



Dy-Mark BondCover Touch Up Aerosol All Colours

Dy-Mark

Chemwatch: 85-7415

Version No: 5.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 01/11/2019

Print Date: 06/04/2020

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

Product Identifier

Product name	Dy-Mark BondCover Touch Up Aerosol All Colours
Synonyms	12533001 Dune, 12533002 Classic Cream, 12533003 Surfmist, 12533004 Woodland Grey,; 12533005 Manor Red, 12533006 Deep Ocean, 12533007 Cottage Green, 12533008 Wallaby,; 12533009 Terrain, 12533010 Pale Eucalypt, 12533011 Mangrove, 12533012 Paperbark,; 12533013 Gully, 12533014 Cove, 12533015 Basalt, 12533016 Evening Haze, 12533017 Shale Grey,; 12533018 Night Sky, 12533019 Windspray, 12533020 Ironstone, 12533021 Jasper,; 12533022 Monument
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	http://www.dymark.com.au
Email	info@dymark.com.au

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	3		
Toxicity	2		
Body Contact	2		
Reactivity	1		
Chronic	3		

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Flammable Aerosols Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Carcinogenicity Category 2, Reproductive Toxicity Category 1B, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Specific target organ toxicity - repeated exposure Category 2, Aspiration Hazard Category 1, Acute 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

Dy-Mark BondCover Touch Up Aerosol All Colours

Hazard pictogram(s)	
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SIGNAL WORD	DANGER
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Hazard statement(s)

H222	Extremely flammable aerosol.
H302	Harmful if swallowed.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H351	Suspected of causing cancer.
H360FD	May damage fertility. May damage the unborn child.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H373	May cause damage to organs through prolonged or repeated exposure.
H304	May be fatal if swallowed and enters airways.
H401	Toxic to aquatic life.
AUH044	Risk of explosion if heated under confinement.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Pressurized container: Do not pierce or burn, even after use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P321	Specific treatment (see advice on this label).
P331	Do NOT induce vomiting.
P362	Take off contaminated clothing and wash before reuse.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container 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
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Dy-Mark BondCover Touch Up Aerosol All Colours

108-88-3	10-30	<u>toluene</u>
1330-20-7	10-30	<u>xylene</u>
Not Available	1-10	pigments determined not to be hazardous
117-81-7	1-10	<u>di-sec-octyl phthalate</u>
100-41-4	1-5	<u>ethylbenzene</u>
68476-85-7.	10-30	<u>hydrocarbon propellant</u>
115-10-6	10-30	<u>dimethyl ether</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

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

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

Following acute or short term repeated exposures to toluene:

- ▶ Toluene is absorbed across the alveolar barrier, the blood/air mixture being 11.2/15.6 (at 37 degrees C.) The concentration of toluene, in expired breath, is of the order of 18 ppm following sustained exposure to 100 ppm. The tissue/blood proportion is 1/3 except in adipose where the proportion is 8/10.
- ▶ Metabolism by microsomal mono-oxygenation, results in the production of hippuric acid. This may be detected in the urine in amounts between 0.5 and 2.5 g/24 hr which represents, on average 0.8 gm/gm of creatinine. The biological half-life of hippuric acid is in the order of 1-2 hours.
- ▶ Primary threat to life from ingestion and/or inhalation is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (eg cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 <50 mm Hg or pCO2 > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial damage has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.

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Dy-Mark BondCover Touch Up Aerosol All Colours

- ▶ Epinephrine (adrenaline) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- ▶ Lavage is indicated in patients who require decontamination; ensure use.

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

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

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

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 (pO₂ < 50 mm Hg or pCO₂ > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial 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
Methylhippu-ric 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 CO₂

LARGE FIRE:

- ▶ Water spray or fog.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

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

Dy-Mark BondCover Touch Up Aerosol All Colours

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Wear protective clothing, impervious gloves and safety glasses. ▶ Shut off all possible sources of ignition and increase ventilation. ▶ Wipe up. ▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely. 																																																																											
Major Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse / absorb vapour. ▶ Absorb or cover spill with sand, earth, inert materials or vermiculite. ▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely. ▶ Collect residues and seal in labelled drums for disposal. <p>Chemical Class: aromatic hydrocarbons For release onto land: recommended sorbents listed in order of priority.</p> <table border="1"> <thead> <tr> <th>SORBENT TYPE</th> <th>RANK</th> <th>APPLICATION</th> <th>COLLECTION</th> <th>LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>Feathers - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>DGC, RT</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R,W,SS</td> </tr> <tr> <td>cross-linked polymer- pillow</td> <td>2</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I, P,</td> </tr> <tr> <td>treated clay/ treated natural organic - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I</td> </tr> <tr> <td>wood fibre - pillow</td> <td>4</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td colspan="5">LAND SPILL - MEDIUM</td> </tr> <tr> <td>cross-linked polymer -particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R, W, SS</td> </tr> <tr> <td>treated clay/ treated natural organic - particulate</td> <td>2</td> <td>blower</td> <td>skiploader</td> <td>R, I</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>W, SS, DGC</td> </tr> <tr> <td>feathers - pillow</td> <td>3</td> <td>throw</td> <td>skiploader</td> <td>DGC, RT</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> </tbody> </table> <p>Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988</p> <ul style="list-style-type: none"> ▶ Clear area of all unprotected personnel and move upwind. ▶ Alert Emergency Authority and advise them of the location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear full body clothing with breathing apparatus. ▶ Prevent by any means available, spillage from entering drains and water-courses. ▶ Consider evacuation. ▶ Shut off all possible sources of ignition and increase ventilation. ▶ No smoking or naked lights within area. ▶ Use extreme caution to prevent violent reaction. ▶ Stop leak only if safe to do so. ▶ Water spray or fog may be used to disperse vapour. ▶ DO NOT enter confined space where gas may have collected. ▶ Keep area clear until gas has dispersed. 	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					Feathers - pillow	1	throw	pitchfork	DGC, RT	cross-linked polymer - particulate	2	shovel	shovel	R,W,SS	cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	3	shovel	shovel	R, I, P,	treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I	wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT	LAND SPILL - MEDIUM					cross-linked polymer -particulate	1	blower	skiploader	R, W, SS	treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I	sorbent clay - particulate	3	blower	skiploader	R, I, P	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	feathers - pillow	3	throw	skiploader	DGC, RT	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
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Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly
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Dy-Mark BondCover Touch Up Aerosol All Colours

