

Etch Primer

DNA Custom Paints

Chemwatch: 34-7796
Version No: 5.1.1.1
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 26/10/2015
Print Date: 26/11/2015
Initial Date: **Not Available**
L.GHS.AUS.EN

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

Product Identifier

Product name	Etch Primer
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) (contains ethanol and methyl ethyl ketone)
Other means of identification	Part No.: PE

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. Primer.
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Details of the supplier of the safety data sheet

Registered company name	DNA Custom Paints
Address	5-7 Keith Campbell Court Scoresby 3179 VIC Australia
Telephone	+61 3 9764 2088
Fax	+61 3 9764 1244
Website	www.dna-paints.com
Email	Not Available

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	+61 3 9573 3112
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	3	
Toxicity	1	
Body Contact	2	
Reactivity	1	
Chronic	2	

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

Poisons Schedule	Not Applicable
GHS Classification [1]	Flammable Liquid Category 2, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Germ Cell Mutagen Category 2, Reproductive Toxicity Category 2, STOT - SE (Narcosis) Category 3, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	
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SIGNAL WORD	DANGER
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Hazard statement(s)

H225	Highly flammable liquid and vapour
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Continued...

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H315	Causes skin irritation
H319	Causes serious eye irritation
H341	Suspected of causing genetic defects
H361	Suspected of damaging fertility or the unborn child
H336	May cause drowsiness or dizziness
H401	Toxic to aquatic life
H411	Toxic to aquatic life with long lasting effects
AUH066	Repeated exposure may cause skin dryness and cracking

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
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.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
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.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
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.
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
64-17-5	10-30	<u>ethanol</u>
78-93-3	10-30	<u>methyl ethyl ketone</u>
1330-20-7	1-10	<u>xylene</u>
108-88-3	1-10	<u>toluene</u>
7779-90-0	1-10	<u>zinc phosphate</u>
71-36-3	1-10	<u>n-butanol</u>
64742-95-6	1-10	<u>naphtha petroleum, light aromatic solvent</u>
108-95-2	1-10	<u>phenol</u>
98-82-8	1-10	<u>isopropyl benzene - cumene</u>
108-67-8	1-10	<u>1,3,5-trimethyl benzene</u>

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95-63-6

1-10

1,2,4-trimethyl benzene

SECTION 4 FIRST AID MEASURES

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. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

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.

for simple ketones:

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 (5mL/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.
- ▶ Consider intubation at first sign of upper airway obstruction resulting from oedema.
- ▶ 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

