



MIROCAT PC 3220 Clear Topcoat

Mirotone (NZ) Ltd

Chemwatch: 5129-37

Version No: 6.1.1.1

Safety Data Sheet according to HSNO Regulations

Chemwatch Hazard Alert Code: 3

Issue Date: 05/05/2015

Print Date: 06/09/2015

Initial Date: Not Available

L.GHS.NZL.EN

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

Product Identifier

Product name	MIROCAT PC 3220 Clear Topcoat
Synonyms	MIROCAT PC 3220 Clear Gloss Topcoat, MIROCAT PC 3220/10 Clear Silky Matt Topcoat, MIROCAT PC 3220/30 Clear Satin Topcoat., MIROCAT PC 3220/60 Clear Semi-Gloss Topcoat
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Other means of identification	Not Available

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

Relevant identified uses	The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. Use according to manufacturer's directions. Coating for furniture.
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Details of the manufacturer/importer

Registered company name	Mirotone (NZ) Ltd
Address	32 Cryers Road New Zealand
Telephone	0800 FINISH (0800 346 474)
Fax	0800 346 434
Website	mirotone.com
Email	information@mirotone.co.nz

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	111
Other emergency telephone numbers	Not Available

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
+800 2436 2255	+612 9186 1132	Not Available

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

**Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation.
Classified as Dangerous Goods for transport purposes.**

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	3	4
Toxicity	2	3
Body Contact	3	4
Reactivity	2	3
Chronic	3	4

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

GHS Classification [1]	Flammable Liquid Category 2, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin
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	Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Carcinogen Category 2, Reproductive Toxicity Category 1B, STOT - SE (Resp. Irr.) Category 3, STOT - SE (Narcosis) Category 3, STOT - RE Category 2, Aspiration Hazard Category 1, Chronic Aquatic Hazard Category 3, Acute Terrestrial Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	3.1B, 6.1D (dermal), 6.1D (inhalation), 6.1D (oral), 6.1E (aspiration), 6.3A, 6.7B, 6.8A, 6.9 (narcotic), 6.9 (respiratory), 6.9B (inhalation), 8.3A, 9.1C, 9.2C

Label elements

GHS label elements	
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SIGNAL WORD

DANGER

Hazard statement(s)

H225	Highly flammable liquid and vapour
H302	Harmful if swallowed
H312	Harmful in contact with skin
H332	Harmful if inhaled
H315	Causes skin irritation
H318	Causes serious eye damage
H351	Suspected of causing cancer
H360	May damage fertility or 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
H412	Harmful to aquatic life with long lasting effects
H423	Harmful to the soil environment

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P331	Do NOT induce vomiting.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

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P403+P233 Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501 Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	15-70	resins
108-88-3	5-35	<u>toluene</u>
123-86-4	5-35	<u>n-butyl acetate</u>
78-93-3	5-35	<u>methyl ethyl ketone</u>
71-36-3	5-35	<u>n-butanol</u>
1330-20-7	5-35	<u>xylene</u>
128-37-0	<0.5	<u>2,6-di-tert-butyl-4-methylphenol</u>
1843-05-6	<5	<u>octabenzene</u>
117-81-7	<5	<u>di-sec-octyl phthalate</u>
67-63-0	<5	<u>isopropanol</u>
	balance	Ingredients determined not to be hazardous

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 FIRST AID MEASURES

NZ Poisons Centre 0800 POISON (0800 764 766) | NZ Emergency Services: 111

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with 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. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ 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 skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation. <p>In case of burns:</p> <ul style="list-style-type: none"> ▶ Immediately apply cold water to burn either by immersion or wrapping with saturated clean cloth. ▶ DO NOT remove or cut away clothing over burnt areas. DO NOT pull away clothing which has adhered to the skin as this can cause further injury. ▶ DO NOT break blister or remove solidified material. ▶ Quickly cover wound with dressing or clean cloth to help prevent infection and to ease pain. ▶ For large burns, sheets, towels or pillow slips are ideal; leave holes for eyes, nose and mouth. ▶ DO NOT apply ointments, oils, butter, etc. to a burn under any circumstances. ▶ Water may be given in small quantities if the person is conscious. ▶ Alcohol is not to be given under any circumstances. ▶ Reassure. ▶ Treat for shock by keeping the person warm and in a lying position. ▶ Seek medical aid and advise medical personnel in advance of the cause and extent of the injury and the estimated time of arrival of the patient.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

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Treat symptomatically.

To treat poisoning by the higher aliphatic alcohols (up to C7):

- ▶ Gastric lavage with copious amounts of water.
- ▶ It may be beneficial to instill 60 ml of mineral oil into the stomach.
- ▶ Oxygen and artificial respiration as needed.
- ▶ Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- ▶ To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- ▶ Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5]

BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ Monitor and treat, where necessary, for pulmonary oedema.
- ▶ 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.
- ▶ Give activated charcoal.

ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Acidosis may respond to hyperventilation and bicarbonate therapy.
- ▶ Haemodialysis might be considered in patients with severe intoxication.
- ▶ Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.

Symptomatic and supportive therapy is advised in managing patients.

- ▶ Heavy and persistent skin contamination over many years may lead to dysplastic changes. Pre-existing skin disorders may be aggravated by exposure to this product.
- ▶ In general, emesis induction is unnecessary with high viscosity, low volatility products, i.e. most oils and greases.
- ▶ High pressure accidental injection through the skin should be assessed for possible incision, irrigation and/or debridement.

NOTE: Injuries may not seem serious at first, but within a few hours tissue may become swollen, discoloured and extremely painful with extensive subcutaneous necrosis. Product may be forced through considerable distances along tissue planes.

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

- ▶ Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- ▶ Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- ▶ Water spray or fog.
 - ▶ Alcohol stable foam.
 - ▶ Dry chemical powder.
 - ▶ Carbon dioxide.
- Do not** use a water jet to fight fire.

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

Advice for firefighters

Fire Fighting

WARNING: EXPLOSIVE MATERIALS / ARTICLES PRESENT!

