



MIROTONE

MIROTHANE PU 5608

Mirotone (NZ) Ltd

Chemwatch: 42-5595

Version No: 4.1.1.1

Safety Data Sheet according to HSNO Regulations

Chemwatch Hazard Alert Code: 3

Issue Date: 07/05/2015

Print Date: 22/04/2016

Initial Date: Not Available

L.GHS.NZL.EN

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

Product Identifier

Product name	MIROTHANE PU 5608
Synonyms	MIROTHANE PU 5608/30 Satin CTB, MIROTHANE PU 5608/30 Satin WTB, Product Code: 5608-3A-WB, 5608-3A-CTB
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. For full details on application and properties consult the technical datasheet. Two-pack rapid cure pigmented polyurethane topcoat used for fixtures and furniture finishing. Designed for interior use only.
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Details of the supplier of the safety data sheet

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	
Toxicity	2	
Body Contact	2	
Reactivity	1	
Chronic	2	

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

Classification [1]	Flammable Liquid Category 2, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Reproductive Toxicity
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Continued...

	Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Specific target organ toxicity - repeated exposure Category 2, Aspiration Hazard Category 1
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 (oral), 6.1E (aspiration), 6.3A, 6.4A, 6.8B, 6.9 (narcotic), 6.9B (inhalation)

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.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H361	Suspected of damaging fertility or the unborn child.
H336	May cause drowsiness or dizziness.
H373	May cause damage to organs.
H304	May be fatal if swallowed and enters airways.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P281	Use personal protective equipment as required.
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.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P331	Do NOT induce vomiting.
P362	Take off contaminated clothing and wash before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
108-88-3	20-40	<u>toluene</u>
13463-67-7	0-30	<u>titanium dioxide</u>
Not Available	10-30	resins
123-86-4	5-30	<u>n-butyl acetate</u>
78-93-3	1-10	<u>methyl ethyl ketone</u>
67-63-0	1-10	<u>isopropanol</u>
	balance	Ingredients determined not to be hazardous

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"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes 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.
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.

Treat symptomatically.

for simple esters:

BASIC TREATMENT

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

ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

Following acute or short term repeated exposures to toluene:

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

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

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

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

For acute or short term repeated exposures to xylene:

- ▶ Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- ▶ Pulmonary absorption is rapid with about 60-65% retained at rest.
- ▶ Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ < 50 mm Hg or pCO₂ > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
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BIOLOGICAL EXPOSURE INDEX - BEI

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

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

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- ▶ 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
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ Fight fire from a safe distance, with adequate cover. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control the fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ Do not approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Liquid and vapour are highly flammable. ▶ Severe fire hazard when exposed to heat, flame and/or oxidisers. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include: carbon dioxide (CO₂) 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	<ul style="list-style-type: none"> ▶ Remove all ignition sources. ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb small quantities with vermiculite or other absorbent material. ▶ Wipe up. ▶ Collect residues in a flammable waste container. 																																																																											
Major Spills	<p>Chemical Class: ester and ethers For release onto land: recommended sorbents listed in order of priority.</p> <table border="1"> <thead> <tr> <th>SORBENT TYPE</th> <th>RANK</th> <th>APPLICATION</th> <th>COLLECTION</th> <th>LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>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> <tr> <td colspan="5">LAND SPILL - MEDIUM</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>blower</td> <td>skid loader</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>2</td> <td>throw</td> <td>skid loader</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skid loader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skid loader</td> <td>W, SS, DGC</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skid loader</td> <td>R, I, W, P, DGC</td> </tr> <tr> <td>wood fiber - particulate</td> <td>4</td> <td>blower</td> <td>skid loader</td> <td>R, W, P, DGC</td> </tr> </tbody> </table> <p>Legend DGC: Not effective where ground cover is dense R: Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT: Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse /absorb vapour. ▶ Contain spill with sand, earth or vermiculite. ▶ Use only spark-free shovels and explosion proof equipment. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services. 	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					cross-linked polymer - particulate	1	shovel	shovel	R, W, SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R, I, P	wood fiber - particulate	3	shovel	shovel	R, W, P, DGC	wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT	treated wood fiber - pillow	3	throw	pitchfork	DGC, RT	LAND SPILL - MEDIUM					cross-linked polymer - particulate	1	blower	skid loader	R, W, SS	cross-linked polymer - pillow	2	throw	skid loader	R, DGC, RT	sorbent clay - particulate	3	blower	skid loader	R, I, P	polypropylene - particulate	3	blower	skid loader	W, SS, DGC	expanded mineral - particulate	4	blower	skid loader	R, I, W, P, DGC	wood fiber - particulate	4	blower	skid loader	R, W, P, DGC
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Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<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.
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	<ul style="list-style-type: none"> ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights, heat or ignition sources. ▶ When handling, DO NOT eat, drink or smoke. ▶ Vapour may ignite on pumping or pouring due to static electricity. ▶ DO NOT use plastic buckets. ▶ Earth and secure metal containers when dispensing or pouring product. ▶ Use spark-free tools when handling. ▶ Avoid contact with incompatible materials. ▶ Keep containers securely sealed. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
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 SDS.

