Duram Metal Etch Primer

Duram Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **4866-06**
Version No: **5.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **10/03/2023**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Duram Metal Etch Primer
Chemical Name	Not Applicable
Synonyms	Not Available
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)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Industrial paint. Application is usually by spray atomisation

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Duram Pty Ltd**
Address	51 Prince William Drive Seven Hills NSW 2147 Australia
Telephone	+61 2 9624 4007
Fax	+61 2 9624 4079
Website	www.duram.com.au
Email	mail@duram.com.au

Emergency telephone number

Association / Organisation	**CHEMTREC Australia (Sydney)**
Emergency telephone numbers	+612 9037 2994 24 hours / 7 days
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

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

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

Poisons Schedule	S5
Classification [1]	Flammable Liquids Category 2, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Carcinogenicity Category 1B, Hazardous to the Aquatic Environment Acute Hazard Category 3
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H225	Highly flammable liquid and vapour.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H350	May cause cancer.
H402	Harmful to aquatic life.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

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

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1330-20-7	10-30	xylene
123-86-4	10-30	n-butyl acetate
64742-95-6.	1-10	C9-aromatic hydrocarbon solvent
78-83-1	1-10	isobutanol
108-65-6	1-10	propylene glycol monomethyl ether acetate, alpha-isomer
8008-20-6	1-10	kerosene

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CAS No	%[weight]	Name
71-36-3	1-10	n-butanol
Not Available	balance	other ingredients not contributing to the classification
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ 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	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ 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.
Treat symptomatically.
For acute or short term repeated exposures to xylene:

- Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- Pulmonary absorption is rapid with about 60-65% retained at rest.
- Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases ($pO_2 < 50$ mm Hg or $pCO_2 > 50$ mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

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

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

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

Advice for firefighters

Fire Fighting	▸ 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).

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	▸ 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	▸ 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). Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material.
HAZCHEM	•3YE

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ 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	▸ 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	▸ 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. · Wait 2 minutes after tank filling (for tanks such as those on · road tanker vehicles) before opening hatches or manholes. · Wait 30 minutes after tank filling (for large storage tanks) · before opening hatches or manholes. Even with proper · grounding and bonding, this material can still accumulate an · electrostatic charge. If sufficient charge is allowed to · accumulate, electrostatic discharge and ignition of flammable · air-vapour mixtures can occur. Be aware of handling · operations that may give rise to additional hazards that result · from the accumulation of static charges. These include but are · not limited to pumping (especially turbulent flow), mixing, · filtering, splash filling, cleaning and filling of tanks and · containers, sampling, switch loading, gauging, vacuum truck · operations, and mechanical movements. These activities may · lead to static discharge e.g. spark formation. Restrict line · velocity during pumping in order to avoid generation of · electrostatic discharge (= 1 m/s until fill pipe submerged to

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	· twice its diameter, then = 7 m/s). 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	▸ Store in original containers in approved flame-proof area. ▸ No smoking, naked lights, heat or ignition sources. ▸ **DO NOT** store in pits, depression, basement or areas where vapours may be trapped. ▸ Keep containers securely sealed. ▸ Store away from incompatible materials in a cool, dry well ventilated area. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this MSDS. ▸ Tank storage: Tanks must be specifically designed for use with this product. Bulk storage tanks should be diked (bunded). Locate tanks away from heat and other sources of ignition. Cleaning, inspection and maintenance of storage tanks is a specialist operation, which requires the implementation of strict procedures and precautions. ▸ Keep in a cool place. Electrostatic charges will be generated during pumping. Electrostatic discharge may cause fire. Ensure electrical continuity by bonding and grounding (earthing) all equipment to reduce the risk. The vapours in the head space of the storage vessel may lie in the flammable/explosive range and hence may be flammable. ▸ For containers, or container linings use mild steel, stainless steel. Examples of suitable materials are: high density polyethylene (HDPE), polypropylene (PP), and Viton (FMK), which have been specifically tested for compatibility with this product. ▸ For container linings, use amine-adduct cured epoxy paint. ▸ For seals and gaskets use: graphite, PTFE, Viton A, Viton B. ▸ Unsuitable material: Some synthetic materials may be unsuitable for containers or container linings depending on the material specification and intended use. Examples of materials to avoid are: natural rubber (NR), nitrile rubber (NBR), ethylene propylene rubber (EPDM), polymethyl methacrylate (PMMA), polystyrene, polyvinyl chloride (PVC), polyisobutylene. However, some may be suitable for glove materials. ▸ Do not cut, drill, grind, weld or perform similar operations on or near containers. Containers, even those that have been emptied, can contain explosive vapours.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ 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	▸ Avoid strong acids, bases. ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	n-butyl acetate	n-Butyl acetate	150 ppm / 713 mg/m3	950 mg/m3 / 200 ppm	Not Available	Not Available
Australia Exposure Standards	isobutanol	Isobutyl alcohol	50 ppm / 152 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Not Available
Australia Exposure Standards	kerosene	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	n-butanol	n-Butyl alcohol	Not Available	Not Available	50 ppm / 152 mg/m3	Not Available