- ▶ Evacuate all personnel and move upwind.
- ▶ Prevent re-entry.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ May detonate and burning material may be propelled from fire.
- ▶ Wear full-body protective clothing with breathing apparatus.
- ▶ Prevent, by any means available, spillage and fire effluent from entering drains and water courses.
- ▶ Fight fire from safe distances and from protected locations.
- ▶ Use flooding quantities of water.

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	<ul style="list-style-type: none"> ▶ DO NOT approach containers or packages suspected to be hot. ▶ Cool any exposed containers not involved in fire from a protected location. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<p>WARNING: EXPLOSION HAZARD!</p> <ul style="list-style-type: none"> ▶ Combustible. ▶ Detonation may occur from heavy impact or excessive heating. ▶ Mixing with incompatible chemicals may cause expansion, decomposition or detonation. ▶ Heat affected containers remain hazardous. ▶ Explosives can supply own oxygen for combustion and smothering action of foam or dry chemical may be ineffective. ▶ Combustion or decomposition produces oxides of nitrogen (NOx), carbon monoxide (CO) and carbon dioxide (CO2). <p>Combustion products include; carbon dioxide (CO2) aldehydes nitrogen oxides (NOx) other pyrolysis products typical of burning organic material Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	Environmental hazard - contain spillage. Slippery when spilt.																																		
Major Spills	Environmental hazard - contain spillage. Chemical Class: ester and ethers For release onto land: recommended sorbents listed in order of priority.																																		
	<table><tr><td>SORBENT TYPE</td><td>RANK</td><td>APPLICATION</td><td>COLLECTION</td><td>LIMITATIONS</td></tr></table>					SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS																									
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	LAND SPILL - SMALL																																		
	<table><tr><td>cross-linked polymer - particulate</td><td>1</td><td>shovel</td><td>shovel</td><td>R, W, SS</td></tr><tr><td>cross-linked polymer - pillow</td><td>1</td><td>throw</td><td>pitchfork</td><td>R, DGC, RT</td></tr><tr><td>sorbent clay - particulate</td><td>2</td><td>shovel</td><td>shovel</td><td>R,I, P</td></tr><tr><td>wood fiber - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, W, P, DGC</td></tr><tr><td>wood fiber - pillow</td><td>3</td><td>throw</td><td>pitchfork</td><td>R, P, DGC, RT</td></tr><tr><td>treated wood fiber - pillow</td><td>3</td><td>throw</td><td>pitchfork</td><td>DGC, RT</td></tr></table>					cross-linked polymer - particulate	1	shovel	shovel	R, W, SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R,I, P	wood fiber - particulate	3	shovel	shovel	R, W, P, DGC	wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT	treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
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	<table><tr><td>cross-linked polymer - particulate</td><td>1</td><td>blower</td><td>skiploader</td><td>R,W, SS</td></tr><tr><td>cross-linked polymer - pillow</td><td>2</td><td>throw</td><td>skiploader</td><td>R, DGC, RT</td></tr><tr><td>sorbent clay - particulate</td><td>3</td><td>blower</td><td>skiploader</td><td>R, I, P</td></tr><tr><td>polypropylene - particulate</td><td>3</td><td>blower</td><td>skiploader</td><td>W, SS, DGC</td></tr><tr><td>expanded mineral - particulate</td><td>4</td><td>blower</td><td>skiploader</td><td>R, I, W, P, DGC</td></tr><tr><td>wood fiber - particulate</td><td>4</td><td>blower</td><td>skiploader</td><td>R, W, P, DGC</td></tr></table>					cross-linked polymer - particulate	1	blower	skiploader	R,W, SS	cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT	sorbent clay - particulate	3	blower	skiploader	R, I, P	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC	wood fiber - particulate	4	blower	skiploader	R, W, P, DGC
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Legend DGC: Not effective where ground cover is dense R: Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988 Slippery when spilt.																																			
Personal Protective Equipment advice is contained in Section 8 of the MSDS.																																			

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Containers, even those that have been emptied, may contain explosive vapours. ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. <p>Contains low boiling substance: Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.</p> <ul style="list-style-type: none"> ▶ Check for bulging containers. ▶ Vent periodically ▶ Always release caps or seals slowly to ensure slow dissipation of vapours ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Electrostatic discharge may be generated during pumping - this may result in fire. ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (≤ 1 m/sec until fill pipe submerged to twice its diameter, then ≤ 7 m/sec). ▶ Avoid splash filling. ▶ Do NOT use compressed air for filling discharging or handling operations. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area.
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	<ul style="list-style-type: none"> ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights, heat or ignition sources. ▶ When handling, DO NOT eat, drink or smoke. ▶ Vapour may ignite on pumping or pouring due to static electricity. ▶ DO NOT use plastic buckets. ▶ Earth and secure metal containers when dispensing or pouring product. ▶ Use spark-free tools when handling. ▶ Avoid contact with incompatible materials. ▶ Keep containers securely sealed. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this MSDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers in approved flame-proof area. ▶ No smoking, naked lights, heat or ignition sources. ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped. ▶ Keep containers securely sealed. ▶ Store away from incompatible materials in a cool, dry well ventilated area. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this MSDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Packing as supplied by manufacturer. ▶ Plastic containers may only be used if approved for flammable liquid. ▶ Check that containers are clearly labelled and free from leaks. ▶ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▶ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▶ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▶ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid strong acids, bases. ▶ Avoid reaction with oxidising agents

