



14684, 14685, 14688 GPX markers

Signet Pty Ltd

Chemwatch: 5463-59

Version No: 3.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Chemwatch Hazard Alert Code: 2

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L.GHS.AUS.EN.RISK

SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	14684, 14685, 14688 GPX markers
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	PRINTING INK, flammable or PRINTING INK RELATED MATERIAL (including printing ink thinning or reducing compound), flammable
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	Marking. Use according to manufacturer's directions.
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Details of the supplier of the safety data sheet

Registered company name	Signet Pty Ltd
Address	56 Ingleston Road Wakerley QLD 4154 Australia
Telephone	+61 7 3364 2100
Fax	+1 300 304 305
Website	www.signet.net.au
Email	sales@signet.net.au

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 2 9186 1132

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

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Flammable Liquids Category 3, Aspiration Hazard Category 1, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Carcinogenicity Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 3 *LIMITED EVIDENCE
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	  
Signal word	Danger

Hazard statement(s)

AUH019	May form explosive peroxides.
H226	Flammable liquid and vapour.
H304	May be fatal if swallowed and enters airways.
H312	Harmful in contact with skin.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H336	May cause drowsiness or dizziness.
H351	Suspected of causing cancer.
H402	Harmful to aquatic life.

*LIMITED EVIDENCE

Precautionary statement(s) General

P101	If medical advice is needed, have product container or label at hand.
P102	Keep out of reach of children.
P103	Read carefully and follow all instructions.

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 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.
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.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
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.

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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.
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SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1330-20-7	40-80	<u>xylene</u>
13463-67-7	<30	<u>titanium dioxide (anatase)</u>
100-41-4	5-20	<u>ethylbenzene</u>
107-98-2	<20	<u>propylene glycol monomethyl ether - alpha isomer</u>
1333-86-4	<10	<u>carbon black</u>
98-82-8	<1	<u>cumene</u>
71-43-2	<0.1	<u>benzene</u>
91-20-3	<0.1	<u>naphthalene</u>
108-88-3	<0.1	<u>toluene</u>

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	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ 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

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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 naphthalene intoxication: Naphthalene requires hepatic and microsomal activation prior to the production of toxic effects. Liver microsomes catalyse the initial synthesis of the reactive 1,2-epoxide intermediate which is subsequently oxidised to naphthalene dihydrodiol and alpha-naphthol. The 2-naphthoquinones are thought to produce haemolysis, the 1,2-naphthoquinones are thought to be responsible for producing cataracts in rabbits, and the glutathione-adducts of naphthalene-1,2-oxide are probably responsible for pulmonary toxicity. Suggested treatment regime:

- Induce emesis and/or perform gastric lavage with large amounts of warm water where oral poisoning is suspected.
- Instill a saline cathartic such as magnesium or sodium sulfate in water (15 to 30g).
- Demulcents such as milk, egg white, gelatin, or other protein solutions may be useful after the stomach is emptied but oils should be avoided because they promote absorption.
- If eyes/skin contaminated, flush with warm water followed by the application of a bland ointment.
- Severe anaemia, due to haemolysis, may require small repeated blood transfusions, preferably with red cells from a non-sensitive individual.
- Where intravascular haemolysis, with haemoglobinuria occurs, protect the kidneys by promoting a brisk flow of dilute urine with, for example, an osmotic diuretic such as mannitol. It may be useful to alkalinise the urine with small amounts of sodium bicarbonate but many researchers doubt whether this prevents blockage of the renal tubules.
- Use supportive measures in the case of acute renal failure. GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, 5th Ed.

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

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ If safe, switch off electrical equipment until vapour fire hazard removed. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ DO NOT approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▸ Liquid and vapour are flammable. ▸ Moderate fire hazard when exposed to heat or flame. ▸ Vapour forms an explosive mixture with air.