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

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)	titanium dioxide	Titanium dioxide	10 mg/m ³	Not Available	Not Available	The value for inhalable dust containing no asbestos and less than 1% free silica.
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)	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
titanium dioxide	Titanium oxide; (Titanium dioxide)	10 mg/m ³	10 mg/m ³	10 mg/m ³
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
isopropanol	Isopropyl alcohol	400 ppm	400 ppm	12000 ppm

Ingredient	Original IDLH	Revised IDLH
toluene	2,000 ppm	500 ppm
titanium dioxide	N.E. mg/m ³ / N.E. ppm	5,000 mg/m ³
resins	Not Available	Not Available
n-butyl acetate	10,000 ppm	1,700 [LEL] ppm
methyl ethyl ketone	3,000 ppm	3,000 [Unch] ppm
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)

Animals exposed by inhalation to 10 mg/m3 titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

for propylene glycol monomethyl ether acetate (PGMEA)

Saturated vapour concentration: 4868 ppm at 20 C.

A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternbral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

For toluene:

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

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

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

Odour Safety Factor(OSF)

OSF=17 (TOLUENE)

for xylenes:

IDLH Level: 900 ppm

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

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

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

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

Odour Safety Factor(OSF)

OSF=4 (XYLENE)

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 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

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>	
	<p>Type of Contaminant:</p> <p>solvent, vapours, degreasing etc., evaporating from tank (in still air).</p>	<p>Air Speed:</p> <p>0.25-0.5 m/s (50-100 f/min.)</p>
	<p>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)</p>	<p>0.5-1 m/s (100-200 f/min.)</p>
	<p>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</p>	<p>1-2.5 m/s (200-500 f/min.)</p>
<p>Within each range the appropriate value depends on:</p>		
<p>Lower end of the range</p> <p>1: Room air currents minimal or favourable to capture</p>	<p>Upper end of the range</p> <p>1: Disturbing room air currents</p>	

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

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

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Material	CPI
##n-butyl	acetate
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C

Respiratory protection

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

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

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)

