ColorSpec Tinter (Colorspec No Mix_E Basecoat)
Motor Active

Chemwatch Hazard Alert Code: **3**

Chemwatch: **4798-80**
Version No: **10.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **09/02/2021**
Print Date: **09/02/2021**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	ColorSpec Tinter (Colorspec No Mix_E Basecoat)
Chemical Name	Not Applicable
Synonyms	E-01 Black Tinter; E-02 Blue Tinter; E-03 Bright Blue Tinter; E-04 Bright Gold Tinter; E-05 Bright Red Tinter; E-06 Custard Tinter; E-07 Copper Tinter; E-10 Deep Black Tinter; E-11 Cobalt Blue Tinter; E-12 Maroon Tinter; E-13 Deep Maroon Tinter; E-14 Garnet Tinter; E-15 Green Tinter; E-16 Green Blue Tinter; E-17 Green Gold Tinter; E-18 Grey Black Tinter; E-19 Empress Black Tinter; E-20 Light Red Oxide Tinter; E-21 Lime Tinter; E-22 Magenta Tinter; E-23 Midnight Blue Tinter; E-24 Red Gold Tinter; E-25 Red Maroon Tinter; E-28 Turquoise Tinter; E-30 Violet Tinter; E-31 White Tinter; E-32 Yellow Gold Tinter; E-33 Yellow Ochre Tinter; E-34 Special Violet Tinter; E-35 Port Wine Red Tinter; E-36 Deep Blue Tinter; E-37 Special Deep Black Tinter; E-38 Special Red Maroon Tinter; E-39 HS Special Red Tinter; E-41 Reduced Black Tinter; E-51 Red Yellow Tinter; E-52 Topaz Tinter; E-53 Organic Orange Tinter; E-54 Special Silver Bright Fine; E-55 Special Silver Coarse; E-56 Silver Dollar Bright Coarse; E-57 Silver Dollar Bright Fine; E-59 Metallic Additive Tinter; E-60 Stabilizer Additive Tinter; E-61 Effect White Tinter; E-62 HS Special White Metallic/Aluminium Tinter; E-63 HS Special Yellow Metallic/Aluminium Tinter; E-65 Fine Metallic Alluminium Tinter; E-66 Medium Metallic Alluminium Tinter; E-67 Coarse Metallic Alluminium Tinter; E-68 Extra Fine Silver Metallic/Aluminium Tinter; E-69 Fine Silver Metallic/Aluminium Tinter; E-70 Silver Metallic/Aluminium Tinter; E-71 Medium Silver Metallic/Aluminium Tinter; E-72 Coarse Silver Metallic/Aluminium Tinter; E-74 Coarse Aluminium Metallic Tinter; E-75 Extra Coarse Aluminium Metallic Tinter; E-99 Metallic Raiser Aluminium Tinter; E-77 Fine White Pearl Tinter; E-78 White Sparkle Pearl Tinter; E-80 Yellow Pearl Tinter; E-82 Fine Yellow Gold Pearl Tinter; E-83 Orange Pearl Tinter; E-86 Copper Pearl Tinter; E-87 Bright Russet Pearl Tinter; E-88 Fine Russet Pearl Tinter; E-89 Blue Russet Pearl Tinter; E-90 Red Blue Pearl Tinter; E-91 Fine Blue Pearl Tinter; E-92 Green Blue Pearl Tinter; E-93 Fine Green Pearl Tinter; E-95 Blue Green Pearl Tinter; E-96 Red Pearl Tinter; E-97 Fine Silver Pearl Tinter; E-98 Fine Violet Pearl Tinter; E-42 Silk Silver Xirallic Tinter; E-43 Silk Russet Xirallic Tinter; E-44 Silk Gold Xirallic Tinter; E-45 Silk Blue Xirallic Tinter; E-46 Silk Red Xirallic Tinter; E-47 Silk Green Xirallic Tinter; E-48 Silk Copper Xirallic Tinter; E-101 Red Candy; E-102 Brandy Wine Candy; E-103 Yellow Candy; E-104 Green Candy; TU50ML- ColorSpec Touch-Up Bottle 50ml; TL250ML- ColorSpec Tinters, 250ml; TL1L- ColorSpec Tinters, 1 Litre; TL2L- ColorSpec Tinters, 2 Litre; TL500- ColorSpec Tinters, 500ml
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Automotive refinish. Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	**Motor Active**
Address	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia
Telephone	+61 2 9737 9422|1800 350 622
Fax	+61 2 9737 9414
Website	www.motoractive.com.au
Email	andrew.spira@motoractive.com.au