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

Conditions for safe storage, including any incompatibilities

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

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	toluene	Toluene	50 ppm / 191 mg/m3	574 mg/m3 / 150 ppm	Not Available	Not Available
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	di-sec-octyl phthalate	Di-sec-octyl phthalate	5 mg/m3	10 mg/m3	Not Available	Not Available
Australia Exposure Standards	ethylbenzene	Ethyl benzene	100 ppm / 434 mg/m3	543 mg/m3 / 125 ppm	Not Available	Not Available
Australia Exposure Standards	hydrocarbon propellant	LPG (liquified petroleum gas)	1000 ppm / 1800 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
toluene	Toluene	Not Available	Not Available	Not Available
xylene	Xylenes	Not Available	Not Available	Not Available
di-sec-octyl phthalate	Di-sec-octylphthalate	10 mg/m3	1,000 mg/m3	6,100 mg/m3
ethylbenzene	Ethyl benzene	Not Available	Not Available	Not Available
hydrocarbon propellant	Liquified petroleum gas; (L.P.G.)	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm
dimethyl ether	Methyl ether; (Dimethyl ether)	3,000 ppm	3800* ppm	7200* ppm

Ingredient	Original IDLH	Revised IDLH
toluene	500 ppm	Not Available
xylene	900 ppm	Not Available
di-sec-octyl phthalate	5,000 mg/m3	Not Available
ethylbenzene	800 ppm	Not Available
hydrocarbon propellant	2,000 ppm	Not Available
dimethyl ether	Not Available	Not Available

MATERIAL DATA

IFRA Prohibited Fragrance Substance

Continued...

Dy-Mark BondCover Touch Up Aerosol All Colours

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

For liquefied petroleum gases (LPG):

TLV TWA: 1000 ppm, 1800 mg/m³ (as LPG)

ES TWA: 1000 ppm, 1800 mg/m³ (as LPG)

OES TWA: 1000 ppm, 1750 mg/m³; STEL: 1250 ppm, 2180 mg/m³ (as LPG)

IDLH Level: 2000 ppm (lower explosive limit)

No chronic systemic effects have been reported from occupational exposure to LPG. The TLV-TWA is based on good hygiene practices and is thought to minimise the risk of fire or explosion.

Odour Safety Factor(OSF)

OSF=0.16 (hydrocarbon propellant)

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 ethyl benzene:

Odour Threshold Value: 0.46-0.60 ppm

NOTE: Detector tubes for ethylbenzene, measuring in excess of 30 ppm, are commercially available.

Ethyl benzene produces irritation of the skin and mucous membranes and appears to produce acute and chronic effects on the central nervous system. Animal experiments also suggest the effects of chronic exposure include damage to the liver, kidneys and testes. In spite of structural similarities to benzene, the material does not appear to cause damage to the haemopoietic system. The TLV-TWA is thought to be protective against skin and eye irritation. Exposure at this concentration probably will not result in systemic effects.

Subjects exposed at 200 ppm experienced transient irritation of the eyes; at 1000 ppm there was eye irritation with profuse lachrymation; at 2000 ppm eye irritation and lachrymation were immediate and severe accompanied by moderate nasal irritation, constriction in the chest and vertigo; at 5000 ppm exposure produced intolerable irritation of the eyes and throat.

Odour Safety Factor(OSF)

OSF=43 (ETHYL BENZENE)

For toluene:

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

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

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

Odour Safety Factor(OSF)

OSF=17 (TOLUENE)

for 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 K: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.1%w/w 1,3-butadiene (EINECS No 203-450-8). - European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Appropriate engineering controls	<p>CARE: Use of a quantity of this material in confined space or poorly ventilated area, where rapid build up of concentrated atmosphere may occur, could require increased ventilation and/or protective gear</p> <p>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.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p>					
	<p>General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.</p> <p>Provide adequate ventilation in warehouse or closed storage areas.</p> <p>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.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="text-align: left;">Type of Contaminant:</th> <th style="text-align: left;">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> <p>Within each range the appropriate value depends on:</p>	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)
Type of Contaminant:	Speed:					
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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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	<p>Lower end of the range</p> <p>1: Room air currents minimal or favourable to capture</p> <p>2: Contaminants of low toxicity or of nuisance value only.</p> <p>3: Intermittent, low production.</p> <p>4: Large hood or large air mass in motion</p>	<p>Upper end of the range</p> <p>1: Disturbing room air currents</p> <p>2: Contaminants of high toxicity</p> <p>3: High production, heavy use</p> <p>4: Small hood-local control only</p>
	<p>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.</p>	
Personal protection		
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] ▶ Close fitting gas tight goggles <p>DO NOT wear contact lenses.</p> <ul style="list-style-type: none"> ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 	
Skin protection	See Hand protection below	
Hands/feet protection	<ul style="list-style-type: none"> ▶ No special equipment needed when handling small quantities. ▶ OTHERWISE: ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear. 	
Body protection	See Other protection below	
Other protection	<p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces. ▶ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton. ▶ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. <p>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:

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Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C

Respiratory protection

Type AX-P 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 P2	-	AX-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AX-AUS / Class 1 P2	-
up to 100 x ES	-	AX-2 P2	AX-PAPR-2 P2 ^

^ - Full-face

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

- ▶ 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

Continued...