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	toluene	Toluene	188 mg/m ³ / 50 ppm	Not Available	Not Available	Skin absorption
New Zealand Workplace Exposure Standards (WES)	n-butyl acetate	n-Butyl acetate	713 mg/m ³ / 150 ppm	950 mg/m ³ / 200 ppm	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	methyl ethyl ketone	Methyl ethyl ketone	445 mg/m ³ / 150 ppm	890 mg/m ³ / 300 ppm	Not Available	Exposure can also be estimated by biological monitoring.
New Zealand Workplace Exposure Standards (WES)	n-butanol	n-Butyl alcohol	Not Available	Not Available	150 mg/m ³ / 50 ppm	Skin absorption
New Zealand Workplace Exposure Standards (WES)	xylene	Xylene (o-, m-, p-isomers)	217 mg/m ³ / 50 ppm	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	2,6-di-tert-butyl-4-methylphenol	2,6-Di-tert-butyl-p-cresol	10 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	di-sec-octyl phthalate	Di-sec-octyl phthalate	5 mg/m ³	10 mg/m ³	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	isopropanol	Isopropyl alcohol	983 mg/m ³ / 400 ppm	1230 mg/m ³ / 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
n-butyl acetate	Butyl acetate, n-	Not Available	Not Available	Not Available
methyl ethyl ketone	Butanone, 2-; (Methyl ethyl ketone; MEK)	Not Available	Not Available	Not Available
n-butanol	Butyl alcohol, n-; (n-Butanol)	20 ppm	50 ppm	8000 ppm
xylene	Xylenes	Not Available	Not Available	Not Available

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2,6-di-tert-butyl-4-methylphenol	Bis(1,1-dimethylethyl)-4-methylphenol, 2,6-; (BHT (food grade)); 2,6-Di-tert-butyl-p-cresol)	6 mg/m3	16 mg/m3	180 mg/m3
di-sec-octyl phthalate	Di-sec-octylphthalate	10 mg/m3	31 mg/m3	5900 mg/m3
isopropanol	Isopropyl alcohol	400 ppm	400 ppm	12000 ppm

Ingredient	Original IDLH	Revised IDLH
resins	Not Available	Not Available
toluene	2,000 ppm	500 ppm
n-butyl acetate	10,000 ppm	1,700 [LEL] ppm
methyl ethyl ketone	3,000 ppm	3,000 [Unch] ppm
n-butanol	8,000 ppm	1,400 [LEL] ppm
xylene	1,000 ppm	900 ppm
2,6-di-tert-butyl-4-methylphenol	Not Available	Not Available
octabenzene	Not Available	Not Available
di-sec-octyl phthalate	Unknown mg/m3 / Unknown ppm	5,000 mg/m3
isopropanol	12,000 ppm	2,000 [LEL] ppm

MATERIAL DATA

For n-butyl acetate

Odour Threshold Value: 0.0063 ppm (detection), 0.038-12 ppm (recognition)

Exposure at or below the recommended TLV-TWA is thought to prevent significant irritation of the eyes and respiratory passages as well as narcotic effects. In light of the lack of substantive evidence regarding teratogenicity and a review of acute oral data a STEL is considered inappropriate.

Odour Safety Factor(OSF)

OSF=3.8E2 (n-BUTYL ACETATE)

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)

2,6-di-tert-butyl-4-methylphenol (syn: butylated hydroxytoluene - BHT)

Because high dose levels are required to produce toxic effects and because there is little evidence of either acute or chronic effects amongst workers the recommended TLV-TWA is identical to that proposed for nuisance particulates.

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)

Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)

Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

For n-butanol:

Odour Threshold Value: 0.12-3.4 ppm (detection), 1.0-3.5 ppm (recognition)

NOTE: Detector tubes for n-butanol, measuring in excess of 5 ppm are commercially available.

Exposure at or below the TLV-TWA is thought to provide protection against hearing loss due to vestibular and auditory nerve damage in younger workers and to protect against the significant risk of headache and irritation.

25 ppm may produce mild irritation of the respiratory tract 50 ppm may produce headache and vertigo.

Higher concentrations may produce marked irritation, sore throat, coughing, nausea, shortness of breath, pulmonary injury and central nervous system depression characterised by headache, dizziness, dullness and drowsiness.

6000 ppm may produce giddiness, prostration, narcosis, ataxia, and death.

Odour Safety Factor (OSF)

OSF=60 (n-BUTANOL)

For methyl ethyl ketone:

Odour Threshold Value: Variously reported as 2 ppm and 4.8 ppm

Odour threshold: 2 ppm (detection); 5 ppm (recognition) 25 ppm (easy recognition); 300 ppm IRRITATING

Exposures at or below the recommended TLV-TWA are thought to prevent injurious systemic effects and to minimise objections to odour and irritation. Where synergism or potentiation may occur stringent control of the primary toxin (e.g. n-hexane or methyl butyl ketone) is desirable and additional consideration should be given to lowering MEK exposures.

Odour Safety Factor(OSF)

OSF=28 (METHYL ETHYL KETONE)

Exposure controls

Continued...

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Appropriate engineering controls

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

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

The basic types of engineering controls are:

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

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

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

For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.

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

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to 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.

Personal protection



Eye and face protection

- ▶ Safety glasses with side shields.
- ▶ Chemical goggles.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption 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

- ▶ Wear chemical protective gloves, e.g. PVC.
- ▶ Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

- ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

For esters:

- ▶ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- ▶ frequency and duration of contact,
- ▶ chemical resistance of glove material,
- ▶ glove thickness and
- ▶ dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- ▶ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- ▶ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- ▶ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- ▶ Contaminated gloves should be replaced.

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

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Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ► Overalls. ► PVC Apron. ► PVC protective suit may be required if exposure severe. ► Eyewash unit. ► Ensure there is ready access to a safety shower. <p>Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.</p>
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
##n-butyl	acetate
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/BUTYL	C
VITON/CHLOROBUTYL	C
VITON/NEOPRENE	C
##methyl ethyl	ketone
##di-sec-octyl	phthalate

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

Type AX-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 / Class 1 P2	-	AX-PAPR-AUS / Class 1 P2
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	AX-3 P2	-
100+ x ES	-	Air-line**	-

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

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Clear to milky yellow highly flammable liquid with a slightly sweet odour; slightly miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.90-0.96
Odour	Not Available	Partition coefficient n-octanol / water	Not Available

Continued...

