BCF : 23

log BCF : 1.17-2.41

#### Environmental Fate

**Terrestrial fate:** Measured Koc values of 166 and 182, indicate that 3-xylene is expected to have moderate mobility in soil. Volatilisation of p-xylene is expected to be important from moist soil surfaces given a measured Henry's Law constant of 7.18x10<sup>-3</sup> atm-cu cm/mole. The potential for volatilisation of 3-xylene from dry soil surfaces may exist based on a measured vapor pressure of 8.29 mm Hg. p-Xylene may be degraded during its passage through soil). The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. p-Xylene, present in soil samples contaminated with jet fuel, was completely degraded aerobically within 5 days. In aquifer studies under anaerobic conditions, p-xylene was degraded, usually within several weeks, with the production of 3-methylbenzylfumaric acid, 3-methylbenzylsuccinic acid, 3-methylbenzoate, and 3-methylbenzaldehyde as metabolites.

**Aquatic fate:** Koc values indicate that p-xylene may adsorb to suspended solids and sediment in water. p-Xylene is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. BCF values of 14.8, 23.4, and 6, measured in goldfish, eels, and clams, respectively, indicate that bioconcentration in aquatic organisms is low. p-Xylene in water with added humic substances was 50% degraded following 3 hours irradiation suggesting that indirect photooxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. Although p-xylene is biodegradable and has been observed to degrade in pond water, there are insufficient data to assess the rate of this process in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater in several studies; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high.

#### Atmospheric fate:

Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere primarily by reaction with photochemically-produced hydroxyl radicals, with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylenes' susceptibility to photochemical oxidation in the troposphere is to the extent that they may contribute to photochemical smog formation.

According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and from its vapour pressure, p-xylene, is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase p-xylene is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 16 hours. A half-life of 1.0 hr in summer and 10 hr in winter was measured for the reaction of p-xylene with photochemically-produced hydroxyl radicals.

p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers, with loss rates varying from 9-42% per hr. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzyl nitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

#### Ecotoxicity:

for xylenes

Fish LC50 (96 h) Pimephales promelas 13.4 mg/l; Oncorhynchus mykiss 8.05 mg/l; Lepomis macrochirus 16.1 mg/l (all flow through values); Pimephales promelas 26.7 (static)

Daphnia EC50 948 h): 3.83 mg/l

Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/l

Gammarus lacustris LC50 (48 h): 0.6 mg/l

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

#### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
titanium dioxide	HIGH	HIGH
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
dimethyl ether	LOW	LOW

#### Bioaccumulative potential

Ingredient	Bioaccumulation
xylene	MEDIUM (BCF = 740)
titanium dioxide	LOW (BCF = 10)
ethanol	LOW (LogKOW = -0.31)
dimethyl ether	LOW (LogKOW = 0.1)

#### Mobility in soil

Ingredient	Mobility
titanium dioxide	LOW (KOC = 23.74)
ethanol	HIGH (KOC = 1)
dimethyl ether	HIGH (KOC = 1.292)

## SECTION 13 DISPOSAL CONSIDERATIONS

### Waste treatment methods

<b>Product / Packaging disposal</b>	<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"> <li>▶ Reduction</li> <li>▶ Reuse</li> <li>▶ Recycling</li> <li>▶ Disposal (if all else fails)</li> </ul> <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"> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Consult State Land Waste Management Authority for disposal.</li> <li>▶ Discharge contents of damaged aerosol cans at an approved site.</li> <li>▶ Allow small quantities to evaporate.</li> <li>▶ <b>DO NOT incinerate or puncture aerosol cans.</b></li> <li>▶ Bury residues and emptied aerosol cans at an approved site.</li> </ul>
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## SECTION 14 TRANSPORT INFORMATION

### Labels Required

	
<b>Marine Pollutant</b>	
<b>HAZCHEM</b>	Not Applicable

### Land transport (ADG)

<b>UN number</b>	1950
<b>UN proper shipping name</b>	AEROSOLS
<b>Transport hazard class(es)</b>	Class : 2.1 Subrisk : Not Applicable
<b>Packing group</b>	Not Applicable
<b>Environmental hazard</b>	Environmentally hazardous
<b>Special precautions for user</b>	Special provisions : 63 190 277 327 344 381 Limited quantity : 1000ml

### Air transport (ICAO-IATA / DGR)

<b>UN number</b>	1950
<b>UN proper shipping name</b>	Aerosols, flammable (engine starting fluid); Aerosols, flammable
<b>Transport hazard class(es)</b>	ICAO/IATA Class : 2.1 ICAO / IATA Subrisk : Not Applicable ERG Code : 10L
<b>Packing group</b>	Not Applicable
<b>Environmental hazard</b>	Environmentally hazardous
<b>Special precautions for user</b>	Special provisions : A145 A167 A802; A1 A145 A167 A802 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)**

<b>UN number</b>	1950	
<b>UN proper shipping name</b>	AEROSOLS	
<b>Transport hazard class(es)</b>	IMDG Class	2.1
	IMDG Subrisk	Not Applicable
<b>Packing group</b>	Not Applicable	
<b>Environmental hazard</b>	Marine Pollutant	
<b>Special precautions for user</b>	EMS Number	F-D , S-U
	Special provisions	63 190 277 327 344 381 959
	Limited Quantities	1000 ml

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

Not Applicable

**SECTION 15 REGULATORY INFORMATION****Safety, health and environmental regulations / legislation specific for the substance or mixture****XYLENE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

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

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**TITANIUM DIOXIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)  
 Chemical Footprint Project - Chemicals of High Concern List  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B : Possibly carcinogenic to humans  
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5  
 Chemical Footprint Project - Chemicals of High Concern List

**ETHANOL IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

**ZINC PHOSPHATE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

**CARBON BLACK IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australia Inventory of Chemical Substances (AICS)  
 Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B : Possibly carcinogenic to humans  
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**DIMETHYL ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

**National Inventory Status**

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (xylene; dimethyl ether; naphtha petroleum, light aromatic solvent; ethanol; carbon black)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (zinc phosphate)

Vietnam - NCI	Yes
Russia - ARIPS	Yes
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

## SECTION 16 OTHER INFORMATION

<b>Revision Date</b>	01/11/2019
<b>Initial Date</b>	01/11/2009

### SDS Version Summary

Version	Issue Date	Sections Updated
10.1.1.1	26/07/2016	Acute Health (skin), Chronic Health, Classification
11.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

### Other information

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

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

### Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average  
 PC—STEL: Permissible Concentration-Short Term Exposure Limit  
 IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit.  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 OSF: Odour Safety Factor  
 NOAEL :No Observed Adverse Effect Level  
 LOAEL: Lowest Observed Adverse Effect Level  
 TLV: Threshold Limit Value  
 LOD: Limit Of Detection  
 OTV: Odour Threshold Value  
 BCF: BioConcentration Factors  
 BEI: Biological Exposure Index

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