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

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

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

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

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation hazard is increased at higher temperatures.</p>
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	<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 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>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. WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes) Considered an unlikely route of entry in commercial/industrial environments The liquid may produce considerable gastrointestinal discomfort and may be harmful or toxic if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption. Open cuts, abraded or irritated skin should not be exposed to this material 51r38mod51ay</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. 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>
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Harmful: danger of serious damage to health by prolonged exposure through inhalation. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>2-Ethylhexanoic acid (2-EHA) its esters and its salts are of concern to human health because of their potential to induce carcinogenicity, liver toxicity and developmental/reproductive toxicity. 2-EHA is of low acute oral and dermal toxicity, is a mild skin irritant and a severe eye irritant. It is not mutagenic in Ames test, but is capable of inducing chromosome aberration and sister chromatid exchanges in vitro, liver toxicity and liver tumours after repeated dose treatment, In addition, 2-EHA has been associated with reproductive and developmental toxicity in experimental animals. 2-EHA is quickly resorbed orally, dermally and following inhalation and almost fully excreted mainly in urine. As in the case of fatty acids, degradation mainly takes place by means of peroxisomal beta-oxidation. 2-EHA has been shown to be a liver and developmental toxicant in animal studies at high doses; in developmental toxicity studies, it was postulated that the maternal liver toxicity began a cascade of effects that included metallothionein (MT) induction, zinc accumulation in the liver due to MT binding, and a resulting zinc deficiency in the developing embryo; the zinc deficiency causes the developmental toxicity; a reproductive/developmental toxicity study was also performed with up to 1% dietary di-2-ethylhexyl terephthalate (DEHT; a 2-ethylhexanoic acid precursor); no reproductive or developmental effects were observed, suggesting that the process of metabolic conversion of DEHT to 2-ethylhexanol and subsequent hydrolysis to 2-ethylhexanoic acid results in a time course of 2-ethylhexanoic acid appearance such that allows clearance before sufficient levels can arise to produce acute liver toxicity. The closely related substance, 2-ethylhexanol is metabolised to form 2-ethylhexanoic acid and so presents a similar toxicity profile.. 2-Ethylhexanoic acid (CAS No. 149-57-5) is classified as hazardous, as a Category 3 reproductive toxin, with the risk phrase 'Possible risk of harm to the unborn child' (Xn; R63). These effects were noted in the absence of signs of maternal toxicity. The lowest observed adverse effect level (LOAEL) for developmental toxicity was reported to be 100 mg/kg bw/day. Effects on fertility were also reported, with evidence being sufficient to warrant classification as potentially toxic to fertility. 2-Ethylhexanol (CAS No. 104-76-7) was reported to cause developmental toxicity, but not teratogenicity, in rats following treatment via the oral route (NICNAS a). These effects were noted in the absence of signs of marked maternal toxicity. The no observed adverse effect level (NOAEL) for developmental toxicity was reported to be 130 mg/kg bw/day. Various studies on reproduction toxicity have produced indications of an embryotoxic effect of 2-EHA. After oral administration, NOAEL values for maternal toxicity and foetotoxic effects of 2-EHA were determined in rabbits at 25 and >250 mg/kg body weight/day and in rats at 250 and 100 mg/kg body weight/day. The foetotoxic findings in rats were based on a reduced skeleton ossification at the next higher dose (250 mg/kg body weight/day). No teratogenic effects were observed in this study. In comparison with the structural isomer valproic acid, a known human teratogen, 2-EHA does have similar reprotoxic effects at maternal toxic doses in animal experiments but a far lower potency Following sub-chronic oral administration of 2-EHA, critical effects like liver changes (higher relative liver weight, histological changes in hepatocytes) were observed in rats and mice and histological renal tubule results were observed in mice. Furthermore, statistically significant, higher cholesterol values were found in all treated male rats (61, 303 and 917 mg/kg body weight/day) and in male and female mice in the middle and high dose groups (885-3139 mg/kg body weight/day). In rats the maximum dose with no adverse effect (NOAEL) was 61 mg/kg body weight/day In bacterial test systems, mutagenicity studies produced negative findings. In test systems with mammalian cells, by contrast, the findings were weakly positive. Cytogenetic and SCE studies involving CHO cells were positive, one SCE test in human lymphocytes was questionably positive and one experiment concerning tritium-thymidine incorporation into the DNA of mouse lymphocytes was negative. Furthermore, An unpublished micronucleus study on the bone marrow of CD-1 mice was conducted in compliance with OECD Guideline 474. No significant increase in the micronuclei was observed at doses of 400, 800 or 1,600 mg/kg body weight (Inveresk Research International Ltd, 1994). Furthermore, <i>in vitro</i> and <i>in vivo</i> genotoxicity data (micronucleus test, dominant lethal test) are available for 2-ethylhexanol which is rapidly and quantitatively converted into 2-EHA in metabolism studies. This data do not indicate any genotoxic potential which means that such an effect of 2-EHA is not likely either. As 2-EHA can induce both DNA synthesis and inhibition of intercellular communication in hepatic cells, a tumour-promoting potential in rodents, comparable to that of other peroxisome proliferators, cannot be ruled out. The carcinogenic effect of peroxisome</p>

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proliferators in rodents (e.g. of di(2-ethylhexyl)phthalate, DEHP) is not deemed to be relevant for humans. Calcium/zinc and barium/zinc salts of 2-EHA are used as thermo-stabilisers for PVC, together with co-stabilisers like polyols or epoxy compounds, in order to capture the hydrochloride cleaved during the thermal loading of PVC; in addition various salts are used in other food and beverage containers as plasticisers. The migration of 2-EHA from the sealing compounds in the metal lids. has been demonstrated in food contamination. The potential for human exposure to 2-EHA therefore is significant.