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Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
xylene	Not Available	Not Available	Not Available
n-butyl acetate	Not Available	Not Available	Not Available
C9-aromatic hydrocarbon solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
isobutanol	150 ppm	1,300 ppm	8000* ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available	Not Available
kerosene	Not Available	Not Available	4,800 mg/m3
n-butanol	60 ppm	800 ppm	8000** ppm

Ingredient	Original IDLH	Revised IDLH
xylene	900 ppm	Not Available
n-butyl acetate	1,700 ppm	Not Available
C9-aromatic hydrocarbon solvent	Not Available	Not Available
isobutanol	1,600 ppm	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
kerosene	2,500 mg/m3	Not Available
n-butanol	1,400 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
C9-aromatic hydrocarbon solvent	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

For trimethyl benzene as mixed isomers (of unstated proportions)
Odour Threshold Value: 2.4 ppm (detection)
Use care in interpreting effects as a single isomer or other isomer mix. Trimethylbenzene is an eye, nose and respiratory irritant. High concentrations cause central nervous system depression. Exposed workers show CNS changes, asthmatic bronchitis and blood dyscrasias at 60 ppm. The TLV-TWA is thought to be protective against the significant risk of CNS excitation, asthmatic bronchitis and blood dyscrasias associated with exposures above the limit.
Odour Safety Factor (OSF)
OSF=10 (1,2,4-TRIMETHYLBENZENE)

For n-butyl acetate
Odour Threshold Value: 0.0063 ppm (detection), 0.038-12 ppm (recognition)
Exposure at or below the recommended TLV-TWA is thought to prevent significant irritation of the eyes and respiratory passages as well as narcotic effects. In light of the lack of substantive evidence regarding teratogenicity and a review of acute oral data a STEL is considered inappropriate.
Odour Safety Factor(OSF)
OSF=3.8E2 (n-BUTYL ACETATE)

for propylene glycol monomethyl ether acetate (PGMEA)
Saturated vapour concentration: 4868 ppm at 20 C.
A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternabral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

for kerosene CAS 8008-20-6
TLV TWA: 100 mg/m3 as total hydrocarbon vapour Skin A3
OEL TWA: 14 ppm, 100 mg/m3 [NIOSH, 1985]
REL TWA: 150 ppm [Shell]
CEL TWA: 300 ppm, 900 mg/m3
(CEL = Chemwatch Exposure Limit)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

for petroleum distillates:

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CEL TWA: 500 ppm, 2000 mg/m3 (compare OSHA TWA)
(CEL = Chemwatch Exposure Limit)
for xylenes:
IDLH Level: 900 ppm
Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)
NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).
Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.
Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially contribute to the dose received by inhalation.
Odour Safety Factor(OSF)
OSF=4 (XYLENE)

For n-butanol:
Odour Threshold Value: 0.12-3.4 ppm (detection), 1.0-3.5 ppm (recognition)
NOTE: Detector tubes for n-butanol, measuring in excess of 5 ppm are commercially available.
Exposure at or below the TLV-TWA is thought to provide protection against hearing loss due to vestibular and auditory nerve damage in younger workers and to protect against the significant risk of headache and irritation.
25 ppm may produce mild irritation of the respiratory tract 50 ppm may produce headache and vertigo.
Higher concentrations may produce marked irritation, sore throat, coughing, nausea, shortness of breath, pulmonary injury and central nervous system depression characterised by headache, dizziness, dullness and drowsiness.
6000 ppm may produce giddiness, prostration, narcosis, ataxia, and death.
Odour Safety Factor (OSF)
OSF=60 (n-BUTANOL)

For isobutanol:
Odour Threshold Value: 0.66-40 ppm (detection), 1.8-53 ppm (recognition) Although there do not appear to be reports of isobutyl alcohol causing auditory impairment or vestibular damage in humans (as with n-butanol) the recommended TLV-TWA recognises the slightly greater acute toxic potential of isobutanol versus n-butanol. Exposure at or below this limit is thought to significantly reduce the risk of skin irritation.
Odour Safety Factor (OSF)
OSF=31 (ISOBUTYL ALCOHOL)

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

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.
For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.
Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
· Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance.
· Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures.
· Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered.. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the

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	space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)
Individual protection measures, such as personal protective equipment	
Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber For esters: ▸ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. 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. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential 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.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower. ▸ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▸ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▸ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

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:
Duram Metal Etch Primer

Material	CPI
BUTYL	C

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

Continued...