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	<ul style="list-style-type: none"> ▸ Moderate explosion hazard when exposed to heat or flame. ▸ Vapour may travel a considerable distance to source of ignition. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include: carbon dioxide (CO₂) carbon monoxide (CO) metal oxides other pyrolysis products typical of burning organic material.</p>
HAZCHEM	•3Y

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	<ul style="list-style-type: none">‣ Remove all ignition sources.‣ Clean up all spills immediately.‣ Avoid breathing vapours and contact with skin and eyes.‣ Control personal contact with the substance, by using protective equipment.‣ Contain and absorb small quantities with vermiculite or other absorbent material.‣ Wipe up.‣ Collect residues in a flammable waste container.				
Major Spills	Chemical Class: aromatic hydrocarbons				
	For release onto land: recommended sorbents listed in order of priority.				
	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
	LAND SPILL - SMALL				
	Feathers - pillow	1	throw	pitchfork	DGC, RT
	cross-linked polymer - particulate	2	shovel	shovel	R,W,SS
	cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT
	sorbent clay - particulate	3	shovel	shovel	R, I, P,
	treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I
	wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT
	LAND SPILL - MEDIUM				
	cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
	treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I
	sorbent clay - particulate	3	blower	skiploader	R, I, P
	polypropylene - particulate	3	blower	skiploader	W, SS, DGC
	feathers - pillow	3	throw	skiploader	DGC, RT
	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
Legend					
DGC: Not effective where ground cover is dense					
R; Not reusable					
I: Not incinerable					
P: Effectiveness reduced when rainy					
RT:Not effective where terrain is rugged					
SS: Not for use within environmentally sensitive sites					
W: Effectiveness reduced when windy					
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;					
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988					
<ul style="list-style-type: none">‣ Clear area of personnel and move upwind.‣ Alert Fire Brigade and tell them location and nature of hazard.‣ May be violently or explosively reactive.‣ Wear breathing apparatus plus protective gloves.‣ Prevent, by any means available, spillage from entering drains or water course.‣ Consider evacuation (or protect in place).‣ No smoking, naked lights or ignition sources.					

Continued...

- ▶ 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**
- The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe
- ▶ **DO NOT concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential.**
 - ▶ Any static discharge is also a source of hazard.
 - ▶ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina.
 - ▶ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage.
 - ▶ Add inhibitor to any distillate as required.
 - ▶ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely.
- The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example.
- Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.
- ▶ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date.
 - ▶ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date.
 - ▶ Unopened containers received from the supplier should be safe to store for 18 months.
 - ▶ Opened containers should not be stored for more than 12 months.
 - ▶ Electrostatic discharge may be generated during pumping - this may result in fire.
 - ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment.
 - ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (≤ 1 m/sec until fill pipe submerged to twice its diameter, then ≤ 7 m/sec).
 - ▶ Avoid splash filling.
 - ▶ Do NOT use compressed air for filling discharging or handling operations.
 - ▶ Avoid all personal contact, including inhalation.
 - ▶ Wear protective clothing when risk of overexposure occurs.
 - ▶ Use in a well-ventilated area.
 - ▶ Prevent concentration in hollows and sumps.
 - ▶ **DO NOT enter confined spaces until atmosphere has been checked.**
 - ▶ Avoid smoking, naked lights or ignition sources.
 - ▶ Avoid generation of static electricity.
 - ▶ **DO NOT use plastic buckets.**
 - ▶ Earth all lines and equipment.
 - ▶ Use spark-free tools when handling.
 - ▶ Avoid contact with incompatible materials.
 - ▶ **When handling, DO NOT eat, drink or smoke.**
 - ▶ Keep containers securely sealed when not in use.
 - ▶ 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.

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Other information	<ul style="list-style-type: none"> Store in original containers in approved flammable liquid storage area. Store away from incompatible materials in a cool, dry, well-ventilated area. DO NOT store in pits, depressions, basements or areas where vapours may be trapped. No smoking, naked lights, heat or ignition sources. Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access. Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances. Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems. Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors. Keep adsorbents for leaks and spills readily available. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS. <p>In addition, for tank storages (where appropriate):</p> <ul style="list-style-type: none"> Store in grounded, properly designed and approved vessels and away from incompatible materials. For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up. Storage tanks should be above ground and diked to hold entire contents.
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Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> Packing as supplied by manufacturer. Plastic containers may only be used if approved for flammable liquid. Check that containers are clearly labelled and free from leaks. For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	<p>For alkyl aromatics:</p> <p>The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.</p> <ul style="list-style-type: none"> Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids. Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides. Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily. Alkali metals accelerate the oxidation while CO₂ as co-oxidant enhances the selectivity. Microwave conditions give improved yields of the oxidation products. Photo-oxidation products may occur following reaction with hydroxyl radicals and NO_x - these may be components of photochemical smogs. <p>Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007</p> <ul style="list-style-type: none"> Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. Aromatics can react exothermically with bases and with diazo compounds. WARNING: Avoid or control reaction with peroxides. All <i>transition metal</i> peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive. Avoid reaction with borohydrides or cyanoborohydrides