Principal route of occupational exposure to the gas is by inhalation.

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

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

For di-sec-octyl phthalate:

Oral studies of 90-days to 2-years in rat, 1-year in guinea pig and up to 1-year in dog have shown a no-effect level of about 60 mg/kg/day. Higher doses produced growth retardation and increased weights of livers and kidneys.

Rats and mice fed on diets containing 6000-12000 (rats) and 3000-6000 (mice) mg/kg body weight for 103 weeks showed an increased incidence of hepatocellular carcinomas in female rats and male and female mice, and an increased incidence of either hepatocellular carcinomas or neoplastic nodules in male rats. About 35% of the hepatocellular carcinomas in mice had metastasised to the lungs.

The substance can cause testicular damage in rats (dietary and gavage studies) with a no-effect level in 0.3% to 0.5% in the diet. Inhalation or dermal exposures did not produce testicular effects. When the substance was fed to pregnant rats (5 ml/kg) it produced slight effects on embryonic and foetal development with skeletal abnormalities more common.

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.

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.

Xylene has been classed as a developmental toxin in some jurisdictions.

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.

Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

Dy-Mark BondCover Touch Up Aerosol All Colours	TOXICITY	IRRITATION
	Not Available	Not Available
toluene	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation (rat) LC50: 49 mg/l/4h ^[2]	Eye (rabbit):0.87 mg - mild
	Oral (rat) LD50: 636 mg/kg ^[2]	Eye (rabbit):100 mg/30sec - mild
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
xylene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 4994.295 mg/l/4h ^[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 3523-8700 mg/kg ^[2]	Eye (rabbit): 87 mg mild
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit):500 mg/24h moderate
di-sec-octyl phthalate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 25000 mg/kg ^[2]	Eye (rabbit): 500 mg/24h mild
	Oral (rat) LD50: ~6860 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg/24h mild
		Skin: adverse effect observed (irritating) ^[1]

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ethylbenzene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye (rabbit): 500 mg - SEVERE
	Inhalation (mouse) LC50: 17.75 mg/l/2H ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: 3500 mg/kg ^[2]	Skin (rabbit): 15 mg/24h mild
		Skin: no adverse effect observed (not irritating) ^[1]
hydrocarbon propellant	TOXICITY	IRRITATION
	Not Available	Not Available
dimethyl ether	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 309 mg/l/4H ^[2]	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	

Dy-Mark BondCover Touch Up Aerosol All Colours	<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>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.</p> <p>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.</p>
XYLENE	<p>Reproductive effector in rats</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
DI-SEC-OCTYL PHTHALATE	<p>Di-sec-octyl phthalate (DEHP) is not acutely toxic in small laboratory animals via the oral route. The oral LD50 reported for mice is 26.3 g/kg; for rats, it is 33.8 g/kg. No skin irritation or sensitisation potential has been demonstrated in either animals or humans, and the lethal dose in rabbits is about 25 ml/kg. Deaths in rats and chronic diffuse inflammation of the lung in mice exposed to DEHP at unspecified levels have been reported.</p> <p>Long-term dietary toxicity studies in rats, guinea pigs, and dogs have established a no-effect dose level of about 60 mg/kg/day, and no carcinogenic or histologic abnormalities were observed at this level. Higher doses were associated with growth retardation and increased liver and kidney weights but not histologic abnormalities. Metabolic studies have demonstrated that laboratory animals do not appreciably metabolise DEHP. Teratogenicity studies in pregnant rats indicated that fertility is unaffected at doses of 0.1, 0.2, or 0.33 percent of the acute intraperitoneal LD50 dose for rats, although slight effects on embryonic and foetal development were observed in these animals; skeletal deformities were the most common teratogenic effects observed. Mutagenic effects were observed at intravenous doses of one-third, one-half, and two-thirds of the acute LD50; these effects are consistent with DEHP's ability to produce dominant lethal mutations.</p> <p>A study of workers exposed to a mixture of the vapors of diethyl phthalate, dibutyl phthalate, and di-2-ethylhexyl phthalate reported that exposures to 1 to 6 ppm caused no peripheral polyneuritis. However, Russian investigators examined male and female workers exposed to between 1.7 and 66 mg/m³ of various combinations of airborne phthalates (including butyl phthalate, higher aryl phthalates, dioctyl phthalate and others) and noted complaints of pain, numbness, and spasms in the upper and lower extremities after six to seven years of exposure. Polyneuritis was observed in 32 percent of the workers studied, and 78 percent of these workers showed depression of vestibular receptors. The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens. However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure.</p> <p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>Transitional Phthalate Esters: produced from alcohols with straight-chain carbon backbones of C4 to C6. This subcategory also includes a phthalate produced from benzyl alcohol as one ester group with the second ester composed of an alkyl group with a C5 carbon backbone and butyrate group. Phthalate esters containing >10% C4 to C6 molecules were conservatively included in this subcategory. Branched C7 and C8 isomers (di-iso-heptyl, di-iso-octyl and diethylhexyl phthalates) in contrast to linear dihexyl and dioctyl phthalate are members of this family. Transitional phthalates have varied uses, but are largely used as plasticisers for PVC. Physicochemical properties also vary in that the lower molecular weight transitional phthalates are more water-soluble than higher molecular weight transitional phthalates, but none would be characterised as highly water soluble. Transitional phthalates have lower water solubility than the low molecular weight phthalates and except for butylbenzyl phthalate (BBP), existing data suggest they do not exhibit acute or chronic aquatic toxicity. What distinguishes some of the transitional phthalates from others is their greater mammalian toxicity potential, particularly with regard to reproductive and developmental effects, compared to either the low or high molecular weight phthalate subcategories.</p> <p>Acute Toxicity. The available data on phthalates spanning the carbon range from C4 to C6 indicate that phthalate esters in the transitional subcategory are minimally toxic by acute oral and dermal administration. The oral LD50 value for BBP exceeds 2 g/kg, and for materials with higher molecular weights, the LD50 values exceed the maximum amounts which can be administered to the animals in a manner consistent with the principles of responsible animal use.</p> <p>One member of this subcategory, diethylhexyl phthalate (DEHP), has been tested for acute inhalation toxicity. It did not cause an effect at the</p>