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

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

up to 10 x ES	A-AUS	-	A-PAPR-AUS / Class 1
up to 50 x ES	-	A-AUS / Class 1	-
up to 100 x ES	-	A-2	A-PAPR-2 ^

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured highly flammable liquid with solvent odour; partially mixes with water. Volatile Component: 42.1 %wt

Physical state	Liquid	**Relative density (Water = 1)**	1.23
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	118	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	20	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	8	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Partly miscible	**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	- 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

Continued...

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur. Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body. Human subjects exposed to 24 ppm n-butanol experienced mild irritation which became objectionable. Headaches were reported at 50 ppm. Exposure by mice to 6600 ppm produced signs of marked central nervous system (CNS) depression, including prostration after 2 hours, narcosis after 3 hours and some deaths. Although n-butanol is odourous and generally possesses adequate warning properties, the olfactory senses may become fatigued. Inhalation hazard is increased at higher temperatures. A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias. High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement. Isobutanol appears to be more toxic than n-butyl alcohol. A 4-hour inhalation exposure of rats at 8000 ppm resulted in deaths. Mice exposed at 2125 ppm isobutyl alcohol for 223 hours in a series of intermittent exposures, each lasting 9.25 hours did not show signs of toxic injury. In a second study mice were narcotised repeatedly following a series of intermittent exposures that totalled 136 hours at a concentration of 6400 ppm - no mortalities were recorded. 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 Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue. Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Following a single dose of isobutanol in rats, deaths were delayed for several days and hepatic degeneration was evident. Swallowing of n-butanol may cause breathing difficulty, headache, nausea, vomiting, upper respiratory tract irritation, mucous membrane irritation, central nervous system depression. Considered an unlikely route of entry in commercial/industrial environments The liquid may produce considerable gastrointestinal discomfort and may be harmful or toxic if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ 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. 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. Application of isobutanol to human skin produced slight erythema and hyperaemia. Open cuts, abraded or irritated skin should not be exposed to this material 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.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Instillation of isobutanol into a rabbit's eye caused moderate to severe irritation but no permanent injury to the cornea. No evidence of irritation was found when human volunteers were exposed to repeated 8 hour exposures to 100 ppm vapour. Workers exposed to 200 ppm n-butanol showed ocular symptoms including corneal inflammation, burning sensation, blurring of vision, lachrymation, and photophobia. 100 ppm produced no systemic effects and reports of irritation of the eyes was rare. The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Three out of 19 rats dosed orally with 0.2 ml isobutanol developed either forestomach carcinomas, lever cell carcinoma or myelogenous leukaemia and benign tumours were more prevalent than those found in a control group of animals Serious systemic effects from exposure to n-butanol in the form of auditory and vestibular nerve damage have been reported amongst workers in

Continued...

France and Mexico. Audiologic impairment was produced in workers exposed to 80 ppm n-butanol with unprotected noise exposure. Workers exposed over a 15 year period (1929-1944) exhibited severe vertigo and vertiges gravis. Workers exposed from 3-11 years without personal protective equipment from noise experienced greater hearing loss (hypoacusia) in direct relation to exposure time when compared to a control group exposed to industrial noise of 90-100 dB but with n-butanol exposure. Average hearing loss was not large but the workers had central frequencies of 21.98 dB (11.59 dB minimum and 32.30 dB maximum) with a mean widening of the break between 3000 and 4000 Hz of 42.22 dB. There was a tendency of the averages to decrease as the frequencies moved away from the central zone. Affected workers were aged from 20-39 years. [ACGIH Documentation of TLVs]
Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mix ed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.
Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers. Xylene has been classed as a developmental toxin in some jurisdictions.
Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.
On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.
Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).
Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