Emergency telephone number

Association / Organisation	**MotorActive**
Emergency telephone numbers	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
Other emergency telephone numbers	13 11 26 (In Case of Emergency contact: Poison Information Hotline)

SECTION 2 Hazards identification

Classification of the substance or mixture

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

ChemWatch Hazard Ratings

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

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

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Poisons Schedule	S5
Classification [1]	Flammable Liquid Category 2, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Germ cell mutagenicity Category 2, Carcinogenicity Category 1A, Specific target organ toxicity - repeated exposure Category 2, Chronic Aquatic Hazard Category 2
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)

H225	Highly flammable liquid and vapour.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H373	May cause damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection/hearing protection/...
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof [electrical/ventilating/lighting/...] equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P321	Specific treatment (see ... on this label).
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/... if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Continued...

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
123-86-4	>60	n-butyl acetate
98-56-6	30-60	4-chlorobenzotrifluoride
13463-67-7	10-30	titanium dioxide
108-65-6	10-30	propylene glycol monomethyl ether acetate, alpha-isomer
112926-00-8	10-30	silica precipitated, crystalline free
78-93-3	10-20	methyl ethyl ketone
1330-20-7	10-20	xylene
67-64-1	10-20	acetone
763-69-9	<10	ethyl-3-ethoxypropionate
108-10-1	<10	methyl isobutyl ketone
64742-48-9.	<10	naphtha petroleum, heavy, hydrotreated
64742-95-6.	<10	naphtha petroleum, light aromatic solvent
1309-37-1	<10	ferric oxide
7782-42-5	<10	graphite
1333-86-4	<10	carbon black
7429-90-5	<10	aluminium powder coated
12001-26-2	<10	mica
64742-94-5	<10	solvent naphtha petroleum, heavy aromatic
6358-30-1	<10	C.I. Pigment Violet 23
5567-15-7	<10	C.I. Pigment Yellow 83
71-36-3	<10	n-butanol
123-42-2	<10	diacetone alcohol

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ 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	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
for simple esters:

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.

Continued...

- Monitor and treat, where necessary, for pulmonary oedema .
- Monitor and treat, where necessary, for shock.
- **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

ADVANCED TREATMENT

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

EMERGENCY DEPARTMENT

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

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

For acute or short term repeated exposures to xylene:

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

- Water spray or fog.
- Alcohol stable foam.
- Dry chemical powder.
- Carbon dioxide.

Do not use a water jet to fight fire.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Consider evacuation (or protect in place). ▸ Fight fire from a safe distance, with adequate cover. ▸ If safe, switch off electrical equipment until vapour fire hazard removed. ▸ Use water delivered as a fine spray to control the fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **Do not** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ Liquid and vapour are highly flammable. ▸ Severe fire hazard when exposed to heat, flame and/or oxidisers. ▸ Vapour may travel a considerable distance to source of ignition. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO_2) hydrogen chloride phosgene hydrogen fluoride other pyrolysis products typical of burning organic material.
HAZCHEM	•3YE

SECTION 6 Accidental release measures

Continued...

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb small quantities with vermiculite or other absorbent material. ▸ Wipe up. ▸ Collect residues in a flammable waste container.
Major Spills	▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Consider evacuation (or protect in place). ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse /absorb vapour. ▸ Contain spill with sand, earth or vermiculite. ▸ Use only spark-free shovels and explosion proof equipment. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ If contamination of drains or waterways occurs, advise emergency services.

Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Electrostatic discharge may be generated during pumping - this may result in fire. ▸ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▸ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). ▸ Avoid splash filling. ▸ Do NOT use compressed air for filling discharging or handling operations. ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights, heat or ignition sources. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Vapour may ignite on pumping or pouring due to static electricity. ▸ **DO NOT** use plastic buckets. ▸ Earth and secure metal containers when dispensing or pouring product. ▸ Use spark-free tools when handling. ▸ Avoid contact with incompatible materials. ▸ Keep containers securely sealed. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	▸ Store in original containers in approved flame-proof area. ▸ No smoking, naked lights, heat or ignition sources. ▸ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped. ▸ Keep containers securely sealed. ▸ Store away from incompatible materials in a cool, dry well ventilated area. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ 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;

Continued...