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highest concentration tested. Further, considering the low volatility of these substances, inhalation exposure at toxicologically significant levels is not anticipated.

Repeated Dose Toxicity. Several substances in the C4 to C6 range, including BBP, have been tested for repeated dose toxicity in studies ranging from 3 weeks to 2 years. The principal effects found in these studies were those associated with peroxisome proliferation including liver enlargement and induction of peroxisomal enzymes. As shown in a comparative study of liver effects, the strongest inducers of peroxisome proliferation are diisononyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) with substances of shorter chain length (e.g., BBP) showing much less pronounced effects. Thus it is reasonable to conclude that other members of this subcategory would show effects similar to BBP and less pronounced than DINP or DIDP. It should also be noted that the relevance of these findings to human health is, at best, questionable. It has been shown that these effects are mediated through the peroxisome proliferation-activated receptor alpha (PPARA) and that levels of PPARa are much higher in rodents than they are in humans. Thus one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence that this is true is provided by studies in primates in which repeated administration of DINP had no effects on liver, kidney or testicular parameters.

Several of the substances in the transitional phthalate esters subcategory, however, have been shown to produce testicular atrophy when given to juvenile rats at high levels. Testicular atrophy has been associated with BBP and other substances with C4 to C6 linear carbon chains. However, molecules with fewer than 4 or more than 6 carbons did not produce testicular atrophy in these studies. Although the relevance of these data are uncertain, as the testes is not a target organ for diethylhexyl phthalate (DEHP) in primates, these data do provide one of the distinguishing toxicological characteristics of this subcategory and are one of the underlying reasons supporting the differentiation of phthalate esters on the basis of length of the linear region of the carbon chain.

Genetic Toxicity (Salmonella). A number of the substances in this subcategory including the reference substance BBP has been assessed in the Salmonella and mouse lymphoma assays. All of these substances were inactive in these assays.

Chromosomal Aberrations. BBP and dihexyl phthalate (DHP) were inactive in micronucleus assays in mice. DEHP was inactive in a cytogenetics assay in rat bone marrow. Diisooheptyl phthalate was inactive in CHO cells, in vitro.

Reproductive toxicity: A series of studies assessed the structure-activity relationship of the effects of phthalate esters on fertility using a continuous breeding protocol. The test substances included in these studies were diethyl-, dipropyl-, dibutyl-, dipentyl-, d-n-hexyl-, di-2(ethylhexyl)-, and di-n-octyl phthalates. The most profound effects were on fertility (i.e., number of females delivering/ number mated) and number of live births. The substance showing the greatest activity was DEHP which produced effects at dietary levels of 0.1 % with a no effect level of 0.01 %. The next most active compounds were di-n-hexyl- and di-n-pentyl phthalate which showed effects in the range of 0.3 to 0.5 %; no effect levels were not experimentally defined. Dipropyl phthalate had an effect on live birth index at 2.5 % but produced no effects at 1.25 %. Diethyl phthalate and di-n-octyl phthalate were inactive at the highest levels tested, 2.5 % and 5.0 %, respectively. These data demonstrated that molecules with linear alkyl chains of 4 to 6 carbons profoundly affect fertility in rodents, with DEHP being the most active. Molecules with longer or shorter side chains are essentially inactive in these assays. These data were also a basis for the separation of phthalates into three categories based on length of side chain.

In addition to these data there are reproductive toxicity studies on BBP and DEHP.

A 2-generation reproductive study was conducted in rats in which BBP was administered via the diet. Parental effects were limited to changes in body weight, weight gain, and increased absolute and relative liver weights. In the F1 parents, treatment with BBP affected mating and fertility indices and sperm number and motility. The F1 male offspring exhibited shortened anogenital distance, delayed acquisition of puberty and retention of nipples and areolae as well as reproductive effects. The NOAEL of the study was reported to be 3750 mg/ kg for reproductive effects. However, for male F1 and F2 offspring, the NOEL for reproductive effects was reported to be 50 mg/ kg based on reductions in anogenital distance. These studies along with previous data provide a good basis to assess the reproductive effects of C4 to C6 phthalate esters. Although several substances (diheptyl, heptyl nonyl, heptyl undecyl) have ester side chain constituents that predominately fall in the high molecular weight subcategory, these substances are conservatively assumed to exhibit reproductive effects similar to other transitional phthalates.

Developmental toxicity: There have been extensive studies of the developmental toxicity of BBP and DEHP. These substances produce structural malformations and also affect male reproductive development. No effect levels are in the range of 50 to 300 mg/ kg bw/ day. There is also an unpublished developmental toxicity study of di-isooheptyl phthalate (DIHP). The results of these studies are broadly consistent with the structure-activity relationships previously described, i.e., that phthalate esters with linear carbon chains of C4 to C6 carbons produce much more profound effects than either shorter or longer molecules.