Duram Metal Etch Primer	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

xylene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1700 mg/kg[2]	Eye (human): 200 ppm irritant
	Inhalation(Rat) LC50: 5000 ppm4h[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (Mouse) LD50; 2119 mg/kg[2]	Eye (rabbit): 87 mg mild
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):500 mg/24h moderate
		Skin: adverse effect observed (irritating)[1]

n-butyl acetate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3200 mg/kg[2]	Eye (human): 300 mg * [PPG]
	Inhalation(Rat) LC50: 0.74 mg/l4h[2]	Eye (rabbit): 20 mg (open)-SEVERE
	Oral (Rabbit) LD50; 3200 mg/kg[2]	Eye (rabbit): 20 mg/24h - moderate
		Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating)[1]

C9-aromatic hydrocarbon solvent	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: >4.42 mg/L4h[1]	Skin: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: >4500 mg/kg[1]	

isobutanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (rabbit): 2 20 mg/24h-moderate
	Inhalation(Rabbit) LC50; 2.63 mg/L4h[2]	Eye (rabbit): 2 mg/24h - SEVERE
	Oral (Rat) LD50: 2460 mg/kg[2]	Skin (rabbit): mg (open)-SEVERE

propylene glycol monomethyl ether acetate, alpha-isomer	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 3739 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

kerosene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: >4.3 mg/l4h[1]	Skin (rabbit): 500 mg SEVERE
	Oral (Rat) LD50: >5000 mg/kg[2]	Skin: adverse effect observed (irritating)[1]

n-butanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3400 mg/kg[2]	Eye (human): 50 ppm - irritant
	Inhalation(Rat) LC50: 8000 ppm4h[2]	Eye (rabbit): 1.6 mg-SEVERE

Continued...

	Oral (Rat) LD50: 790 mg/kg[2]	Eye (rabbit): 24 mg/24h-SEVERE
		Eye: adverse effect observed (irreversible damage)[1]
		Skin (rabbit): 405 mg/24h-moderate
		Skin: adverse effect observed (irritating)[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*	

XYLENE	Reproductive effector in rats The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
C9-AROMATIC HYDROCARBON SOLVENT	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-trimethyl- benzene 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 trimethyl- benzenes 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 tymphimurium/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 For C9 aromatics (typically trimethylbenzenes - TMBs) Acute Toxicity Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50 s range from 6,000 to 10,000 mg/m 3 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 neuro/behavioral 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, the NOELs are 25 ppm or 123 mg/m3 for respiratory irritation and 250 ppm or 1230 mg/m3 for systemic

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	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 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. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Not available. Refer to individual constituents.
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I] *Shin-Etsu SDS for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure.

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	Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.
KEROSENE	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable. 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. For "kerosenes" **Acute toxicity:** Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg The dermal LD50s of the same three kerosenes were all >2.0 g//kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No. 8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same material produced an LC50 of >6.4 mg/l When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced “moderate” to “severe” irritation. Six additional skin irritation studies on a range of kerosenes produced “mild” to “severe” irritation. An eye irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed and washed eyes) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurized kerosene and jet fuel. These materials produced more irritation in the unwashed eyes at 1 hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but by day 7 had resolved. Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurized kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in guinea pigs **Repeat-Dose toxicity:** Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24 respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from “slight” to “moderate” in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss. Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related:

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	• increased relative heart weights for the mid- and high- dose males and females, • increased absolute and relative spleen weights in treated females, and • differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stress-related and therefore, indirectly related to treatment). Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group. These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes. In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Opthalomological examination of all animals also found no treatment-related effects. There were no treatment-related effects on growth rates, hematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin. A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study . In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m3 kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues. **Carcinogenicity:** In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. .Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation . If the equivalent dose of kerosene was applied to the skin in manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred . Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration . The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice . Animal survivals were not effected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity. ***In-Vitro* (Genotoxicity):** The potential *in vitro* genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation . Modified Ames assays on four kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation . The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results . Hydrodesulfurized kerosene tested in a sister chromatid exchange assay produced negative results (with/without activation) ***In-Vivo* Genotoxicity:** Multiple *in vivo* genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in *in vivo* bone marrow cytogenetic tests in Sprague-Dawley rats . One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay . Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation. **Reproductive/Developmental Toxicity** Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days premating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day. Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported . There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds.
N-BUTANOL	for n-butanol **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. 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 t1/2 = 0.41 minute). Thus, organisms exposed to BAc can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAc can be used as supplemental, surrogate data to provide information on the toxicity of BA. 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.
XYLENE & N-BUTYL ACETATE & ISOBUTANOL & N-BUTANOL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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XYLENE & N-BUTYL ACETATE & C9-AROMATIC HYDROCARBON SOLVENT & ISOBUTANOL & N-BUTANOL	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.
N-BUTYL ACETATE & PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	Generally,linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic. The JEFCA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods **Internationl Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)** **Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998**
C9-AROMATIC HYDROCARBON SOLVENT & ISOBUTANOL & N-BUTANOL	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