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Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	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)	-6.7	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)	72-75 (VOC=665-700 g/l)
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Partly miscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	>1	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ► Presence of shock and friction ► Presence of heat source and ignition source ► Unstable in the presence of incompatible materials. ► 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>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>The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.</p> <p>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.</p> <p>Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.</p> <p>Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed</p> <p>Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p> <p>The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression, headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures.</p> <p>Prolonged exposure may cause headache, nausea and ultimately loss of consciousness.</p> <p>Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms.</p>
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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.</p> <p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>The substance and/or its metabolites may bind to haemoglobin inhibiting normal uptake of oxygen. This condition, known as "methaemoglobinemia", is a form of oxygen starvation (anoxia).</p> <p>Symptoms include cyanosis (a bluish discolouration skin and mucous membranes) and breathing difficulties. Symptoms may not be evident until several hours after exposure.</p> <p>At about 15% concentration of blood methaemoglobin there is observable cyanosis of the lips, nose and earlobes. Symptoms may be absent although euphoria, flushed face and headache are commonly experienced. At 25-40%, cyanosis is marked but little disability occurs other than that produced on physical exertion. At 40-60%, symptoms include weakness, dizziness, lightheadedness, increasingly severe headache, ataxia, rapid shallow respiration, drowsiness, nausea, vomiting, confusion, lethargy and stupor. Above 60% symptoms include dyspnea, respiratory depression, tachycardia or bradycardia, and convulsions. Levels exceeding 70% may be fatal.</p> <p>High molecular weight material; on single acute exposure would be expected to pass through gastrointestinal tract with little change / absorption. Occasionally accumulation of the solid material within the alimentary tract may result in formation of a bezoar (concretion), producing discomfort.</p> <p>Phthalates (aromatic dicarboxylic acid esters), in general, exhibit low toxicity, partly because of poor absorption but mainly as a result of rapid metabolism in which the esters are saponified to phthalic acid (which is rapidly excreted) and the parent alcohol (which is subsequently metabolised). The pathology of these compounds seems to be related to the released alcohol and its biological effects. The rate of absorption of ingested phthalate esters is influenced by the content of dietary fat. Ingested phthalate esters may to a lesser degree be absorbed as the monoester derivatives or in the case of di(2-ethylhexyl)phthalate, as the diester. Cumulative toxicity of the phthalates has been observed on repeated administration. Both di-n-octyl phthalate and di(2-ethylhexyl)phthalate were found to have 22-28 times greater toxicity (based on LD50s) following repeated administration to animals. The liver has been shown to be the target organ affected by the phthalates. In general phthalates have induced liver enlargement; this increase in liver weight has been attributed to rapid cell division (hyperplasia) along with the detachment of cells (hypertrophy). The increase in liver weight caused by phthalates has been found to reverse to normal or even below normal levels on prolonged exposure.</p> <p>Exposure to phthalates, in general, has been found to be associated with a reduction in circulating cholesterol and serum triglyceride levels which accounted for a reduction in liver steroidogenesis. The phthalates also effect carbohydrate metabolism in the liver producing depleted glycogen electron transport inhibitors following interaction with mitochondria. Testicular atrophy produced in rats during feeding studies depends on the length and structure of the alcohol; in general the lower molecular weight esters produce the more severe effects. The toxicity of phthalic acid isomers decreases in the order o-phthalic acid, isophthalic acid and terephthalic acid. Phthalic acid is not metabolised but is excreted, unchanged, in the urine and faeces. Terephthalic acid appears to potentiate the biological effects of substances such as antibiotics, thiamine and sulfonamides.</p> <p>Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.</p> <p>Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality</p> <p>Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.</p> <p>Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either:</p> <ul style="list-style-type: none"> ▶ produces severe inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant and severe inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. ▶ Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. <p>NOTE: Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>The material may accentuate any pre-existing dermatitis condition</p>
Eye	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</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.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>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.</p> <p>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.</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p>

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Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.

Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.

There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.

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.

The material contains a substantial proportion of a polymer considered to be of low concern (PLC). The trend towards production of lower molecular weight polymers (thus reducing the required level of solvent use and creating a more "environmentally-friendly" material) has brought with it the need to define PLCs as those

- ▶ having molecular weights of between 1000 and 10000 and containing less than 10% of the molecules with molecular weight below 500 and less than 25% of the molecules with a molecular weight below 1000. These may contain unlimited low concern functional groups or moderate concern reactive functional groups with a combined functional group equivalent weight (FGEW, a concept developed by the US EPA describing whether the reactive functional group is sufficiently diluted by polymeric material) of a 1000 or more (provided no high concern groups are present) or high concern reactive functional groups with a FGEW of 5000 or more (FGEW includes moderate concern groups if present).
- ▶ having molecular weights exceeding 10000 (without restriction on reactive groups).

Reactive functional groups are in turn classified as being of low, moderate or high concern. Classification of the polymer as a PLC, in accordance with established criteria, does not mean that hazards will not be associated with the polymer (during its import, manufacture, use, storage, handling or disposal). The polymer may, for example, contain a large number of particles in the respirable range, a hazard which may need to be assessed in the health and safety risk assessment. Similarly a polymer with low concern reactive may be released into the environment in large quantities and produce an environmental hazard. Whilst it is generally accepted that polymers with a molecular weight exceeding 1000 are unlikely to pass through biological membranes, oligomers with lower molecular weight and specifically, those with a molecular weight below 500, may. Estimations based on a "highly" dispersed polymer population (polydispersity = 10) suggests that the molecular weight of the polymer carrying a reactive group of high concern must be 5000 to be considered a PLC; similarly a polymer of approximate molecular weight 1000 could contain no more than one reactive group of moderate concern (for two moderate concern groups, the molecular weight would be about 2500).