Phthalate esters with >10% C4 to C6 isomers were conservatively placed in the transitional subcategory. This conclusion is supported by developmental test data on "711P" (which showed structural malformations in rats at 1000 mg/ kg/ day with a NOAEL of 200 mg/ kg/ day). "711P" is an equal composition mixture of six phthalate esters consisting of linear and methyl-branched C7, C9, and C11 ester side chains. This test substance is considered by EPA under the following CAS Numbers: 68515-44-6 (di C7), 68515-45-7 (di C9), 3648-20-2 (di C11), 111381-89-6 (C7, C9), 111381-90-9 (C7, C11), and 111381-91-0 (C9, C11). The overall content of C4 to C6 isomers in "711P" is approximately 10%, based on the contribution from methyl-branched C7 isomers e.g., di C7 (30% C4-C6); C7, C9 (15% C4-C6); and C7, C11 (15% C4-C6). Test data on 711P were used selectively as read-across data to the C7-containing substances in the mixture, based on the C4 to C6 content of each substance in the mixture.

Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen

[National Toxicology Program: U.S. Dep. of Health & Human Services 2002]

Oral (rat) NOAEL: 28.9-36.1 mg/kg/day Gastrointestinal changes, respiratory system changes, somnolence, haemorrhage, necrotic changes in GI tract, lowered blood pressure, liver, endocrine tumours, foetotoxicity, paternal effects, maternal effects, specific developmental abnormalities (hepatobiliary system, musculoskeletal system, cardiovascular system, urogenital system, central nervous system, eye/ear), foetoletality recorded.

ETHYLBENZENE

Liver changes, uterine tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded.

Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylglyoxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances.

Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys.

Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (400 ppm and greater) of ethylbenzene. In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncytial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland.

In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity.

Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination.

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<p>Dy-Mark BondCover Touch Up Aerosol All Colours & HYDROCARBON PROPELLANT</p>	<p>No significant acute toxicological data identified in literature search for Petroleum Hydrocarbon Gases:</p> <p>In many cases, there is more than one potentially toxic constituent in a refinery gas. In those cases, the constituent that is most toxic for a particular endpoint in an individual refinery stream is used to characterize the endpoint hazard for that stream. The hazard potential for each mammalian endpoint for each of the petroleum hydrocarbon gases is dependent upon each petroleum hydrocarbon gas constituent endpoint toxicity values (LC50, LOAEL, etc.) and the relative concentration of the constituent present in that gas. It should also be noted that for an individual petroleum hydrocarbon gas, the constituent characterizing toxicity may be different for different mammalian endpoints, again, being dependent upon the concentration of the different constituents in each, distinct petroleum hydrocarbon gas.</p> <p>All Hydrocarbon Gases Category members contain primarily hydrocarbons (i.e., alkanes and alkenes) and occasionally asphyxiant gases like hydrogen. The inorganic components of the petroleum hydrocarbon gases are less toxic than the C1 - C4 and C5 - C6 hydrocarbon components to both mammalian and aquatic organisms. Unlike other petroleum product categories (e.g. gasoline, diesel fuel, lubricating oils, etc.), the inorganic and hydrocarbon constituents of hydrocarbon gases can be evaluated for hazard individually to then predict the screening level hazard of the Category members</p> <p>Acute toxicity: No acute toxicity LC50 values have been derived for the C1 -C4 and C5- C6 hydrocarbon (HC) fractions because no mortality was observed at the highest exposure levels tested (~ 5 mg/l) for these petroleum hydrocarbon gas constituents. The order of acute toxicity of petroleum hydrocarbon gas constituents from most to least toxic is: C5-C6 HCs (LC50 > 1063 ppm) > C1-C4 HCs (LC50 > 10,000 ppm) > benzene (LC50 = 13,700 ppm) > butadiene (LC50 = 129,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Repeat dose toxicity: With the exception of the asphyxiant gases, repeated dose toxicity has been observed in individual selected petroleum hydrocarbon gas constituents. Based upon LOAEL values, the order of order of repeated-dose toxicity of these constituents from most toxic to the least toxic is: Benzene (LOAEL .>=10 ppm) >C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) > C5-C6 HCs (LOAEL = 6,625 ppm) > butadiene (LOAEL = 8,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Genotoxicity: In vitro: The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vitro</i> genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian <i>in vitro</i> test systems. In vivo: The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vivo</i> genotoxicity. The exceptions are benzene and 1,3-butadiene, which are genotoxic in <i>in vivo</i> test systems</p> <p>Developmental toxicity: Developmental effects were induced by two of the petroleum hydrocarbon gas constituents, benzene and the C5 -C6 hydrocarbon fraction. No developmental toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for developmental toxicity. Based on LOAEL and NOAEL values, the order of acute toxicity of these constituents from most to least toxic is: Benzene (LOAEL = 20 ppm) > butadiene (NOAEL .>=1,000 ppm) > C5-C6 HCs (LOAEL = 3,463 ppm) > C1-C4 HCs (NOAEL >=5,000 ppm; assumed to be 100% 2-butene) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Reproductive toxicity: Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the C1-C4 hydrocarbon fraction). No reproductive toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for reproductive toxicity. Based on LOAEL and NOAEL values, the order of reproductive toxicity of these constituents from most to least toxic is: Benzene (LOAEL = 300 ppm) > butadiene (NOAEL .>=6,000 ppm) > C5-C6 HCs (NOAEL .>=6,521 ppm) > C1-C4 HCs (LOAEL = 9,000 ppm; assumed to be 100% isobutane) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen)</p>
<p>Dy-Mark BondCover Touch Up Aerosol All Colours & XYLENE & ETHYLBENZENE</p>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p>Dy-Mark BondCover Touch Up Aerosol All Colours & TOLUENE & XYLENE</p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<p>Dy-Mark BondCover Touch Up Aerosol All Colours & TOLUENE</p>	<p>For toluene:</p> <p>Acute Toxicity Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies. Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy. Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days. Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death Toluene can also strip the skin of lipids causing dermatitis</p> <p>Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days</p> <p>Subchronic/Chronic Effects: Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.</p> <p>Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin. Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L</p> <p>Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .</p> <p>Developmental/Reproductive Toxicity Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals. Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours</p>