X — Must not be stored together

O — May be stored together with specific precautions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes

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of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

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	titanium dioxide (anatase)	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ethylbenzene	Ethyl benzene	100 ppm / 434 mg/m3	543 mg/m3 / 125 ppm	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether	100 ppm / 369 mg/m3	553 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	cumene	Cumene	25 ppm / 125 mg/m3	375 mg/m3 / 75 ppm	Not Available	Not Available
Australia Exposure Standards	benzene	Benzene	1 ppm / 3.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	naphthalene	Naphthalene	10 ppm / 52 mg/m3	79 mg/m3 / 15 ppm	Not Available	Not Available
Australia Exposure Standards	toluene	Toluene	50 ppm / 191 mg/m3	574 mg/m3 / 150 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
xylene	Not Available	Not Available	Not Available
titanium dioxide (anatase)	30 mg/m3	330 mg/m3	2,000 mg/m3
ethylbenzene	Not Available	Not Available	Not Available
propylene glycol monomethyl ether - alpha isomer	100 ppm	160 ppm	660 ppm
carbon black	9 mg/m3	99 mg/m3	590 mg/m3
cumene	Not Available	Not Available	Not Available
benzene	Not Available	Not Available	Not Available
naphthalene	15 ppm	83 ppm	500 ppm
toluene	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
xylene	900 ppm	Not Available
titanium dioxide (anatase)	5,000 mg/m3	Not Available
ethylbenzene	800 ppm	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
cumene	900 ppm	Not Available
benzene	500 ppm	Not Available
naphthalene	250 ppm	Not Available
toluene	500 ppm	Not Available

MATERIAL DATA


NOTE E: Substances with specific effects on human health that are classified as carcinogenic, mutagenic and/ or toxic for reproduction in categories 1 or 2 are ascribed Note E if they are classified as very toxic (T+), toxic (T) or harmful (Xn). For these substances the risk phrases R20, R21, R22, R23, R24, R25, R26, R27, R28, R39, R68, R48 and R65 and all combinations of these risk phrases shall be preceded by the word "Also".

R45-23: May cause cancer. Also toxic by inhalation

This note applies only to certain complex oil-derived substances in Annex VI.

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

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>	
	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.)
Appropriate engineering controls	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
Appropriate engineering controls	4: Large hood or large air mass in motion	4: Small hood-local control only
	<p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p> <ul style="list-style-type: none"> 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 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) 	
Personal protection		

Eye and face protection	<ul style="list-style-type: none"> ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · 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 <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower. ▸ 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 and shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

Respiratory protection

Type A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Continued...

"Forsberg Clothing Performance Index".

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

computer-generated selection:

14684, 14685, 14688 GPX markers

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on 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	Flammable liquid with characteristic odour; not miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	120	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	24	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	7.8	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	1.0	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (Not Available%)	Not Available

Continued...

Vapour density (Air = 1) Not Available

VOC g/L Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information**Information on toxicological effects**

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Considered an unlikely route of entry in commercial/industrial environments The liquid may produce considerable gastrointestinal discomfort and may be harmful or toxic if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>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.</p>
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular</p>

Continued...

14684, 14685, 14688 GPX markers

atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats (which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure.

Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer. Beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

In a two-year inhalation study, groups of mice were exposed at 0, 10 or 30 ppm naphthalene, 6 hours/day, 5 days/week for 103 weeks. Female mice showed an increase of pulmonary alveolar/bronchiolar adenomas at 30 ppm. There was no increase in the incidence of tumours in male mice. Naphthalene inhalation was associated with an increase in the incidence and severity of chronic inflammation, metaplasia of the olfactory epithelium, and hyperplasia of the respiratory epithelium in the nose, and chronic inflammation of the lungs of both sexes.