* 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.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Colourless to slightly amber to white low viscosity highly flammable liquid with a characteristic solvent odour; not miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.9-1.3
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>333
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	100-500 @25C
Initial boiling point and boiling range (°C)	107	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	7-8	Taste	Not Available
Evaporation rate	2.7-2.8 BuAC = 1	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	7.7	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.4	Volatile Component (%vol)	57-88 (VOC = 547-699 g/l)
Vapour pressure (kPa)	4.7-5.1	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	3.1-3.2	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ 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>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.</p> <p>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.</p> <p>Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.</p> <p>Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed</p> <p>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 odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal.</p> <p>Acute exposure of humans to high concentrations of methyl ethyl ketone produces irritation to the eyes, nose, and throat. Other effects reported from acute inhalation exposure in humans include central nervous system depression, headache, and nausea.</p> <p>Easy odour recognition and irritant properties of methyl ethyl ketone means that high vapour levels are readily detected and should be avoided by application of control measures; however odour fatigue may occur with loss of warning of exposure.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis</p>
Skin Contact	<p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<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>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>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body 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>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Chronic toluene habituation occurs following intentional abuse (glue sniffing) or from occupational exposure. Ataxia, incoordination and tremors of the hands and feet (as a consequence of diffuse cerebral atrophy), headache, abnormal speech, transient memory loss, convulsions, coma, drowsiness, reduced colour perception, frank blindness, nystagmus (rapid, involuntary eye-movements), hearing loss leading to deafness and mild dementia have all been associated with chronic abuse. Peripheral nerve damage, encephalopathy, giant axonopathy electrolyte disturbances in the cerebrospinal fluid and abnormal computer tomographic (CT scans) are common amongst toluene addicts. Although toluene abuse has been linked with kidney disease, this does not commonly appear in cases of occupational toluene exposures. Cardiac and haematological toxicity are however associated with chronic toluene exposures. Cardiac arrhythmia, multifocal and premature ventricular contractions and supraventricular tachycardia are present in 20% of patients who abused toluene-containing paints.</p> <p>Previous suggestions that chronic toluene inhalation produced human peripheral neuropathy have been discounted. However central nervous system (CNS) depression is well documented where blood toluene exceeds 2.2 mg%. Toluene abusers can achieve transient circulating concentrations of 6.5 %. Amongst workers exposed for a median time of 29 years, to toluene, no subacute effects on neuroathenic complaints and psychometric test results could be established.</p> <p>The prenatal toxicity of very high toluene concentrations has been documented for several animal species and man. Malformations indicative of specific teratogenicity have not generally been found. Neonatal toxicity, described in the literature, takes the form of embryo death or delayed foetal growth and delayed skeletal system development. Permanent damage of children has been seen only when mothers have suffered from chronic intoxication as a result of "sniffing".</p> <p>Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia.</p> <p>Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.</p> <p>Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.</p> <p>Xylene has been classed as a developmental toxin in some jurisdictions.</p> <p>Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In</p>

all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.

Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain.

Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals.

There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol.

Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects.

NOTE: Commercial isopropanol does not contain "isopropyl oil". An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct "isopropyl oil". Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.

MIROTHANE PU 5608	TOXICITY	IRRITATION
	Not Available	Not Available
toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation (rat) LC50: >26700 ppm/1hd ^[2]	Eye (rabbit): 0.87 mg - mild
	Inhalation (rat) LC50: 49 mg/L/4h ^[2]	Eye (rabbit): 100 mg/30sec - mild
	Oral (rat) LD50: 636 mg/kg ^[2]	Skin (rabbit): 20 mg/24h-moderate Skin (rabbit): 500 mg - moderate
titanium dioxide	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2.28 mg/l4 h ^[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Inhalation (rat) LC50: >3.56 mg/l4 h ^[1]	
	Inhalation (rat) LC50: >6.82 mg/l4 h ^[1]	
	Inhalation (rat) LC50: 3.43 mg/l4 h ^[1]	
	Oral (rat) LD50: >2000 mg/kg ^[1]	
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 Skin (rabbit): 500 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 Skin (rabbit): 402 mg/24 hr - mild Skin (rabbit): 13.78mg/24 hr open
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 Eye (rabbit): 100mg/24hr-moderate Skin (rabbit): 500 mg - mild

Legend: 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

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No significant acute toxicological data identified in literature search.

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

For 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 sensitiser and fatal cardiotoxin.

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

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

Developmental/Reproductive Toxicity

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

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

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

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

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

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

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

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

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 of 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 sensitiser and fatal cardiotoxin.

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

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

Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely affect 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.