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

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: alcohols and glycols For release onto land: recommended sorbents listed in order of priority.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 30%;">SORBENT TYPE</th> <th style="width: 10%;">RANK</th> <th style="width: 30%;">APPLICATION</th> <th style="width: 10%;">COLLECTION</th> <th style="width: 20%;">LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td></td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td></td> <td>throw</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td></td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>wood fiber - pillow</td> <td>3</td> <td></td> <td>throw</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>treated wood fiber - pillow</td> <td>3</td> <td></td> <td>throw</td> <td>DGC, RT</td> </tr> <tr> <td>foamed glass - pillow</td> <td>4</td> <td></td> <td>throw</td> <td>R, P, DGC, RT</td> </tr> <tr> <td colspan="5">LAND SPILL - MEDIUM</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td></td> <td>blower</td> <td>R, W, SS</td> </tr> <tr> <td>polypropylene - particulate</td> <td>2</td> <td></td> <td>blower</td> <td>W, SS, DGC</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td></td> <td>blower</td> <td>R, I, W, P, DGC</td> </tr> <tr> <td>polypropylene - mat</td> <td>3</td> <td></td> <td>throw</td> <td>DGC, RT</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>3</td> <td></td> <td>blower</td> <td>R, I, W, P, DGC</td> </tr> <tr> <td>polyurethane - mat</td> <td>4</td> <td></td> <td>throw</td> <td>DGC, RT</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 Chemical Class: ketones For release onto land: recommended sorbents listed in order of priority.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 30%;">SORBENT TYPE</th> <th style="width: 10%;">RANK</th> <th style="width: 30%;">APPLICATION</th> <th style="width: 10%;">COLLECTION</th> <th style="width: 20%;">LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td></td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td></td> <td>throw</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td></td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>wood fiber - pillow</td> <td>3</td> <td></td> <td>throw</td> <td>R, P, DGC, RT</td> </tr> </tbody> </table>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					cross-linked polymer - particulate	1		shovel	R, W, SS	cross-linked polymer - pillow	1		throw	R, DGC, RT	sorbent clay - particulate	2		shovel	R, I, P	wood fiber - pillow	3		throw	R, P, DGC, RT	treated wood fiber - pillow	3		throw	DGC, RT	foamed glass - pillow	4		throw	R, P, DGC, RT	LAND SPILL - MEDIUM					cross-linked polymer - particulate	1		blower	R, W, SS	polypropylene - particulate	2		blower	W, SS, DGC	sorbent clay - particulate	2		blower	R, I, W, P, DGC	polypropylene - mat	3		throw	DGC, RT	expanded mineral - particulate	3		blower	R, I, W, P, DGC	polyurethane - mat	4		throw	DGC, RT	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					cross-linked polymer - particulate	1		shovel	R, W, SS	cross-linked polymer - pillow	1		throw	R, DGC, RT	sorbent clay - particulate	2		shovel	R, I, P	wood fiber - pillow	3		throw	R, P, DGC, RT
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treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
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LAND SPILL - MEDIUM

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

Legend

DGC: Not effective where ground cover is dense

R: Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT: Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

- ▶ 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.

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. ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Electrostatic discharge may be generated during pumping - this may result in fire. ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (≤ 1 m/sec until fill pipe submerged to twice its diameter, then ≤ 7 m/sec). ▶ Avoid splash filling. ▶ Do NOT use compressed air for filling discharging or handling operations. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ 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"> ▶ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ▶ Aromatics can react exothermically with bases and with diazo compounds. <p>For alkyl aromatics: The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.</p> <ul style="list-style-type: none"> ▶ Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen ▶ Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids. ▶ Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides. ▶ Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily. ▶ Alkali metals accelerate the oxidation while CO₂ as co-oxidant enhances the selectivity. ▶ Microwave conditions give improved yields of the oxidation products. ▶ Photo-oxidation products may occur following reaction with hydroxyl radicals and NO_x - these may be components of photochemical smogs. <p>Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007</p> <ul style="list-style-type: none"> ▶ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates.



+ X X X + + +

X — Must not be stored together
0 — May be stored together with specific preventions
+ — May be stored together

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	methyl ethyl ketone	Methyl ethyl ketone (MEK)	445 mg/m ³ / 150 ppm	890 mg/m ³ / 300 ppm	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p- isomers)	350 mg/m ³ / 80 ppm	655 mg/m ³ / 150 ppm	Not Available	Not Available
Australia Exposure Standards	toluene	Toluene	191 mg/m ³ / 50 ppm	574 mg/m ³ / 150 ppm	Not Available	Sk
Australia Exposure Standards	zinc phosphate	Fume (thermally generated) (respirable dust)	2 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	n-butanol	n-Butyl alcohol	Not Available	Not Available	152 mg/m ³ / 50 ppm	Sk
Australia Exposure Standards	phenol	Phenol	4 mg/m ³ / 1 ppm	Not Available	Not Available	Sk
Australia Exposure Standards	isopropyl benzene - cumene	Cumene	125 mg/m ³ / 25 ppm	375 mg/m ³ / 75 ppm	Not Available	Sk