Repeated oral doses of 3 g/kg for propylene glycol monomethyl ether (PGME) produced minor changes in the liver and kidneys in rats. Repeated doses on the skin over a 90-day period resulted in absorption and anaesthetic death at 7-10 ml/kg/day. Mild narcosis was observed after topical application of 2-4 ml/kg/day.

Administration of 2% PGME in drinking water ad libitum to males for 25 days did not elicit significant changes in testes or seminal vesicle and coagulating gland weights or in peripheral leukocyte counts. No significant testicular toxicity was found in rats or rabbits that were exposed at up to 3000 PGME, 6 hours/day, 5 days/week for 13 weeks. Oral and parenteral administration to pregnant rabbits, mice and rats did not induce congenital malformations at concentrations up to 1800 mg/kg/day.

In a study on the teratogenic potential of the acetate of the beta-isomer (2-methoxy-1-propyl acetate), a significant increase in the number of litters with abnormal rats and rabbits was found after inhalation exposure by the mothers to 2700 ppm or 550 ppm, respectively, on days 6 to 15, or 6 to 18 of gestation. The rabbit inhalation no-observed-adverse effect concentration was 145 ppm. A similar embryotoxicity profile was seen after inhalation of 2-methoxy-1-propanol (beta-PGMA). In contrast to the alpha-isomer, beta-PGMA is oxidised in rats to 2-methoxypropionic acid.

Male dogs exposed to the beta-isomer, developed numerous spermophages in epididymi. Administration of high doses of the beta-isomer to rats, by gavage, caused delayed ossification of the skull of rat foetus.

Whilst alpha-PGMA undergoes hepatic O-demethylation as the principal pathway, the beta-isomer is detoxified by alcohol/aldehyde dehydrogenase. Commercial PGME contains low concentrations of the beta-isomer.

Long term exposure to the dusts of titanium and several of its compounds produces chronic lung disease (fibrosis) in animals. Radiological evidence exists amongst titanium dioxide workers suggesting chronic lung changes which resemble a slight form of silicosis. Workers chronically exposed to titanium or titanium dioxide dusts show a high incidence of chronic bronchitis (endobronchitis and peribronchitis). Early stages of this disease are characterised by impaired pulmonary respiration and ventilatory capacity and by reduced blood alkalinity. Cardiac changes characteristic of pulmonary disease (with hypertrophy of the right auricle) have also been observed amongst workers.

Titanium employed in implants has provoked immune responses which occur locally as metallosis and systemically as raised serum levels of activated T-lymphocytes. Some concern has been expressed about the potential for generating bone-resorbing mediators associated with titanium wear-debris.

The largest of the cohort studies was among white male production workers in the titanium dioxide industry in six European countries. The study indicated a slightly increased risk for lung cancer compared with the general population. However, there was no evidence of an exposure-response relationship within the cohort. No increase in the mortality rates for kidney cancer was found when the cohort was compared with the general population, but there was a suggestion of an exposure-response relationship in internal analyses. The other cohort studies, both of which were conducted in the USA, did not report an increased risk for lung cancer or cancer at any other site; no results for kidney cancer were reported, presumably because there were few cases.

One population-based case-control study conducted in Montreal did not indicate an increased risk for lung or kidney cancer.

In summary, the studies do not suggest an association between occupational exposure to titanium dioxide as it occurred in recent decades in western Europe and North America and risk for cancer.

All the studies had methodological limitations; misclassification of exposure could not be ruled out. None of the studies was designed to assess the impact of particle size (fine or ultrafine) or the potential effect of the coating compounds on the risk for lung cancer.

An increased incidence of lung adenomas in rats of both sexes and of cystic keratinising lesions, diagnosed as squamous cell carcinomas in female rats, was seen in animals subject to high doses of inhaled titanium dioxide. Intratracheal delivery of titanium dioxide in combination with benz[a]pyrene produced an increase in benign and malignant tumours of the larynx, trachea and lungs in hamsters.