TOLUENE

	<p>Animals - Sterebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m³. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.</p> <p>Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor. Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene.</p> <p>Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues.</p> <p>Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites</p> <p>Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.</p>
TITANIUM DIOXIDE	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>For titanium dioxide:</p> <p>Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.</p> <p>Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.</p> <p>Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.</p> <p>Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.</p> <p>Animal carcinogenicity data</p> <p>Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.</p> <p>In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.</p> <p>Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.</p> <p>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. * IUCLID</p>
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>

	Combinations with chloroform also show increase in toxicity
ISOPROPANOL	<p>For isopropanol (IPA):</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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</p> <p>Genotoxicity: All genotoxicity assays reported for isopropanol have been negative</p> <p>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</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>

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

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
toluene	BCF	24	Algae or other aquatic plants	10mg/L	4
toluene	EC50	3	Algae or other aquatic plants	0.1336030mg/L	4
toluene	EC50	48	Crustacea	0.01151750mg/L	4
toluene	EC50	72	Algae or other aquatic plants	12.5mg/L	4
toluene	LC50	96	Fish	0.0031704mg/L	4
toluene	NOEC	168	Crustacea	0.74mg/L	2
titanium dioxide	LC50	96	Fish	9.214mg/L	3
titanium dioxide	EC50	72	Algae or other aquatic plants	5.83mg/L	4
titanium dioxide	NOEC	336	Fish	0.089mg/L	4
titanium dioxide	EC50	48	Crustacea	1.23mg/L	2
titanium dioxide	EC50	504	Crustacea	0.46mg/L	2
n-butyl acetate	EC50	48	Crustacea	=32mg/L	1
n-butyl acetate	EC50	96	Algae or other aquatic plants	1.675mg/L	3
n-butyl acetate	EC50	96	Fish	18mg/L	2
n-butyl acetate	LC50	96	Fish	18mg/L	2
n-butyl acetate	NOEC	504	Crustacea	23mg/L	2
methyl ethyl ketone	EC50	384	Crustacea	52.575mg/L	3
methyl ethyl ketone	LC50	96	Fish	228.130mg/L	3
methyl ethyl ketone	EC50	96	Algae or other aquatic plants	>500mg/L	4
methyl ethyl ketone	EC50	48	Crustacea	308mg/L	2
methyl ethyl ketone	NOEC	48	Crustacea	68mg/L	2
isopropanol	EC50	384	Crustacea	42.389mg/L	3

Continued...

isopropanol	EC50	96	Algae or other aquatic plants	993.232mg/L	3
isopropanol	LC50	96	Fish	183.844mg/L	3
isopropanol	NOEC	5760	Fish	0.02mg/L	4
isopropanol	EC50	48	Crustacea	12500mg/L	5

Legend:

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

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 methyl ethyl ketone:

log Kow : 0.26-0.69

log Koc : 0.69

Koc : 34

Half-life (hr) air : 2.3

Half-life (hr) H₂O surface water : 72-288

Henry's atm m³/mol: 1.05E-05

BOD 5 : 1.5-2.24, 46%

COD : 2.2-2.31, 100%

ThOD : 2.44

BCF : 1

Environmental fate:

TERRESTRIAL FATE: Measured Koc values of 29 and 34 were obtained for methyl ethyl ketone in silt loams. Methyl ethyl ketone is expected to have very high mobility in soil. Volatilisation of methyl ethyl ketone from dry soil surfaces is expected based upon an experimental vapor pressure of 91 mm Hg at 25 deg C. Volatilization from moist soil surfaces is also expected given the measured Henry's Law constant of 4.7x10⁻⁵ atm-cu m/mole. The volatilisation half-life of methyl ethyl ketone from silt and sandy loams was measured as 4.9 days. Methyl ethyl ketone is expected to biodegrade under both aerobic and anaerobic conditions as indicated by numerous screening tests.

AQUATIC FATE: Based on Koc values, methyl ethyl ketone is not expected to adsorb to suspended solids and sediment in water. Methyl ethyl ketone is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated half-lives for a model river and model lake are 19 and 197, hours respectively. Biodegradation of this compound is expected based upon numerous screening tests. An estimated BCF value of 1 based on an experimental log Kow of 0.29, suggests that bioconcentration in aquatic organisms is low.

ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatiles organic compounds in the atmosphere, methyl ethyl ketone, which has an experimental vapor pressure of 91 mm Hg at 25 deg C, will exist solely as a vapor in the ambient atmosphere. Vapour-phase methyl ethyl ketone is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 14 days. Methyl ethyl ketone is also expected to undergo photodecomposition in the atmosphere by natural sunlight. Photochemical degradation of methyl ethyl ketone by natural sunlight is expected to occur at approximately 1/5 the rate of degradation by photochemically produced hydroxyl radicals.

Ecotoxicity:

Fish LC50 (24 h): bluegill sunfish (*Lepomis macrochirus*) 1690-5640 mg/l; guppy (*Lebistes reticulatus*) 5700 mg/l; goldfish (*Carassius auratus*) >5000 mg/l

Fish LC50 (96 h): fathead minnow (*Pimephales promelas*) 3200 mg/l; bluegill sunfish (*Lepomis macrochirus*) 4467 mg/l; mosquito fish (*Gambusia affinis*) 5600 mg/l

Daphnia magna LC50 (48 h): <520-1382 mg/l

Daphnia magna LC50 (24 h): 8890 mg/l

Brine shrimp (*Artemia salina*) LC50 (24 h): 1950 mg/l

For xylenes:

log Koc : 2.05-3.08

Koc : 25.4-204

Half-life (hr) air : 0.24-42

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

Half-life (hr) H₂O ground : 336-8640

Half-life (hr) soil : 52-672

Henry's Pa m³/mol: 637-879

Henry's atm m³/mol: 7.68E-03

BOD 5 if unstated: 1.4,1%

COD : 2.56,13%

ThOD : 3.125

BCF : 23

log BCF : 1.17-2.41

Environmental Fate

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

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

Atmospheric fate:

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

According to a model of gas/particle partitioning of semivolatiles organic compounds in the atmosphere and from its vapour pressure, p-xylene, is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase p-xylene is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 16 hours. A half-life of 1.0 hr in summer and 10 hr in winter was measured for the reaction of p-xylene with photochemically-produced hydroxyl radicals. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers, with loss rates varying from 9-42% per hr. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzyl nitrate, m-tolaldehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

Ecotoxicity:

for xylenes

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

Daphnia EC50 948 h): 3.83 mg/l
 Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/l
 Gammarus lacustris LC50 (48 h): 0.6 mg/l

For 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)

For n-butyl acetate:

Half-life (hr) air : 144

Half-life (hr) H₂O surface water : 178-27156

Henry's atm m³/mol: 3.20E-04

BOD 5 if unstated: 0.15-1.02, 7%

COD : 78%

ThOD : 2.207

BCF : 4-14

Environmental Fate:

TERRESTRIAL FATE: An estimated Koc value of 200 determined from a measured log Kow of 1.78 indicates that n-butyl acetate is expected to have moderate mobility in soil. Volatilization of n-butyl acetate is expected from moist soil surfaces given its Henry's Law constant of 2.8x10⁻⁴ atm-cu m/mole. Volatilization from dry soil surfaces is expected based on a measured vapor pressure of 11.5 mm Hg. Using a standard BOD dilution technique and a sewage inoculum, theoretical BODs of 56 % to 86 % were observed during 5-20 day incubation periods, which suggests that n-butyl acetate may biodegrade in soil.

AQUATIC FATE: An estimated Koc value indicates that n-butyl acetate is not expected to adsorb to suspended solids and sediment in water. Butyl acetate is expected to volatilize from water surfaces based on a Henry's Law constant of 2.8x10⁻⁴ atm-cu m/mole. Estimated half-lives for a model river and model lake are 7 and 127, hours respectively. An estimated BCF value of 10 based on the log Kow, suggests that bioconcentration in aquatic organisms is low. Using a filtered sewage seed, 5-day and 20-day theoretical BODs of 58 % and 83 % were measured in freshwater dilution tests; 5-day and 20-day theoretical BODs of 40 % and 61 % were measured in salt water. A 5-day theoretical BOD of 56.8 % and 51.8 % were measured for n-butyl acetate in distilled water and seawater, respectively. Hydrolysis may be an important environmental fate for this compound based upon experimentally determined hydrolysis half-lives of 114 and 11 days at pH 8 and 9 respectively.

ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, n-butyl acetate, which has a vapour pressure of 11.5 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapour-phase n-butyl acetate is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 4 days

Environmental fate:

Fish LC50 (96 h, 23 C): island silverside (*Menidia beryllina*) 185 ppm (static bioassay in synthetic seawater, mild aeration applied after 24 h); bluegill sunfish (*Lepomis macrochirus*) 100 ppm (static bioassay in fresh water, mild aeration applied after 24 h)

Fish EC50 (96 h): fathead minnow (*Pimephales promelas*) 18 mg/l (affected fish lost equilibrium prior to death)

Daphnia LC50 (48 h): 44 ppm

Algal LC50 (96 h): *Scenedesmus* 320 ppm

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
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toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
titanium dioxide	HIGH	HIGH
n-butyl acetate	LOW	LOW
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
toluene	LOW (BCF = 90)
titanium dioxide	LOW (BCF = 10)
n-butyl acetate	LOW (BCF = 14)
methyl ethyl ketone	LOW (LogKOW = 0.29)
isopropanol	LOW (LogKOW = 0.05)

Mobility in soil

Ingredient	Mobility
toluene	LOW (KOC = 268)
titanium dioxide	LOW (KOC = 23.74)
n-butyl acetate	LOW (KOC = 20.86)
methyl ethyl ketone	MEDIUM (KOC = 3.827)
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 SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Dispose of by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material). ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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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	Not Applicable	
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	Not Applicable	
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 solutions, 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	163 367
	Limited Quantities	5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture**

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
HSR002528	Cleaning Products (Flammable) Group Standard 2006
HSR002583	Fuel Additives (Flammable) Group Standard 2006
HSR002662	Surface Coatings and Colourants (Flammable) Group Standard 2006
HSR002611	Metal Industry Products (Flammable) Group Standard 2006
HSR002621	N.O.S. (Flammable) Group Standard 2006
HSR002641	Polymers (Flammable) Group Standard 2006
HSR002637	Photographic Chemicals (Flammable) Group Standard 2006
HSR002495	Additives, Process Chemicals and Raw Materials (Flammable) Group Standard 2006
HSR002576	Food Additives and Fragrance Materials (Flammable) Group Standard 2006
HSR002563	Embalming Products (Flammable) Group Standard 2006
HSR002556	Dental Products (Flammable) Group Standard 2006

Continued...

HSR100425	Pharmaceutical Active Ingredients Group Standard 2010
HSR002599	Leather and Textile Products (Flammable) Group Standard 2006
HSR002603	Lubricants (Flammable) Group Standard 2006
HSR002650	Solvents (Flammable) Group Standard 2006
HSR002552	Cosmetic Products Group Standard 2006
HSR002548	Corrosion Inhibitors (Flammable) 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
HSR100628	Straight-chained Lepidopteran Sex Pheromone Group Standard 2012

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs	New Zealand Inventory of Chemicals (NZIoC)
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals	New Zealand Workplace Exposure Standards (WES)

TITANIUM DIOXIDE(13463-67-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs	New Zealand Workplace Exposure Standards (WES)
New Zealand Inventory of Chemicals (NZIoC)	

N-BUTYL ACETATE(123-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals	New Zealand Workplace Exposure Standards (WES)
New Zealand Inventory of Chemicals (NZIoC)	

METHYL ETHYL KETONE(78-93-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals	New Zealand Workplace Exposure Standards (WES)
New Zealand Inventory of Chemicals (NZIoC)	