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
methyl ethyl ketone	Butanone, 2-; (Methyl ethyl ketone; MEK)	Not Available	Not Available	Not Available
xylene	Xylenes	Not Available	Not Available	Not Available
toluene	Toluene	Not Available	Not Available	Not Available
zinc phosphate	Zinc phosphate (3:2)	1.8 mg/m ³	20 mg/m ³	120 mg/m ³
n-butanol	Butyl alcohol, n-; (n-Butanol)	20 ppm	50 ppm	8000 ppm
naphtha petroleum, light aromatic solvent	Aromatic hydrocarbon solvents; (High flash naphtha distillates; Solvent naphtha (petroleum), light aromatic)	3.1 ppm	34 ppm	410 ppm
phenol	Phenol	Not Available	Not Available	Not Available

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isopropyl benzene - cumene	Cumene; (Isopropyl benzene)	Not Available	Not Available	Not Available
1,3,5-trimethyl benzene	Mesitylene; (1,3,5-Trimethylbenzene)	Not Available	Not Available	360 ppm
1,2,4-trimethyl benzene	Trimethylbenzene, 1,2,4-; (Pseudocumene)	Not Available	Not Available	360 ppm

Ingredient	Original IDLH	Revised IDLH
ethanol	Not Available	Not Available
methyl ethyl ketone	3,000 ppm	3,000 [Unch] ppm
xylene	1,000 ppm	900 ppm
toluene	2,000 ppm	500 ppm
zinc phosphate	Not Available	Not Available
n-butanol	8,000 ppm	1,400 [LEL] ppm
naphtha petroleum, light aromatic solvent	Not Available	Not Available
phenol	250 ppm	250 [Unch] ppm
isopropyl benzene - cumene	8,000 ppm	900 [LEL] ppm
1,3,5-trimethyl benzene	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available

MATERIAL DATA

NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV.

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

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

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

Exposure controls

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>																	
	<table border="1" style="width: 100%;"> <tr> <td>Type of Contaminant:</td> <td>Air Speed:</td> </tr> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" style="width: 100%;"> <tr> <td>Lower end of the range</td> <td>Upper end of the range</td> </tr> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion
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Personal protection																		

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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>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 an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.</p>
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

generated selection:

Etch Primer

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
##isopropyl benzene -	cumene

Respiratory protection

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

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

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

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VITON	C
VITON/CHLOROBUTYL	C
VITON/NEOPRENE	C
##methyl ethyl	ketone