MIROCAT PC 3220 Clear Topcoat	TOXICITY	IRRITATION
	Not Available	Not Available
toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation (rat) LC50: >26700 ppm/1hd ^[2]	Eye (rabbit): 0.87 mg - mild
	Inhalation (rat) LC50: 49 mg/L/4H ^[2]	Eye (rabbit): 100 mg/30sec - mild
	Oral (rat) LD50: 636 mg/kg ^[2]	Skin (rabbit): 20 mg/24h-moderate
n-butyl acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >14080 mg/kg ^[1]	* [PPG]
	Inhalation (rat) LC50: 2000 ppm/4Hg ^[2]	Eye (human): 300 mg
	Inhalation (rat) LC50: 390 ppm/4h ^[2]	Eye (rabbit): 20 mg (open)-SEVERE
	Oral (rat) LD50: 10736 mg/kg ^[1]	Eye (rabbit): 20 mg/24h - moderate
methyl ethyl ketone	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >8100 mg/kg ^[1]	- mild
	Inhalation (rat) LC50: 23.5 mg/L/8H ^[2]	Eye (human): 350 ppm -irritant
	Inhalation (rat) LC50: 50.1 mg/L/8 hr ^[2]	Eye (rabbit): 80 mg - irritant
	Oral (rat) LD50: 3474.9 mg/kg ^[1]	Skin (rabbit): 402 mg/24 hr - mild
n-butanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3434.4 mg/kg ^[1]	Eye (human): 50 ppm - irritant
	Inhalation (rat) LC50: 24 mg/L/4H ^[2]	Eye (rabbit): 1.6 mg-SEVERE
	Inhalation (rat) LC50: 8000 ppm/4hE ^[2]	Eye (rabbit): 24 mg/24h-SEVERE
	Oral (rat) LD50: 2292.3 mg/kg ^[1]	Skin (rabbit): 405 mg/24h-moderate
xylene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 5000 ppm/4h ^[2]	Eye (rabbit): 5 mg/24h SEVERE

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2,6-di-tert-butyl-4-methylphenol	Oral (rat) LD50: 4300 mg/kg ^[2]	Eye (rabbit): 87 mg mild
		Skin (rabbit): 500 mg/24h moderate
	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg/24h-moderate
octabenzene	Oral (rat) LD50: 890 mg/kg ^[2]	Skin (human): 500 mg/48h - mild
		Skin (rabbit): 500 mg/48h-moderate
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >10000 mg/kg ^[2]	Eye (rabbit): non irritating**
di-sec-octyl phthalate	Oral (rat) LD50: >10000 mg/kg ^[2]	Skin (rabbit): irritating
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 25000 mg/kg ^[2]	Eye (rabbit): 500 mg/24h mild
	Oral (rat) LD50: 30000 mg/kg ^[2]	Skin (rabbit): 500 mg/24h mild
isopropanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12792 mg/kg ^[1]	Eye (rabbit): 10 mg - moderate
	Inhalation (rat) LC50: 72.6 mg/L/4h ^[2]	Eye (rabbit): 100 mg - SEVERE
	Oral (rat) LD50: 5000 mg/kg ^[2]	Eye (rabbit): 100mg/24hr-moderate
Legend:		Skin (rabbit): 500 mg - mild
	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's msds. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

MIROCAT PC 3220 Clear Topcoat	<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>No significant acute toxicological data identified in literature search.</p> <p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</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>The material may produce severe 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) thickening of the epidermis.</p> <p>Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p> <p>For toluene:</p> <p>Acute Toxicity</p> <p>Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.</p> <p>Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.</p> <p>Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.</p> <p>Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.</p> <p>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.</p> <p>Toluene can also strip the skin of lipids causing dermatitis.</p> <p>Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days.</p> <p>Subchronic/Chronic Effects:</p> <p>Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.</p> <p>Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitizer and fatal cardiotoxin.</p> <p>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</p>	

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

Developmental/Reproductive Toxicity

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

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

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

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

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

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

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

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure. Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities. Combinations with chloroform also show increase in toxicity

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

For toluene:

Acute Toxicity

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

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

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

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

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

Toluene can also strip the skin of lipids causing dermatitis

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

Subchronic/Chronic Effects:

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

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

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

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

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

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

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

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

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and

TOLUENE

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	excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.
N-BUTYL ACETATE	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The 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>
METHYL ETHYL KETONE	<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>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>Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities.</p> <p>Combinations with chloroform also show increase in toxicity</p>
N-BUTANOL	<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>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>for n-butanol</p> <p>Acute toxicity: n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC0 of 8000 ppm (24000 mg/m3) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA's localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser.</p> <p>The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed.</p> <p>Repeat dose toxicity: An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAc) to BA. Hydrolysis of BAc in blood and brain was estimated to be 99 percent complete within 2.7 minutes (elimination t1/2 = 0.41 minute). Thus, organisms exposed to BAc can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAc can be used as supplemental, surrogate data to provide information on the toxicity of BA.</p> <p>A thirteen-week, subchronic exposure to BAc, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m3) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOEL) of 500 ppm (2395 mg/m3) was reported for systemic effects in rats, and a NOEL of 3000 ppm (14370 mg/m3) was reported for post exposure neurotoxicity in rats.</p> <p>Reproductive toxicity: Several studies indicate that BA is not a reproductive toxicant.</p> <p>Female rats exposed to 6000 ppm (18000 mg/m3) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m3) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity.</p> <p>Developmental toxicity: BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m3) throughout gestation.</p> <p>Genotoxicity: An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic.</p> <p>Carcinogenicity: Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.</p>
XYLENE	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The 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>The substance is classified by IARC as Group 3:</p> <p>NOT classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p> <p>Reproductive effector in rats</p>

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2,6-DI-TERT-BUTYL-
4-METHYLPHENOL

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.

for bridged alkyl phenols:

Acute toxicity: Acute oral and dermal toxicity data are available for all but two of the substances in the group. The data show that acute toxicity of these substances is low. The testing for acute toxicity spans five decades

Repeat dose toxicity: Repeat dose studies on the members of this category include both subchronic and chronic exposures. The liver is identified as the target organ in rats for all of the substances tested. NOAEL's or NOEL's in rats for 13- week studies ranged from 100 ppm (approximately 5 mg/kg/day) to 500 ppm (approximately 25 mg/kg/day) while NOAEL's or NOEL's in rats for chronic studies were the same, 25 mg/kg/day (500 ppm).