Squamous cell carcinomas developed after exposure to 250 mg/m³ for 6 hours/day, 5 days/week for 2 years in rats; the type of carcinoma that developed was considered to be a unique experimentally induced tumour and to be of questionable relevance for extrapolation of the results to humans. Given the extremely high level of dust in the lungs, the carcinomas were postulated to be the result of saturation of the normal pulmonary clearance mechanisms. At 50 mg/m³, massive accumulations of dust-laden macrophages, foamy dust cells and free particles were considered indicative of such overload.

Industrial workers exposed to a maximum level of ethylbenzene 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.

Prolonged and repeated exposure may be harmful to the central nervous system (CNS), upper respiratory tract, and/or may cause liver disorders. It may also cause drying, scaling and blistering of the skin.

Rats and mice exposed to ethylbenzene for 6 hours daily, 5 days a week for 104 and 103 weeks respectively showed a statistically significant increase in kidney tumours in male and female rats, lung tumours in male mice, and liver tumours in female mice exposed to 750 ppm ethylbenzene.

14684, 14685, 14688 GPX markers

Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms. Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.

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

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

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

14684, 14685, 14688 GPX markers	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
titanium dioxide (anatase)	TOXICITY	IRRITATION
	dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; >2.28 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; >=2000 mg/kg ^[1]	
ethylbenzene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 17800 mg/kg ^[2]	Eye (rabbit): 500 mg - SEVERE
	Inhalation(Rat) LC50; 17.2 mg/l4h ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; 3500 mg/kg ^[2]	Skin (rabbit): 15 mg/24h mild
propylene glycol monomethyl ether - alpha isomer	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit) 230 mg mild
	Inhalation(Rat) LC50; >6 mg/l4h ^[2]	Eye (rabbit) 500 mg/24 h. - mild
	Oral (Rat) LD50; 3739 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; >8000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
cumene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2000 mg/kg ^[2]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; 39 mg/L4h ^[2]	Eye (rabbit): 86 mg mild
	Oral (Rat) LD50; 1400 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 10 mg/24h mild
		Skin (rabbit):100 mg/24h moderate
	TOXICITY	IRRITATION
		Skin: no adverse effect observed (not irritating) ^[1]

14684, 14685, 14688 GPX markers

benzene	TOXICITY	IRRITATION
	dermal (mouse) LD50: 48 mg/kg ^[2]	Eye (rabbit): 2 mg/24h - SEVERE
	Inhalation(Rat) LC50; 43.767 mg/L4h ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50; 930 mg/kg ^[2]	SKIN (rabbit):20 mg/24h - moderate Skin: adverse effect observed (irritating) ^[1]
naphthalene	TOXICITY	IRRITATION
	dermal (rat) LD50: >2500 mg/kg ^[2]	Eye (rabbit): 100 mg - mild
	Inhalation(Rat) LC50; >0.4 mg/l4h ^[1]	Skin (rabbit):495 mg (open) - mild
	Oral (Rat) LD50; 490 mg/kg ^[2]	
toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation(Rat) LC50; >13350 ppm4h ^[2]	Eye (rabbit):0.87 mg - mild
	Oral (Rat) LD50; 636 mg/kg ^[2]	Eye (rabbit):100 mg/30sec - mild
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
		Skin: adverse effect observed (irritating) ^[1] Skin: no adverse effect observed (not 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.
TITANIUM DIOXIDE (ANATASE)	<p>For titanium dioxide:</p> <p>Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.</p> <p>Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.</p> <p>Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.</p> <p>Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.</p> <p>Animal carcinogenicity data</p>

14684, 14685, 14688 GPX markers

	<p>Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.</p> <p>In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.</p> <p>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.</p>
ETHYLBENZENE	<p>Liver changes, uterine tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded.</p> <p>Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylglyoxylic acids; whereas rats and rabbits excrete hippuric acid and phenacetic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances.</p> <p>Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys.</p> <p>Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (<i>400 ppm and greater</i>) of ethylbenzene</p> <p>In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncytial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland.</p> <p>In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity</p> <p>Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination.</p> <p>NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p>
PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER	<p>for propylene glycol ethers (PGEs):</p> <p>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).</p> <p>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.</p> <p>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).</p> <p>This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.</p> <p>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.</p> <p>As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. 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.</p> <p>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</p>

14684, 14685, 14688 GPX markers

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 sensitizers.

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.

NOTE: For PGE - mixed isomers: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.