* 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	Highly flammable liquid.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	13 (ethanol)	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Not Available	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ 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>Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.</p> <p>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>
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Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p>												
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. 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> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</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>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.</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>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents.</p> <p>Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size.</p> <p>Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1).</p> <p>(1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996</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. Previous suggestions that chronic toluene inhalation produced human peripheral neuropathy have been discounted. However central nervous system (CNS) depression is well documented where blood toluene exceeds 2.2 mg%. Toluene abusers can achieve transient circulating concentrations of 6.5 %. Amongst workers exposed for a median time of 29 years, to toluene, no subacute effects on neuroathenic complaints and psychometric test results could be established. The prenatal toxicity of very high toluene concentrations has been documented for several animal species and man. Malformations indicative of specific teratogenicity have not generally been found. Neonatal toxicity, described in the literature, takes the form of embryo death or delayed foetal growth and delayed skeletal system development. Permanent damage of children has been seen only when mothers have suffered from chronic intoxication as a result of "sniffing".</p>												
Etch Primer	<table border="1"> <thead> <tr> <th>TOXICITY</th> </tr> </thead> <tbody> <tr> <td>Not Available</td> </tr> </tbody> </table>	TOXICITY	Not Available	<table border="1"> <thead> <tr> <th>IRRITATION</th> </tr> </thead> <tbody> <tr> <td>Not Available</td> </tr> </tbody> </table>	IRRITATION	Not Available							
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toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Not Available
	Inhalation (rat) LC50: >26700 ppm/1hd ^[2]	
	Inhalation (rat) LC50: 49 mg/L/4H ^[2]	
Oral (rat) LD50: 636 mg/kg ^[2]		
zinc phosphate	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg ^[1]	Not Available
n-butanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3434.4 mg/kg ^[1]	Not Available
	Inhalation (rat) LC50: 24 mg/L/4H ^[2]	
	Inhalation (rat) LC50: 8000 ppm/4hE ^[2]	
Oral (rat) LD50: 2292.3 mg/kg ^[1]		
naphtha petroleum, light aromatic solvent	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Nil reported
	Inhalation (rat) LC50: >3670 ppm/8 h ^[2]	
Oral (rat) LD50: >4500 mg/kg ^[1]		
phenol	TOXICITY	IRRITATION
	dermal (rat) LD50: 662.5 mg/kg ^[1]	Eye(rabbit): 100 mg rinse - mild
	Inhalation (rat) LC50: 0.316 mg/L/4H ^[2]	Eye(rabbit): 5 mg - SEVERE
	Oral (rat) LD50: 317 mg/kg ^[2]	Skin(rabbit): 500 mg open -SEVERE
		Skin(rabbit): 500 mg/24hr - SEVERE
isopropyl benzene - cumene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 10602.6 mg/kg ^[1]	Eye (rabbit): 500 mg/24h mild
	Inhalation (rat) LC50: 39 mg/L/4H ^[2]	Eye (rabbit): 86 mg mild
	Oral (rat) LD50: 1400 mg/kg ^[2]	Skin (rabbit): 10 mg/24h mild
		Skin (rabbit):100 mg/24h moderate
1,3,5-trimethyl benzene	TOXICITY	IRRITATION
	dermal (rat) LD50: >3460 mg/kg ^[1]	Eye (rabbit): 500 mg/24h mild
	Inhalation (rat) LC50: 24 mg/L/4hd ^[2]	Skin (rabbit): 20 mg/24h moderate
Oral (rat) LD50: ca.3460 mg/kg ^[1]		
1,2,4-trimethyl benzene	TOXICITY	IRRITATION
	dermal (rat) LD50: 3504 mg/kg ^[1]	Not Available
	Inhalation (rat) LC50: 18 mg/L/4hd ^[2]	
Oral (rat) LD50: ca.3504 mg/kg ^[1]		
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

ETHANOL

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

METHYL ETHYL KETONE

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. 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.

Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities. Combinations with chloroform also show increase in toxicity

<p>XYLENE</p>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>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. Reproductive effector in rats</p>
<p>TOLUENE</p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>For toluene:</p> <p>Acute Toxicity</p> <p>Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.</p> <p>Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.</p> <p>Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.</p> <p>Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.</p> <p>Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death</p> <p>Toluene can also strip the skin of lipids causing dermatitis</p> <p>Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days</p> <p>Subchronic/Chronic Effects:</p> <p>Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.</p> <p>Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin.</p> <p>Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L</p> <p>Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .</p> <p>Developmental/Reproductive Toxicity</p> <p>Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.</p> <p>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</p> <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.</p> <p>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>
<p>N-BUTANOL</p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>for n-butanol</p> <p>Acute toxicity: n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC0 of 8000 ppm (24000 mg/m³) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA's localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser.</p>

Etch Primer

The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed.

Repeat dose toxicity: An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAC) to BA. Hydrolysis of BAC in blood and brain was estimated to be 99 percent complete within

2.7 minutes (elimination $t_{1/2} = 0.41$ minute). Thus, organisms exposed to BAC can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAC can be used as supplemental, surrogate data to provide information on the toxicity of BA.

A thirteen-week, subchronic exposure to BAC, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m³) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOAEL) of 500 ppm (2395 mg/m³) was reported for systemic effects in rats, and a NOAEL of 3000 ppm (14370 mg/m³) was reported for post exposure neurotoxicity in rats.

Reproductive toxicity: Several studies indicate that BA is not a reproductive toxicant.

Female rats exposed to 6000 ppm (18000 mg/m³) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m³) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity.

Developmental toxicity: BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m³) throughout gestation.