Reproductive toxicity: Evaluation of effects on reproduction for the bridged alkyl phenols is supplemented by histopathological data on male and female reproductive organs in repeated dose studies. The data on the effects of bridged alkyl phenols on reproduction and reproductive organs span the range of structures and molecular weights. While not all of the data for reproductive effects are from reproduction studies, microscopic evaluations of reproductive organs along with other short-term tests for reproductive effects provide adequate data to evaluate the effects of these bridged alkyl phenols on reproduction. It can be concluded that reproductive toxicity is low.

Typically a two-year chronic feeding study provides data for 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). No adverse effects were noted on reproductive organs

Genotoxicity: Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. Adequate bacterial gene mutation assays have been conducted with all of the category chemicals except two. Chromosome aberration studies, *in vitro* and/or *in vivo*, are available for all but two substances. The mutagenicity data span the range of structures and molecular weights and data can be bridged from other members of the group to meet any outstanding requirements. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.

Carcinogenicity: The mutagenicity data combined with the animal data plus the long historical use of BHT (128-37-0) indicate that the chemicals in this class are not expected to exhibit any significant potential to cause cancer. The weight of the evidence indicates that these chemicals are not genotoxic. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

For hindered phenols:

Available data shows that acute toxicity of these substances is low.

Mutagenicity. Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.

In Vitro Chromosome Aberration Studies. *In vitro* chromosome aberration studies are available for several members. All except 2,6-di-tert-butyl-p-cresol were negative

In Vivo Chromosome Aberration Studies. *In vivo* studies evaluating chromosome damage are available for six of the hindered phenols. All *in vivo* evaluations were negative.

Repeated Dose Toxicity. Repeated dose toxicity data of approximately three months (90-day, 12- and 13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day)

Carcinogenicity: Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats

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.

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.

* Degussa SDS

OCTABENZONE

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.

**[ICI]

DI-SEC-OCTYL
PHTHALATE

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 dermal 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.

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.

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,

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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.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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.

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.

One member of this subcategory, diethylhexyl phthalate (DEHP), has been tested for acute inhalation toxicity. It did not cause an effect at the 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 (PPARα) and that levels of PPARα 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. Diisoheptyl 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-isoheptyl 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 "711 P" 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.

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.

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.

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), foetolethality recorded.

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ISOPROPANOL

For isopropanol (IPA):

Acute toxicity: Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat.

Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred.

Repeat dose studies: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney.

Reproductive toxicity: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.

Developmental toxicity: The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity.

Genotoxicity: All genotoxicity assays reported for isopropanol have been negative.

Carcinogenicity: rodent inhalation studies were conducted to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment.

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.

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.

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

Legend: ✓ – Data required to make classification available
 ✗ – Data available but does not fill the criteria for classification
 ⊖ – Data Not Available to make classification

CMR STATUS

REPROTOXIN	toluene	ILO Chemicals in the electronics industry that have toxic effects on reproduction				
	methyl ethyl ketone	ILO Chemicals in the electronics industry that have toxic effects on reproduction				
	xylene	ILO Chemicals in the electronics industry that have toxic effects on reproduction				
SKIN	toluene	New Zealand Workplace Exposure Standards (WES) - Skin			Skin absorption	
	n-butanol	New Zealand Workplace Exposure Standards (WES) - Skin			Skin absorption	

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
resins	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
toluene	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
n-butyl acetate	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
methyl ethyl ketone	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
n-butanol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
xylene	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
octabenzene	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
di-sec-octyl phthalate	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
isopropanol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

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.

Continued...

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

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

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

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

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

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

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

For ketones:

Ketones, unless they are alpha, beta-unsaturated ketones, can be considered as narcosis or baseline toxicity compounds

Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrate. The higher molecular weight ketones do not form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions

Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (e.g., ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable.

Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water.

They are unlikely to bioconcentrate or biomagnify.

For toluene:

log Kow : 2.1-3

log Koc : 1.12-2.85

Koc : 37-260

log Kom : 1.39-2.89

Half-life (hr) air : 2.4-104

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

Half-life (hr) H2O ground : 168-2628

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

Henry's Pa m3/mol: 518-694

Henry's atm m3/mol: 5.94E-03

BOD 5 0.86-2.12, 5%

COD : 0.7-2.52, 21-27%

ThOD : 3.13

BCF : 1.67-380

log BCF : 0.22-3.28

Environmental fate:

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

Transformation/Persistence:

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

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

Soil - In surface soil, volatilization 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 volatilization 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)
n-butyl acetate	LOW	LOW
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
2,6-di-tert-butyl-4-methylphenol	HIGH	HIGH
octabenzene	LOW	LOW
di-sec-octyl phthalate	HIGH (Half-life = 389 days)	LOW (Half-life = 1.21 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
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toluene	LOW (BCF = 90)
n-butyl acetate	LOW (BCF = 14)
methyl ethyl ketone	LOW (LogKOW = 0.29)
n-butanol	LOW (BCF = 64)
xylene	MEDIUM (BCF = 740)
2,6-di-tert-butyl-4-methylphenol	HIGH (BCF = 2500)
octabenzene	LOW (BCF = 190)
di-sec-octyl phthalate	HIGH (BCF = 24500)
isopropanol	LOW (LogKOW = 0.05)