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

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

SECTION 12 Ecological information

Toxicity

Duram Metal Etch Primer

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

xylene

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	2.6mg/l	2
EC50	72h	Algae or other aquatic plants	4.6mg/l	2
EC50	48h	Crustacea	1.8mg/l	2
NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2

n-butyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	17-19mg/l	4
EC50	72h	Algae or other aquatic plants	246mg/l	2
EC50	48h	Crustacea	32mg/l	1
EC50(ECx)	96h	Fish	18mg/l	2

C9-aromatic hydrocarbon solvent

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
EC50	72h	Algae or other aquatic plants	19mg/l	1
EC50	96h	Algae or other aquatic plants	64mg/l	2
EC50	48h	Crustacea	6.14mg/l	1

isobutanol

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	4mg/L	5
EC50	72h	Algae or other aquatic plants	593mg/l	2
LC50	96h	Fish	901-1000mg/l	4
EC50	48h	Crustacea	ca.600mg/l	1

propylene glycol monomethyl ether acetate, alpha-isomer

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	100mg/l	1

Continued...

	EC50	72h	Algae or other aquatic plants	>1000mg/l	2
	EC50	48h	Crustacea	373mg/l	2
	NOEC(ECx)	336h	Fish	47.5mg/l	2
	EC50	96h	Algae or other aquatic plants	>1000mg/l	2

kerosene	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

n-butanol	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	4.1mg/l	2
	EC50	96h	Algae or other aquatic plants	225mg/l	2
	EC50	72h	Algae or other aquatic plants	>500mg/l	1
	LC50	96h	Fish	100-500mg/l	4
	EC50	48h	Crustacea	>500mg/l	1

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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*

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
n-butyl acetate	LOW	LOW
isobutanol	LOW (Half-life = 14.42 days)	LOW (Half-life = 4.15 days)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
xylene	MEDIUM (BCF = 740)
n-butyl acetate	LOW (BCF = 14)
isobutanol	LOW (LogKOW = 0.76)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
n-butanol	LOW (BCF = 0.64)

Mobility in soil

Ingredient	Mobility
n-butyl acetate	LOW (KOC = 20.86)
isobutanol	MEDIUM (KOC = 2.048)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
n-butanol	MEDIUM (KOC = 2.443)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.

Otherwise:

- 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.

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.
A Hierarchy of Controls seems to be common - the user should investigate:

- Reduction
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been

Continued...

	contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **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 licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	•3YE

Land transport (ADG)

UN number or ID number	1263
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)
Transport hazard class(es)	Class: 3 Subsidiary risk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 163 367 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

UN number	1263
UN proper shipping name	Paint related material (including paint thinning or reducing compounds); Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base)
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Packing group	II
Environmental hazard	Not Applicable
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
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)
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable

Continued...

Special precautions for user	EMS Number	F-E, S-E
	Special provisions	163 367
	Limited Quantities	5 L

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

Not Applicable

Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
xylene	Not Available
n-butyl acetate	Not Available
C9-aromatic hydrocarbon solvent	Not Available
isobutanol	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
kerosene	Not Available
n-butanol	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
xylene	Not Available
n-butyl acetate	Not Available
C9-aromatic hydrocarbon solvent	Not Available
isobutanol	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
kerosene	Not Available
n-butanol	Not Available

SECTION 15 Regulatory information

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

xylene is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

n-butyl acetate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C9-aromatic hydrocarbon solvent is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

isobutanol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

propylene glycol monomethyl ether acetate, alpha-isomer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

kerosene is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

n-butanol is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes

Continued...

National Inventory	Status
Canada - NDSL	No (xylene; n-butyl acetate; C9-aromatic hydrocarbon solvent; isobutanol; propylene glycol monomethyl ether acetate, alpha-isomer; kerosene; n-butanol)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	05/09/2013

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	10/12/2021	Classification change due to full database hazard calculation/update.
5.1	10/03/2023	Classification change due to full database hazard calculation/update.

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
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
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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