Genotoxicity: An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic.

Carcinogenicity: Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.

For trimethylbenzenes:

Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption.

1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid. The major routes of excretion of 1,2,4-trimethylbenzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.

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

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

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

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

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

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

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

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

For C9 aromatics (typically trimethylbenzenes - TMBs)

Acute Toxicity

Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50's range from 6,000 to 10,000 mg/m³ for C9 aromatic naphtha and 18,000 to 24,000 mg/m³ for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.

Irritation and Sensitization

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

Repeated Dose Toxicity

Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m³). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neurobehavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m³, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m³) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.

The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m³. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4- and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m³).

Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m³ (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers,

**NAPHTHA PETROLEUM,
LIGHT AROMATIC
SOLVENT**

the NOELs are 25 ppm or 123 mg/m³ for respiratory irritation and 250 ppm or 1230 mg/m³ for systemic effects.

Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg-bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with *Salmonella typhimurium* and *Escherichia coli* bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m³) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category

Reproductive and Developmental Toxicity

Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m³, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex/group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex/group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.

Systemic Effects on Parental Generations:

The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m³).

Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m³). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m³), which excludes analysis of the highest concentration due to excessive mortality.

Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m³) based on the body weights reductions observed in the F3 offspring.

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

Inhalation (rat) TCLo: 1320 ppm/6h/90D-1 * [Devoe]

PHENOL

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

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.

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.

ISOPROPYL BENZENE - CUMENE

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. 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 aromatic terpenes:

Acute toxicity: Mammalian LD50 for p-cymene have shown it to have low toxic potential. Similar studies with cumene have concurred with these results. In general, the studies indicate that p-cymene (p-methylisopropylbenzene) or cumene (isopropylbenzene) is rapidly absorbed by oral or inhalation routes. They undergo oxidation (hydroxylation) of the side chain isopropyl substituent and, in the case of p-cymene, the methyl substituent to yield polar oxygenated metabolites. These metabolites are either excreted unchanged in the urine or undergo Phase II conjugation with glucuronic acid and/or glycine followed by excretion in the urine. Unchanged p-cymene or cumene were not detected in the urine or faeces.

Humans (5 males and 5 females/group) exposed to an atmosphere containing 49, 98, or 147 ppm cumene for 7 hours showed 64% absorption at 0.5 hours and 45% at 7 hours. Maximum excretion is observed at 6 to 8 hours and is essentially complete at 48 hours. Approximately 35% of the dose inhaled was excreted as 2-phenyl-2-propanol

Repeat Dose Toxicity: Subacute Studies: Groups of 7 to 12 male rats were exposed to 0, 50, or 250 ppm of p-cymene for 6 hours/day, 5 days/week for 4 weeks with an 8-week recovery period. There was no overt toxicity in the treated rats and no effect on body weight or terminal weight of the brain, cerebellum or whole brain. There was also no effect on regional enzyme activities, regional protein synthesis or regional neurotransmitter concentrations.

Cumene has been tested by the National Toxicology Program (NTP) in both rats and mice. Animals were exposed to up to 4,000 ppm cumene by whole-body inhalation for 12-13 days over a period of 16-17 days. In rats, all animals died at 4,000 ppm, and about half the animals died at the next exposure concentration (2,000 ppm). Varying degrees of ataxia were reported in surviving rats exposed to 500 to 2,000 ppm cumene. Increased relative liver and kidney weights were reported in rats exposed to cumene. In exposed male rats, hyaline droplets in the renal cortical tubules were reported. At 2,000 ppm, superlative inflammation of the lung was reported in 40% of the rats. In mice, all animals died at the 2 highest exposures (2,000 and 4,000 ppm). At 1,000 ppm, 80% of the female mice died and male mice showed varying degrees of ataxia. Increased relative liver and kidney weights were reported in mice exposed to cumene. Decreased thymus weight was reported in male mice exposed to 1,000 ppm of cumene. No histopathological findings accompanied the organ weight changes. A NOAEL of 1,000 ppm was determined for female rats and male mice and a NOAEL of 500 ppm was determined for female mice based on mortality and histopathological findings.