Mobility in soil

Ingredient	Mobility
toluene	LOW (KOC = 268)
n-butyl acetate	LOW (KOC = 20.86)
methyl ethyl ketone	MEDIUM (KOC = 3.827)
n-butanol	MEDIUM (KOC = 2.443)
2,6-di-tert-butyl-4-methylphenol	LOW (KOC = 23030)
octabenzene	LOW (KOC = 92060)
di-sec-octyl phthalate	LOW (KOC = 165400)
isopropanol	HIGH (KOC = 1.06)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and MSDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> Reduction Reuse Recycling Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> DO NOT allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. Explosives which are surplus, deteriorated or considered unsafe for transport, storage or use shall be destroyed and the statutory authorities shall be notified. Explosives must not be thrown away, buried, discarded or placed with garbage. This material may be disposed of by burning or detonation but the operation must be performed under the control of a person competent in the destruction of explosives. <p>Disposal by detonation:</p> <ul style="list-style-type: none"> The explosives to be destroyed must be placed in direct contact with fresh priming charge in a hole which is at least 0.6 metre deep and then adequately stemmed. No detonators shall be inserted into defective explosives. Personnel must be evacuated to a safe distance prior to initiation/firing of the charge. <p>Disposal by burning:</p> <ul style="list-style-type: none"> Make a sawdust bed or trail adequate for the quantity of explosives to be burned, approximately 400 mm wide and 40 mm deep, upon which the explosive will be laid. If sawdust is not available, newspaper may be used. Normal precautions shall be taken to avoid the spread of fire. Individual trails should not be closer together than 600 mm and should contain not more than 12 kg of explosive. Trails should be side by side, NOT in-line, and not more than four should be set up at one time. Remove any explosive that is not to be burnt to a distance of at least 300 metre. Sufficient diesel oil (never petrol or other highly flammable liquid) should be used to thoroughly wet the sawdust (or paper) at least 4 litre per trail is recommended. Light the trail from a long, rolled paper wick which should be placed downwind and in contact with the end 1m of trail that is not covered with explosive. The wind should blow so that the flame from the wick (and later from the burning explosive) will blow away from the unburned explosive as detonation is more likely to occur if the explosive is preheated by the flame. If plastic igniter cord (slow) is available, its use for lighting is recommended instead of paper. One end should be coiled into the sawdust or under the paper and the other end lit from a minimum distance of 7m from the trail. Retire at least 300m or to a safe place. DO NOT return to the site for at least 30 minutes after the burning has apparently finished. If the fire goes out do not approach for at least 15 minutes after all trace of fire has gone. DO NOT add more diesel oil unless certain that the flame is completely extinguished. <p>[DYNO]</p> <ul style="list-style-type: none"> Recycle wherever possible.


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- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

Ensure that the disposal of material is carried out in accordance with Hazardous Substances (Disposal) Regulations 2001.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	NO
HAZCHEM	•3YE

Land transport (UN)

UN number	1263
Packing group	II
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Environmental hazard	No relevant data
Transport hazard class(es)	Class : 3 Subrisk : Not Applicable
Special precautions for user	Special provisions : 163;367 Limited quantity : 5 L

Air transport (ICAO-IATA / DGR)

UN number	1263
Packing group	II
UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class : 3 ICAO / IATA Subrisk : Not Applicable ERG Code : 3L
Special precautions for user	Special provisions : A3 A72 A192 Cargo Only Packing Instructions : 364 Cargo Only Maximum Qty / Pack : 60 L Passenger and Cargo Packing Instructions : 353 Passenger and Cargo Maximum Qty / Pack : 5 L Passenger and Cargo Limited Quantity Packing Instructions : Y341 Passenger and Cargo Limited Maximum Qty / Pack : 1 L

Sea transport (IMDG-Code / GGVSee)

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

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

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	toluene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	n-butyl acetate	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	methyl ethyl ketone	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	xylene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	di-sec-octyl phthalate	X

SECTION 15 REGULATORY INFORMATION

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002596	Laboratory Chemicals and Reagent Kits Group Standard 2006
HSR002584	Fuel Additives (Flammable, Toxic [6.7]) Group Standard 2006
HSR002589	Industrial and Institutional Cleaning Products (Flammable, Toxic [6.7]) Group Standard 2006
HSR002622	N.O.S. (Flammable, Toxic [6.1, 6.7]) Group Standard 2006
HSR002669	Surface Coatings and Colourants (Flammable, Toxic [6.7]) Group Standard 2006
HSR002643	Polymers (Flammable, Toxic [6.7]) Group Standard 2006
HSR002502	Additives, Process Chemicals and Raw Materials (Flammable, Toxic [6.7]) Group Standard 2006
HSR002563	Embalming Products (Flammable) Group Standard 2006
HSR100425	Pharmaceutical Active Ingredients Group Standard 2010
HSR002604	Lubricants (Flammable, Toxic [6.7]) Group Standard 2006
HSR002652	Solvents (Flammable, Toxic [6.7]) Group Standard 2006
HSR100757	Veterinary Medicine (Limited Pack Size, Finished Dose) Standard 2012
HSR100758	Veterinary Medicines (Non-dispersive Closed System Application) Group Standard 2012
HSR100759	Veterinary Medicines (Non-dispersive Open System Application) Group Standard 2012

toluene(108-88-3) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)", "International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs", "New Zealand Workplace Exposure Standards (WES)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals"
n-butyl acetate(123-86-4) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)", "New Zealand Workplace Exposure Standards (WES)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals"
methyl ethyl ketone(78-93-3) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)", "New Zealand Workplace Exposure Standards (WES)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals"
n-butanol(71-36-3) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)", "New Zealand Workplace Exposure Standards (WES)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals"
xylene(1330-20-7) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)", "International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs", "New Zealand Workplace Exposure Standards (WES)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals"
2,6-di-tert-butyl-4-methylphenol(128-37-0) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)", "International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs", "New Zealand Workplace Exposure Standards (WES)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals"
octabenzene(1843-05-6) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)"
di-sec-octyl phthalate(117-81-7) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)", "International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs", "New Zealand Workplace Exposure Standards (WES)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals"
isopropanol(67-63-0) is found on the following regulatory lists	"New Zealand Inventory of Chemicals (NZIoC)", "International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs", "New Zealand Workplace Exposure Standards (WES)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals"

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
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toluene	108-88-3	601-021-00-3	01-2119471310-51-XXXX
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Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2, Asp. Tox. 1, Skin Irrit. 2, STOT SE 3, Repr. 2, STOT RE 2	GHS02, GHS08, Dgr	H225, H304, H315, H336, H361, H373
2	Flam. Liq. 2, Asp. Tox. 1, Skin Irrit. 2, Aquatic Chronic 2, STOT RE 1, Aquatic Chronic 3, Repr. 1A, Acute Tox. 4, Muta. 1B, Carc. 1A, STOT SE 1, Skin Sens. 1	GHS08, Dgr, GHS09, GHS01, GHS06	H225, H304, H315, H319, H411, H372, H362, H301, H332, H228, H360, H340, H350, H370