CARBON BLACK

Inhalation (rat) TCLo: 50 mg/m³/6h/90D-I Nil reported No significant acute toxicological data identified in literature search.

CUMENE

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

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

For aromatic terpenes:

Acute toxicity: Mammalian LD50 for p-cymene have shown it to have low toxic potential. Similar studies with cumene have concurred with these results

In general, the studies indicate that p-cymene (p-methylisopropylbenzene) or cumene (isopropylbenzene) is rapidly absorbed by oral or inhalation routes. They undergo oxidation (hydroxylation) of the side chain isopropyl substituent and, in the case of p-cymene, the methyl substituent to yield polar oxygenated metabolites. These metabolites are either excreted unchanged in the urine or undergo Phase II conjugation with glucuronic acid and/or glycine followed by excretion in the urine. Unchanged p-cymene or cumene were not detected in the urine or faeces.

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

14684, 14685, 14688 GPX markers

	<p>Repeat Dose Toxicity: Subacute Studies: Groups of 7 to 12 male rats were exposed to 0, 50, or 250 ppm of p-cymene for 6 hours/day, 5 days/week for 4 weeks with an 8-week recovery period. there was no overt toxicity in the treated rats and no effect on body weight or terminal weight of the brain, cerebellum or whole brain. There was also no effect on regional enzyme activities, regional protein synthesis or regional neurotransmitter concentrations.</p> <p>Cumene has been tested by the National Toxicology Program (NTP) in both rats and mice. Animals were exposed to up to 4,000 ppm cumene by whole-body inhalation for 12-13 days over a period of 16-17 days. In rats, all animals died at 4,000 ppm, and about half the animals died at the next exposure concentration (2,000 ppm). Varying degrees of ataxia were reported in surviving rats exposed to 500 to 2,000 ppm cumene. Increased relative liver and kidney weights were reported in rats exposed to cumene. In exposed male rats, hyaline droplets in the renal cortical tubules were reported. At 2,000 ppm, superlative inflammation of the lung was reported in 40% of the rats. In mice, all animals died at the 2 highest exposures (2,000 and 4,000 ppm). At 1,000 ppm, 80% of the female mice died and male mice showed varying degrees of ataxia. Increased relative liver and kidney weights were reported in mice exposed to cumene. Decreased thymus weight was reported in male mice exposed to 1,000 ppm of cumene. No histopathological findings accompanied the organ weight changes. A NOAEL of 1,000 ppm was determined for female rats and male mice and a NOAEL of 500 ppm was determined for female mice based on mortality and histopathological findings.</p> <p>Chronic toxicity: The US EPA concluded that there is some evidence that suggests that cumene is not likely to produce a carcinogenic response (i.e., numerous genotoxic tests, including gene mutation, chromosomal aberration, and primary DNA damage tests, all but one of which were negative or not reproducible) In addition, EPA noted that cumene does not appear to metabolise to highly reactive chemical species and in terms of metabolism, cumene is analogous to methyl benzene for which a 2-year inhalation study was conducted by NTP and no evidence of carcinogenic activity was reported in either rats or mice. Given that the only structural difference between p-cymene and cumene is the presence of a second alkyl substituent (isopropylbenzene versus p-methylisopropylbenzene), similar conclusions can be drawn for p-cymene, particularly since the pharmacokinetic, metabolic and toxicologic data that are available support this conclusion.</p> <p>Reproductive toxicity: Taking into consideration the rapid metabolism and excretion of cumene, the US EPA concluded, "cumene has low potential for reproductive toxicity."</p> <p>Developmental toxicity: Even at maternally toxic concentrations exposure to cumene vapor did not produce developmental toxicity in rats. However the US EPA determined that the changes in gestational parameters of the rabbits, though not significant, were consistent in indicating possible developmental effects and therefore set the NOAEL in rabbits for both developmental and maternal effects at 1,206 ppm and the LOAEL at 2,297 ppm, respectively (as reported in EPA, 1997). Since both cumene and p-cymene exhibit such similar pharmacokinetic and metabolic profiles, and show no evidence of toxicity at levels of exposure similar to those experienced by humans, further teratogenic or developmental testing is not recommended</p> <p>Genotoxicity: The genotoxicity database on p-cymene and cumene shows no mutagenic potential in the Ames assay. In cytogenetic assays, there is no evidence of a genotoxic potential in vitro. In whole animals, the genotoxicity results for cumene are mixed showing weakly positive results in micronuclei induction in rats, but no evidence of genotoxicity in mice.</p> <p>Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]</p>
BENZENE	<p>Inhalation (man) TCLo: 150 ppm/1y - I</p> <p>Data demonstrate that during inhalation exposure, aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure.</p> <p>Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism.</p> <p>The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.</p> <p>WARNING: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS.</p>
NAPHTHALENE	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
TOLUENE	<p>For toluene:</p> <p>Acute Toxicity</p> <p>Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.</p> <p>Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.</p> <p>Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.</p> <p>Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.</p> <p>Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea. Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death</p> <p>Toluene can also strip the skin of lipids causing dermatitis</p>