Chronic toxicity: The US EPA concluded that there is some evidence that suggests that cumene is not likely to produce a carcinogenic response (i.e., numerous genotoxic tests, including gene mutation, chromosomal aberration, and primary DNA damage tests, all but one of which were negative or not reproducible). In addition, EPA noted that cumene does not appear to metabolise to highly reactive chemical species and in terms of metabolism, cumene is analogous to methyl benzene for which a 2-year inhalation study was conducted by NTP and no evidence of carcinogenic activity was reported in either rats or mice.

Given that the only structural difference between p-cymene and cumene is the presence of a second alkyl substituent (isopropylbenzene versus p-methylisopropylbenzene), similar conclusions can be drawn for p-cymene, particularly since the pharmacokinetic, metabolic and toxicologic data that are available support this conclusion.

Reproductive toxicity: Taking into consideration the rapid metabolism and excretion of cumene, the US EPA concluded, "cumene has low potential for reproductive toxicity."

Developmental toxicity: Even at maternally toxic concentrations exposure to cumene vapor did not produce developmental toxicity in rats. However the US EPA determined that the changes in gestational parameters of the rabbits, though not significant, were consistent in indicating possible developmental effects and therefore set the NOAEL in rabbits for both developmental and maternal effects at 1,206 ppm and the LOAEL at 2,297 ppm, respectively (as reported in EPA, 1997). Since both cumene and p-cymene exhibit such similar pharmacokinetic and metabolic profiles, and show no evidence of toxicity at levels of exposure similar to those experienced by humans, further teratogenic or developmental testing is not recommended

Genotoxicity: The genotoxicity database on p-cymene and cumene shows no mutagenic potential in the Ames assay. In cytogenetic assays, there is no evidence of a genotoxic potential in vitro. In whole animals, the genotoxicity results for cumene are mixed showing weakly positive results in micronuclei induction in rats, but no evidence of genotoxicity in mice.

Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen
[National Toxicology Program: U.S. Dep. of Health & Human Services 2002]

Cumene is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals. Cumene caused tumours at several tissue sites, including lung and liver in mice and kidney in male rats. Several proposed mechanisms of carcinogenesis support the relevance to humans of lung and liver tumours in experimental animals. Specifically, there is evidence that humans and experimental animals metabolise cumene through similar metabolic pathways. There is also evidence that cumene is genotoxic in some tissues, based on findings of DNA damage in rodent lung and liver. Furthermore, mutations of the K-ras oncogene and p53 tumor-suppressor gene observed in cumene-induced lung tumours in mice, along with altered expression of many other genes, resemble molecular alterations found in human lung and other cancers. The relevance of the kidney tumors to cancer in humans is uncertain; there is evidence that a species-specific mechanism not relevant to humans contributes to their induction, but it is possible that other mechanisms relevant to humans, such as genotoxicity, may also contribute to kidney-tumour formation in male rats.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

For trimethylbenzenes:

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

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

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

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

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

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

1,3,5-TRIMETHYL BENZENE

decreased weight gain, lymphopenia and neutrophilia .