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	<p>of exposure, however none died at 500 mg/m3. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.</p> <p>Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor. Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .</p> <p>Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .</p> <p>Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites</p> <p>Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.</p>		
DI-SEC-OCTYL PHTHALATE & ETHYLBENZENE	<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>NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>		
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 BondCover Touch Up Aerosol All Colours	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
toluene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.0073mg/L	4
	EC50	48	Crustacea	3.78mg/L	5
	EC50	72	Algae or other aquatic plants	12.5mg/L	4
	BCF	24	Algae or other aquatic plants	10mg/L	4
NOEC	168	Crustacea	0.74mg/L	5	
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	
di-sec-octyl phthalate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.023mg/L	3
	EC50	48	Crustacea	>0.003mg/L	2
	EC50	96	Algae or other aquatic plants	0.002mg/L	3
	BCF	24	Fish	50mg/L	4
	EC10	72	Algae or other aquatic plants	>0.003mg/L	2
NOEC	2184	Fish	0.0001mg/L	2	
ethylbenzene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.0043mg/L	4
	EC50	48	Crustacea	1.184mg/L	4
	EC50	96	Algae or other aquatic plants	3.6mg/L	4
NOEC	168	Crustacea	0.96mg/L	5	
hydrocarbon propellant	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	24.11mg/L	2
EC50	96	Algae or other aquatic plants	7.71mg/L	2	

Continued...

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	LC50	96	Fish	24.11mg/L	2
	EC50	96	Algae or other aquatic plants	7.71mg/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:	<i>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</i>				

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

Toxic to aquatic organisms.

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

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

For aromatic hydrocarbons:

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

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

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

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

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

for 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 or 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).

For transitional phthalate esters:

Ecotoxicity:

The lower molecular weight transitional phthalate, butylbenzyl phthalate BBP, is more water soluble than the remaining higher molecular weight transitional phthalates (dihexyl and higher) and causes acute and chronic aquatic toxicity below 1 mg/L. There is an apparent cut-off in acute toxicity at dihexyl phthalate and higher; these results are further confirmed with QSAR modeling. Both calculated and measured values for environmental toxicity endpoints predict no effects at the limit of water solubility for dihexyl phthalate and higher. The data for the higher molecular weight transitional phthalates together with read-across data provide sufficient test data to suggest that these phthalates have no associated acute or chronic aquatic toxicity. This cut-off in acute toxicity is due to the concentration causing acute toxicity being higher than the water solubility of the phthalate ester.

The same situation exists for those phthalates which are more non-polar (higher carbon number) than dihexyl phthalate. This is confirmed by a lack of acute toxicity to fish, invertebrates, or algae for dihexyl phthalate (DHP), di-iso-heptyl phthalate (DIHP). The same situation exists for those phthalates which are more non-polar (higher carbon number) than dihexyl phthalate. This is confirmed by a lack of acute toxicity to fish, invertebrates, or algae for DHP, DIHP,

The better solubility and thus the higher availability of the phthalates with short alkyl chains leads to a higher toxicity. Concurrently they are available for degrading microorganisms and therefore not persistent. The compounds with higher molecular weight and a low solubility are strongly adsorbed to soil and to suspended particulate matter in water. Therefore they are not very accessible to biochemical processes leading to degradation.

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

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Henry's Pa m3 /mol: 637-879
 Henry's atm m3 /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 7.18×10^{-3} atm-cu m/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

For isobutane:

Refrigerant Gas: Saturated Hydrocarbons have zero ozone depletion potential (ODP) and will photodegrade under atmospheric conditions. [Calor Gas]

Environmental Fate

Terrestrial fate: An estimated Koc value of 35 suggests that isobutane will have very high mobility in soil. Its very high Henry's Law constant, 4.08 atm-cu m/mole, (calculated from its vapor pressure and water solubility, high vapor pressure, 2611 mm Hg at 25 deg C, and low adsorptivity to soil indicate that volatilisation will be an important fate process from both moist and dry soil surfaces. Isobutane is biodegradable, especially under acclimated conditions, and may biodegrade in soil.

Aquatic fate: The estimated Koc value suggests that isobutane would not adsorb to sediment and particulate matter in the water column. Additional evidence that isobutane is not removed to sediment has been obtained from microcosm experiments. Isobutane will readily volatilise from water based on its estimated Henry's Law constant of 4.08 atm-cu m/mole. Estimated half-lives for a model river and model lake are 2.2 hr and 3.0 days, respectively. An estimated BCF value of 74 based on the log Kow suggests that isobutane will not bioconcentrate in aquatic organisms.

Results indicate that gas exchange is the dominant removal mechanism for isobutane gases from the water column following a hypothetical input. The volatilisation half-lives for isobutane from the water columns in natural estuaries are estimated to be 4.4 and 6.8 days at 20 and 10 deg C, respectively.