14684, 14685, 14688 GPX markers

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

Subchronic/Chronic Effects:

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

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

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

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

Developmental/Reproductive Toxicity

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

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

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

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

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

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

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

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

XYLENE & ETHYLBENZENE

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

XYLENE & CUMENE & BENZENE & TOLUENE

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.

ETHYLBENZENE & NAPHTHALENE

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

ETHYLBENZENE & CARBON BLACK & CUMENE & NAPHTHALENE

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

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**

14684, 14685, 14688 GPX markers	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
	NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
	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
titanium dioxide (anatase)	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<1.1-9.6	7
	NOEC(ECx)	504h	Crustacea	0.02mg/l	4
	LC50	96h	Fish	1.85-3.06mg/l	4
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
ethylbenzene	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	720h	Fish	0.381mg/L	4
	LC50	96h	Fish	3.381-4.075mg/L	4
	EC50	72h	Algae or other aquatic plants	4.6mg/l	1
	EC50	48h	Crustacea	1.37-4.4mg/l	4
	EC50	96h	Algae or other aquatic plants	3.6mg/l	2
propylene glycol monomethyl ether - alpha isomer	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	168h	Algae or other aquatic plants	>1000mg/l	1
	LC50	96h	Fish	>=1000mg/l	2
	EC50	72h	Algae or other aquatic plants	>500mg/l	2
	EC50	48h	Crustacea	23300mg/l	1
carbon black	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	24h	Crustacea	3200mg/l	1
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	EC50	48h	Crustacea	33.076-41.968mg/l	4
cumene	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	96h	Crustacea	0.4mg/l	1
	LC50	96h	Fish	2.7mg/l	2
	EC50	72h	Algae or other aquatic plants	1.29mg/l	2
	EC50	48h	Crustacea	4mg/l	1
benzene	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4
	ErC50	72h	Algae or other aquatic plants	>1360mg/l	1
benzene	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	2.54-7.217mg/L	4

Continued...

14684, 14685, 14688 GPX markers

	EC50	72h	Algae or other aquatic plants	29mg/l	1
	EC50	48h	Crustacea	7.578-13.983mg/L	4
	EC50	96h	Algae or other aquatic plants	>1360mg/l	1
naphthalene	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	23-146	7
	NOEC(ECx)	48h	Fish	0.013mg/L	4
	LC50	96h	Fish	0.51mg/l	4
	EC50	72h	Algae or other aquatic plants	~0.4~0.5mg/l	2
	EC50	48h	Crustacea	1.09-3.4mg/l	4
toluene	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	168h	Crustacea	0.74mg/L	5
	LC50	96h	Fish	5-35mg/l	4
	EC50	48h	Crustacea	3.78mg/L	5
	EC50	96h	Algae or other aquatic plants	>376.71mg/L	4
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					

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

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

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
titanium dioxide (anatase)	HIGH	HIGH
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
propylene glycol monomethyl ether - alpha isomer	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
cumene	HIGH	HIGH
benzene	HIGH (Half-life = 720 days)	LOW (Half-life = 20.88 days)
naphthalene	HIGH (Half-life = 258 days)	LOW (Half-life = 1.23 days)
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
xylene	MEDIUM (BCF = 740)
titanium dioxide (anatase)	LOW (BCF = 10)
ethylbenzene	LOW (BCF = 79.43)
propylene glycol monomethyl ether - alpha isomer	LOW (BCF = 2)
cumene	LOW (BCF = 35.5)
benzene	HIGH (BCF = 4360)
naphthalene	HIGH (BCF = 18000)
toluene	LOW (BCF = 90)

Mobility in soil

Ingredient	Mobility
titanium dioxide (anatase)	LOW (KOC = 23.74)
ethylbenzene	LOW (KOC = 517.8)

Continued...