ISOPROPANOL(67-63-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs	New Zealand Inventory of Chemicals (NZIoC)
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals	New Zealand Workplace Exposure Standards (WES)

ECHA SUMMARY

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

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 2, Asp. Tox. 1, Skin Irrit. 2, STOT SE 3, Repr. 2, STOT RE 2	GHS07, GHS02, GHS08, Dgr	H225, H304, H315, H336, H361, H373
2	Flam. Liq. 2, Asp. Tox. 1, Skin Irrit. 2, STOT SE 3, Repr. 2, STOT RE 2, Flam. Liq. 3, Eye Irrit. 2, Aquatic Chronic 2, STOT RE 1, Aquatic Chronic 3, Repr. 1A, Acute Tox. 4, Not Classified, Skin Sens. 1, STOT SE 1, Muta. 1B, Carc. 1A	GHS08, Dgr, GHS09, GHS01, GHS06	H225, H304, H315, H336, H319, H372, H362, H335, H301, H332, H360, H340, H350, H370, H228
1	Aquatic Chronic 4	GHS07, GHS02, GHS08, Dgr, GHS09, GHS01, GHS06	H225, H304, H315, H336, H361, H373, H319, H372, H362, H335, H301, H332, H360, H340, H350, H370, H228
2	Aquatic Chronic 4	GHS07, GHS02, GHS08, Dgr, GHS09, GHS01, GHS06	H225, H304, H315, H336, H361, H373, H319, H372, H362, H335, H301, H332, H360, H340, H350, H370, H228

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

Ingredient	CAS number	Index No	ECHA Dossier
titanium dioxide	13463-67-7	Not Available	01-2119954396-27-XXXX, 01-2119489379-17-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Not Classified	GHS08, Dgr, Wng	H302, H351, H315, H319, H332, H335, H372, H350, H318, H312
2	Not Classified, Acute Tox. 4, Carc. 2, Skin Irrit. 2, Eye Irrit. 2, STOT SE 3, STOT RE 1, STOT SE 2, Carc. 1B, Aquatic Chronic 4, STOT RE 2	GHS08, Dgr, Wng	H302, H351, H315, H319, H332, H335, H372, H350, H318, H312

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	Flam. Liq. 3, STOT SE 3, Aquatic Chronic 1, Flam. Liq. 2, Skin Irrit. 2, Eye Irrit. 2, Acute Tox. 2, Not Classified, Acute Tox. 4, Aquatic Chronic 2	Wng, GHS01, Dgr, GHS06, GHS08	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, Not Classified, Eye Irrit. 2A	Dgr, Wng, GHS01, GHS08	H225, H319, H336, H371, H335, H312, H341, H302, H361, H314

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 3, STOT SE 1, Not Classified, Repr. 2, STOT RE 2, Eye Irrit. 2A	GHS02, Dgr, GHS08, Wng, GHS03	H225, H319, H336, H335, H370, H340, H312, 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)

Refer Group Standards for further information

Tracking Requirements

Not Applicable

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (toluene; n-butyl acetate; isopropanol; methyl ethyl ketone)
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

Ingredients with multiple cas numbers

Name	CAS No
titanium dioxide	100292-32-8, 101239-53-6, 116788-85-3, 12000-59-8, 12188-41-9, 12701-76-7, 12767-65-6, 12789-63-8, 1309-63-3, 1317-70-0, 1317-80-2, 1344-29-2, 13463-67-7, 185323-71-1, 185828-91-5, 188357-76-8, 188357-79-1, 195740-11-5, 221548-98-7, 224963-00-2, 246178-32-5, 252962-41-7, 37230-92-5, 37230-94-7, 37230-95-8, 37230-96-9, 39320-58-6, 39360-64-0, 39379-02-7, 416845-43-7, 494848-07-6, 494848-23-6, 494851-77-3, 494851-98-8, 55068-84-3, 55068-85-4, 552316-51-5, 62338-64-1, 767341-00-4, 97929-50-5, 98084-96-9

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average

PC—STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit.

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

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