Isobutane also biodegrades in the microcosm at a rate that is slower than for n-butane and falls between propane and ethane in susceptibility. Biodegradation of isobutane initially occurs with a half-lives of 16-26 days at 20 deg C and 33-139 days at 10 deg C, significantly slower than the loss predicted by gas exchange from typical natural estuaries. However, after a lag of 2-4 weeks, the biodegradation rate increases markedly so that in the case of chronic inputs, biodegradation can become the dominant removal mechanism.

Atmospheric fate: Isobutane is a gas at ordinary temperatures. It is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is 6.9 days, assuming a hydroxyl radical concn of 5×10^5 radicals per cubic cm. When isobutane was exposed to sunlight for 6 hr in a tedlar bag filled with Los Angeles air, 6% of the isobutane degraded. The air contained 4529 ppb-C hydrocarbons and 870 ppb of NOX. The tropospheric loss of volatile hydrocarbons such as isobutane by wet and dry deposition are believed to be of minor importance. Indeed, isobutane assimilated into precipitation may evaporate during transport as well as being reemitted into the atmosphere after deposition. Isobutane is a contributor to the production of PAN (peroxyacyl nitrates) under photochemical smog conditions

For propane:

Environmental Fate

Terrestrial fate: An estimated Koc value of 460 determined from a log Kow of 2.36 indicates that propane is expected to have moderate mobility in soil. Volatilisation of propane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 7.07×10^{-1} atm-cu m/mole, derived from its vapor pressure, 7150 mm Hg, and water solubility, 62.4 mg/L. Propane is expected to volatilise from dry soil surfaces based upon its vapor pressure. Using cell suspensions of microorganisms isolated from soil and water, propane was oxidised to acetone within 24 hours, suggesting that biodegradation may be an important fate process in soil and sediment.

Aquatic fate: The estimated Koc value indicates that propane is expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant. Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 41 minutes and 2.6 days, respectively. An estimated BCF of 13.1 using log Kow suggests the potential for bioconcentration in aquatic organisms is low. After 192 hr, the trace concentration of propane contained in gasoline remained unchanged for both a sterile control and a mixed culture sample collected from ground water contaminated with gasoline. This indicates that biodegradation may not be an important fate process in water.

Atmospheric fate: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and vapour pressure, propane is expected to exist solely as a gas in the ambient atmosphere. Gas-phase propane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 14 days, calculated from its rate constant of 1.15×10^{-12} cu cm/molecule-sec at 25 deg C. Propane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.

For toluene:

log Kow : 2.1-3
 log Koc : 1.12-2.85
 Koc : 37-260
 log Kom : 1.39-2.89
 Half-life (hr) air : 2.4-104
 Half-life (hr) H2O surface water : 5.55-528
 Half-life (hr) H2O ground : 168-2628
 Half-life (hr) soil : <48-240
 Henry's Pa m3 /mol: 518-694
 Henry's atm m3 /mol: 5.94E-03
 BOD 5 0.86-2.12, 5%
 COD : 0.7-2.52,21-27%
 ThOD : 3.13
 BCF : 1.67-380
 log BCF : 0.22-3.28

Environmental fate:

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

Transformation/Persistence:

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

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

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

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

Ecotoxicity:

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

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

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

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

shrimp (*Palaemonetes pugio*) 9.5 mg/l

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
di-sec-octyl phthalate	HIGH (Half-life = 389 days)	LOW (Half-life = 1.21 days)
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
dimethyl ether	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
toluene	LOW (BCF = 90)
xylene	MEDIUM (BCF = 740)
di-sec-octyl phthalate	HIGH (BCF = 24500)
ethylbenzene	LOW (BCF = 79.43)
dimethyl ether	LOW (LogKOW = 0.1)

Mobility in soil

Ingredient	Mobility
toluene	LOW (KOC = 268)
di-sec-octyl phthalate	LOW (KOC = 165400)
ethylbenzene	LOW (KOC = 517.8)
dimethyl ether	HIGH (KOC = 1.292)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Consult State Land Waste Management Authority for disposal. ▶ Discharge contents of damaged aerosol cans at an approved site. ▶ Allow small quantities to evaporate. ▶ DO NOT incinerate or puncture aerosol cans. ▶ Bury residues and emptied aerosol cans at an approved site.
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SECTION 14 TRANSPORT INFORMATION

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Labels Required

	
Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG)

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

Air transport (ICAO-IATA / DGR)

UN number	1950
UN proper shipping name	Aerosols, flammable
Transport hazard class(es)	ICAO/IATA Class : 2.1 ICAO / IATA Subrisk : Not Applicable ERG Code : 10L
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions : A145 A167 A802 Cargo Only Packing Instructions : 203 Cargo Only Maximum Qty / Pack : 150 kg Passenger and Cargo Packing Instructions : 203 Passenger and Cargo Maximum Qty / Pack : 75 kg Passenger and Cargo Limited Quantity Packing Instructions : Y203 Passenger and Cargo Limited Maximum Qty / Pack : 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	IMDG Class : 2.1 IMDG Subrisk : Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	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

TOLUENE IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

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

DI-SEC-OCTYL PHTHALATE 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 10 / Appendix C
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

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

ETHYLBENZENE 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
 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

HYDROCARBON PROPELLANT 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

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 - NDCL	No (toluene; xylene; di-sec-octyl phthalate; ethylbenzene; hydrocarbon propellant; dimethyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECL	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	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

Revision Date	01/11/2019
Initial Date	02/10/2017

SDS Version Summary

Version	Issue Date	Sections Updated
4.1.1.1	11/10/2017	Synonyms, Name
5.1.1.1	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.

Dy-Mark BondCover Touch Up Aerosol All Colours**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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