Ingredient	Mobility
propylene glycol monomethyl ether - alpha isomer	HIGH (KOC = 1)
cumene	LOW (KOC = 817.2)
benzene	LOW (KOC = 165.5)
naphthalene	LOW (KOC = 1837)
toluene	LOW (KOC = 268)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible.
	<p>Otherwise:</p> <ul style="list-style-type: none"> If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> Reduction Reuse Recycling Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> DO NOT allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. Recycle wherever possible. Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. Dispose of by: burial in a land-fill specifically 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	•3Y

Land transport (ADG)

UN number	1210
UN proper shipping name	PRINTING INK, flammable or PRINTING INK RELATED MATERIAL (including printing ink thinning or reducing compound), flammable
Transport hazard class(es)	Class 3
	Subrisk Not Applicable
Packing group	III
Environmental hazard	Not Applicable

Special precautions for user	Special provisions	163 223 367
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

UN number	1210	
UN proper shipping name	Printing ink flammable; Printing ink related material (including printing ink thinning or reducing compound), flammable	
Transport hazard class(es)	ICAO/IATA Class	3
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	3L
Packing group	III	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions	A3 A72 A192
	Cargo Only Packing Instructions	366
	Cargo Only Maximum Qty / Pack	220 L
	Passenger and Cargo Packing Instructions	355
	Passenger and Cargo Maximum Qty / Pack	60 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y344
	Passenger and Cargo Limited Maximum Qty / Pack	10 L

Sea transport (IMDG-Code / GGVSee)

UN number	1210	
UN proper shipping name	PRINTING INK flammable or PRINTING INK RELATED MATERIAL (including printing ink thinning or reducing compound), flammable	
Transport hazard class(es)	IMDG Class	3
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Not Applicable	
Special precautions for user	EMS Number	F-E , S-D
	Special provisions	163 223 367 955
	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
titanium dioxide (anatase)	Not Available
ethylbenzene	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available
carbon black	Not Available
cumene	Not Available
benzene	Not Available
naphthalene	Not Available
toluene	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
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Continued...

Product name	Ship Type
xylene	Not Available
titanium dioxide (anatase)	Not Available
ethylbenzene	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available
carbon black	Not Available
cumene	Not Available
benzene	Not Available
naphthalene	Not Available
toluene	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 Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 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

titanium dioxide (anatase) 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 2B: Possibly carcinogenic to humans
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ethylbenzene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
 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 2B: Possibly carcinogenic to humans

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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

carbon black is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 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 2B: Possibly carcinogenic to humans
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

cumene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 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 2B: Possibly carcinogenic to humans

benzene is found on the following regulatory lists

Australia - New South Wales Work Health and Safety Regulation - Restricted carcinogens

Australia - Northern Territories Work Health and Safety National Uniform Legislation Regulations- Restricted carcinogens

Australia - Queensland Work Health and Safety Regulation - Restricted Carcinogens

Australia - South Australia - Work Health and Safety Regulations - Restricted carcinogens

Australia - Tasmania - Work Health and Safety Regulations - Restricted carcinogens

Australia - Western Australia Carcinogenic substances to be used only for purposes approved by the Commissioner

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

naphthalene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

Australian Inventory of Industrial Chemicals (AIIC)

toluene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

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

Australia Model Work Health and Safety Regulations - Restricted carcinogens

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 7

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

Chemical Footprint Project - Chemicals of High Concern List

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (xylene; ethylbenzene; propylene glycol monomethyl ether - alpha isomer; carbon black; cumene; benzene; naphthalene; toluene)
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:	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</p>

SECTION 16 Other information

Revision Date	20/08/2021
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Initial Date	08/04/2021
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SDS Version Summary

Version	Date of Update	Sections Updated
3.1	20/08/2021	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
AIIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDL: 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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