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

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

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

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

CHEMWATCH 12171 1,2,4-trimethylbenzene

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

CHEMWATCH 2325 1,3,5-trimethylbenzene

1,2,4-TRIMETHYL BENZENE

1,3,5-TRIMETHYL BENZENE & 1,2,4-TRIMETHYL BENZENE

Other Toxicity data is available for

1,3,5-TRIMETHYL BENZENE & 1,2,4-TRIMETHYL BENZENE

CHEMWATCH 12172 1,2,3-trimethylbenzene

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
methyl ethyl ketone	LC50	96	Fish	228.130mg/L	3
methyl ethyl ketone	EC50	48	Crustacea	308mg/L	2
methyl ethyl ketone	EC50	96	Algae or other aquatic plants	>500mg/L	4
methyl ethyl ketone	EC50	384	Crustacea	52.575mg/L	3
methyl ethyl ketone	NOEC	48	Crustacea	68mg/L	2
xylene	LC50	96	Fish	0.0013404mg/L	4
xylene	EC50	48	Crustacea	>3.4mg/L	2
xylene	EC50	72	Algae or other aquatic plants	4.6mg/L	2
xylene	EC50	24	Crustacea	0.711mg/L	4
xylene	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
toluene	LC50	96	Fish	0.0031704mg/L	4
toluene	EC50	48	Crustacea	0.01151750mg/L	4
toluene	EC50	72	Algae or other aquatic plants	12.5mg/L	4
toluene	BCF	24	Algae or other aquatic plants	10mg/L	4
toluene	EC50	3	Algae or other aquatic plants	0.1336030mg/L	4
toluene	NOEC	168	Crustacea	0.74mg/L	2
zinc phosphate	LC50	96	Fish	0.09mg/L	4
zinc phosphate	EC50	48	Crustacea	0.105mg/L	2
zinc phosphate	EC50	48	Crustacea	0.155mg/L	2
zinc phosphate	NOEC	72	Algae or other aquatic plants	0.000501904mg/L	2
n-butanol	LC50	96	Fish	88.462mg/L	3
n-butanol	EC50	48	Crustacea	>500mg/L	1
n-butanol	EC50	96	Algae or other aquatic plants	225mg/L	2
n-butanol	BCF	24	Fish	921mg/L	4
n-butanol	EC50	504	Crustacea	18mg/L	2
n-butanol	NOEC	504	Crustacea	4.1mg/L	2
naphtha petroleum, light aromatic solvent	EC50	48	Crustacea	=6.14mg/L	1
naphtha petroleum, light aromatic solvent	EC50	72	Algae or other aquatic plants	3.29mg/L	1
naphtha petroleum, light aromatic solvent	EC10	72	Algae or other aquatic plants	1.13mg/L	1
naphtha petroleum, light aromatic solvent	NOEC	72	Algae or other aquatic plants	=1mg/L	1
phenol	LC50	96	Fish	0.00175mg/L	4
phenol	EC50	48	Crustacea	=3.1mg/L	1
phenol	EC50	96	Algae or other aquatic plants	0.0611mg/L	4
phenol	BCF	24	Fish	60mg/L	4
phenol	EC50	24	Crustacea	0.000395mg/L	4
phenol	NOEC	144	Crustacea	0.01mg/L	4
isopropyl benzene - cumene	LC50	96	Fish	1.784mg/L	3
isopropyl benzene - cumene	EC50	48	Crustacea	0.00060095mg/L	4
isopropyl benzene - cumene	EC50	72	Algae or other aquatic plants	1.29mg/L	2
isopropyl benzene - cumene	EC50	3	Algae or other aquatic plants	0.00877387mg/L	4
isopropyl benzene - cumene	NOEC	72	Algae or other aquatic plants	0.22mg/L	2
1,3,5-trimethyl benzene	LC50	96	Fish	1.318mg/L	3
1,3,5-trimethyl benzene	EC50	48	Crustacea	0.0060095mg/L	4
1,3,5-trimethyl benzene	EC50	96	Algae or other aquatic plants	2.154mg/L	3
1,3,5-trimethyl benzene	EC50	384	Crustacea	0.328mg/L	3
1,3,5-trimethyl benzene	NOEC	504	Crustacea	0.4mg/L	2

Etch Primer

1,2,4-trimethyl benzene	LC50	96	Fish	1.318mg/L	3
1,2,4-trimethyl benzene	EC50	48	Crustacea	0.0036057mg/L	4
1,2,4-trimethyl benzene	EC50	96	Algae or other aquatic plants	2.154mg/L	3
1,2,4-trimethyl benzene	EC50	384	Crustacea	0.328mg/L	3