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

Ingredient	CAS number	Index No	ECHA Dossier
n-butyl acetate	123-86-4	607-025-00-1	01-2119485493-29-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 3, STOT SE 3	GHS07, GHS02, Wng	H226, H336
2	STOT SE 3, Aquatic Chronic 1, Flam. Liq. 2, Skin Irrit. 2, Eye Irrit. 2, Acute Tox. 2	Wng, GHS01, Dgr, GHS06	H336, H319, H225, H315, H330, H335, H317

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

Ingredient	CAS number	Index No	ECHA Dossier
methyl ethyl ketone	78-93-3	606-002-00-3	01-2119457290-43-XXXX, 01-2119943742-35-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3	GHS07, GHS02, Dgr	H225, H319, H336
2	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3, Skin Irrit. 2, Eye Irrit. 2A	Dgr, Wng, GHS01, GHS08	H225, H319, H371, H312, H302, H341, H361, H314

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

Ingredient	CAS number	Index No	ECHA Dossier
n-butanol	71-36-3	603-004-00-6	01-2119484630-38-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 3, Acute Tox. 4, Skin Irrit. 2, Eye Dam. 1, STOT SE 3	GHS02, GHS05, Dgr	H226, H302, H315, H318, H335, H336
2	Flam. Liq. 3, Acute Tox. 4, Skin Irrit. 2, Eye Dam. 1, STOT SE 3, Acute Tox. 3, Asp. Tox. 1, STOT RE 1	GHS02, GHS05, Dgr, GHS08, GHS06	H315, H318, H370, H301, H332, H225, H304, H372

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

Ingredient	CAS number	Index No	ECHA Dossier
xylene	1330-20-7	601-022-00-9	01-2119488216-32-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 3, Acute Tox. 4, Skin Irrit. 2	GHS07, GHS02, Wng	H226, H312, H315, H332
2	Acute Tox. 4, Skin Irrit. 2, Asp. Tox. 1, Eye Irrit. 2, Acute Tox. 3, Aquatic Chronic 2, Repr. 1B, STOT SE 1, STOT RE 1, Flam. Liq. 2	Wng, GHS08, Dgr, GHS01, GHS09	H312, H315, H332, H304, H411, H360, H370, H372, H225, H318
1	Flam. Liq. 3, Acute Tox. 4, Skin Irrit. 2	GHS07, GHS02, Wng	H226, H312, H315, H332
2	Flam. Liq. 3, Acute Tox. 4, Skin Irrit. 2	GHS07, GHS02, Wng	H226, H312, H315, H332

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

Ingredient	CAS number	Index No	ECHA Dossier
2,6-di-tert-butyl-4-methylphenol	128-37-0	Not Available	01-2119480433-40-XXXX, 01-2119565113-46-XXXX, 01-2119555270-46-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Aquatic Acute 1, Aquatic Chronic 1	GHS09, Wng	H410
2	Aquatic Acute 1, Aquatic Chronic 1, Acute Tox. 4, Skin Irrit. 2, Eye Irrit. 2, STOT RE 2, Skin Sens. 1, STOT SE 1, Muta. 1B, Repr. 2, Resp. Sens. 1, Carc. 1B	GHS09, Wng, Dgr, GHS08, GHS05, GHS02, GHS06, GHS03	H410, H400, H302, H315, H319, H373, H317, H370, H340, H361, H311, H331, H350

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

Ingredient	CAS number	Index No	ECHA Dossier
octabenzene	1843-05-6	Not Available	01-2119557833-30-XXXX

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Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Sens. 1, Aquatic Chronic 4	GHS07, Wng	H317, H413
2	Skin Sens. 1, Flam. Liq. 3, Aquatic Chronic 1, Skin Irrit. 2, Aquatic Acute 1, Skin Sens. 1B, Eye Irrit. 2	GHS07, Wng, GHS02, GHS09	H317, H226, H410, H315, H319

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

Ingredient	CAS number	Index No	ECHA Dossier
di-sec-octyl phthalate	117-81-7	607-317-00-9	01-2119484611-38-XXXX, 01-0000012018-79-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Repr. 1B	GHS08, Dgr	H360
2	Repr. 1B, Lact., Aquatic Acute 1, Aquatic Chronic 1, Eye Irrit. 2, Repr. 1A, Carc. 2	GHS08, Dgr, GHS09	H360, H362, H410, H319, H400

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

Ingredient	CAS number	Index No	ECHA Dossier
isopropanol	67-63-0	603-117-00-0	01-2119457558-25-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2, Eye Irrit. 2, STOT SE 3	GHS07, GHS02, Dgr	H225, H319, H336
2	Flam. Liq. 2, Eye Irrit. 2, STOT SE 1, Eye Irrit. 2A, Repr. 2, STOT RE 2	GHS02, Dgr, GHS08, GHS03	H225, H319, H370, H312, H340, H302, H361, H373

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

Location Test Certificate

Subject to Regulation 55 of the Hazardous Substances (Classes 1 to 5 Controls) Regulations, a location test certificate is required when quantity greater than or equal to those indicated below are present.

Hazard Class	Quantity beyond which controls apply for closed containers	Quantity beyond which controls apply when use occurring in open containers
3.1B	100 L in containers greater than 5 L 250 L in containers up to and including 5 L	50 L 50 L

Approved Handler

Subject to Regulation 56 of the Hazardous Substances (Classes 1 to 5 Controls) Regulations and Regulation 9 of the Hazardous Substances (Classes 6, 8, and 9 Controls) Regulations, the substance must be under the personal control of an Approved Handler when present in a quantity greater than or equal to those indicated below.

Class of substance	Quantities
3.1B	250 L (when in containers greater than 5 L) 500 L (when in containers up to and including 5 L)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Y
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

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.

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

www.chemwatch.net

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

Notes

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