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

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

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

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
phenol	LOW (Half-life = 10 days)	LOW (Half-life = 0.95 days)
isopropyl benzene - cumene	HIGH	HIGH
1,3,5-trimethyl benzene	HIGH	HIGH
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
methyl ethyl ketone	LOW (LogKOW = 0.29)
xylene	MEDIUM (BCF = 740)
toluene	LOW (BCF = 90)
n-butanol	LOW (BCF = 64)
phenol	LOW (BCF = 17.5)
isopropyl benzene - cumene	LOW (BCF = 35.5)
1,3,5-trimethyl benzene	LOW (BCF = 342)
1,2,4-trimethyl benzene	LOW (BCF = 275)

Mobility in soil

Ingredient	Mobility
methyl ethyl ketone	MEDIUM (KOC = 3.827)
toluene	LOW (KOC = 268)
n-butanol	MEDIUM (KOC = 2.443)
phenol	LOW (KOC = 268)
isopropyl benzene - cumene	LOW (KOC = 817.2)
1,3,5-trimethyl benzene	LOW (KOC = 703)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)

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. ▶ 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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SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	
HAZCHEM	•3YE

Land transport (ADG)

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) (contains ethanol and methyl ethyl ketone)
Environmental hazard	No relevant data
Transport hazard class(es)	Class : 3 Subrisk : Not Applicable
Special precautions for user	Special provisions : 163 * 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) (contains ethanol and methyl ethyl ketone)
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class : 3 ICAO / IATA Subrisk : Not Applicable ERG Code : 3L
Special precautions for user	Special provisions : A3 A72 A192 Cargo Only Packing Instructions : 364 Cargo Only Maximum Qty / Pack : 60 L Passenger and Cargo Packing Instructions : 353 Passenger and Cargo Maximum Qty / Pack : 5 L Passenger and Cargo Limited Quantity Packing Instructions : Y341 Passenger and Cargo Limited Maximum Qty / Pack : 1 L

Sea transport (IMDG-Code / GGVSee)

UN number	1263
Packing group	II
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac solutions, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound) (contains ethanol and methyl ethyl ketone)
Environmental hazard	Marine Pollutant
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 73 / 78 and the IBC code

Source	Ingredient	Pollution Category
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Etch Primer

IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	methyl ethyl ketone	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	xylene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	toluene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	naphtha petroleum, light aromatic solvent	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	phenol	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	isopropyl benzene - cumene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	1,3,5-trimethyl benzene	Y; X
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	1,2,4-trimethyl benzene	Y; X

SECTION 15 REGULATORY INFORMATION

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

ETHANOL(64-17-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

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

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

ZINC PHOSPHATE(7779-90-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

N-BUTANOL(71-36-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT(64742-95-6.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

PHENOL(108-95-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

ISOPROPYL BENZENE - CUMENE(98-82-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

1,3,5-TRIMETHYL BENZENE(108-67-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

1,2,4-TRIMETHYL BENZENE(95-63-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	N (ethanol)
Canada - DSL	N (ethanol)

Etch Primer

Canada - NDSL	N (toluene; zinc phosphate; n-butanol; phenol; xylene; naphtha petroleum, light aromatic solvent; ethanol; 1,3,5-trimethyl benzene; isopropyl benzene - cumene; 1,2,4-trimethyl benzene; methyl ethyl ketone)
China - IECSC	N (ethanol)
Europe - EINEC / ELINCS / NLP	N (ethanol)
Japan - ENCS	N (ethanol)
Korea - KECI	N (ethanol)
New Zealand - NZIoC	N (ethanol)
Philippines - PICCS	N (ethanol)
USA - TSCA	N (ethanol)
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
zinc phosphate	7543-51-3, 7779-90-0
naphtha petroleum, light aromatic solvent	25550-14-5., 64742-95-6.

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

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

www.chemwatch.net

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

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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