	(ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▸ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▸ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	▸ Avoid strong acids, bases. ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	n-butyl acetate	n-Butyl acetate	150 ppm / 713 mg/m3	950 mg/m3 / 200 ppm	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	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	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Not Available
Australia Exposure Standards	silica precipitated, crystalline free	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica precipitated, crystalline free	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	methyl ethyl ketone	Methyl ethyl ketone (MEK)	150 ppm / 445 mg/m3	890 mg/m3 / 300 ppm	Not Available	Not Available
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	acetone	Acetone	500 ppm / 1185 mg/m3	2375 mg/m3 / 1000 ppm	Not Available	Not Available
Australia Exposure Standards	methyl isobutyl ketone	Methyl isobutyl ketone	50 ppm / 205 mg/m3	307 mg/m3 / 75 ppm	Not Available	Not Available
Australia Exposure Standards	naphtha petroleum, heavy, hydrotreated	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric oxide	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	graphite	Graphite (all forms except fibres) (respirable dust) (natural & synthetic)	3 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium powder coated	Aluminium, pyro powders (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium powder coated	Aluminium (welding fumes) (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium powder coated	Aluminium (metal dust)	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	mica	Mica	2.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	n-butanol	n-Butyl alcohol	Not Available	Not Available	50 ppm / 152 mg/m3	Not Available
Australia Exposure Standards	diacetone alcohol	Diacetone alcohol	50 ppm / 238 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
n-butyl acetate	Butyl acetate, n-	Not Available	Not Available	Not Available
titanium dioxide	Titanium oxide; (Titanium dioxide)	30 mg/m3	330 mg/m3	2,000 mg/m3
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
silica precipitated, crystalline free	Silica gel, amorphous synthetic	18 mg/m3	200 mg/m3	1,200 mg/m3
methyl ethyl ketone	Butanone, 2-; (Methyl ethyl ketone; MEK)	Not Available	Not Available	Not Available
xylene	Xylenes	Not Available	Not Available	Not Available

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Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
acetone	Acetone	Not Available	Not Available	Not Available
ethyl-3-ethoxypropionate	Propionic acid, 3-ethoxy-, ethyl ester; (Ethyl-3-ethoxypropionate)	1.6 ppm	18 ppm	110 ppm
methyl isobutyl ketone	Methyl isobutyl ketone; (Hexone)	75 ppm	500 ppm	3000* ppm
naphtha petroleum, heavy, hydrotreated	Naphtha, hydrotreated heavy; (Isopar L-rev 2)	350 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha petroleum, light aromatic solvent	Naphtha (coal tar); includes solvent naphtha, petroleum (64742-88-7), naphtha (petroleum) light aliphatic, rubber solvent (64742-89-8), heaevy catalytic cracked (64741-54-4), light straight run (64741-46-4), heavy aliphatic solvent (64742-96-7), high flash aromatic and aromatic solvent naphtha (64742-95-6)	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
ferric oxide	Iron oxide; (Ferric oxide)	15 mg/m3	360 mg/m3	2,200 mg/m3
graphite	Carbon; (Graphite, 7782-42-5)	6 mg/m3	330 mg/m3	2,000 mg/m3
carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3
mica	Mica; (mica silicates)	9 mg/m3	99 mg/m3	590 mg/m3
n-butanol	Butyl alcohol, n-; (n-Butanol)	60 ppm	800 ppm	8000** ppm
diacetone alcohol	Hydroxy-4-methyl-2-pentanone, 4-; (Diacetone alcohol)	150 ppm	350 ppm	2100* ppm

Ingredient	Original IDLH	Revised IDLH
n-butyl acetate	1,700 ppm	Not Available
4-chlorobenzotrifluoride	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
silica precipitated, crystalline free	Not Available	Not Available
methyl ethyl ketone	3,000 ppm	Not Available
xylene	900 ppm	Not Available
acetone	2,500 ppm	Not Available
ethyl-3-ethoxypropionate	Not Available	Not Available
methyl isobutyl ketone	500 ppm	Not Available
naphtha petroleum, heavy, hydrotreated	2,500 mg/m3	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
ferric oxide	2,500 mg/m3	Not Available
graphite	1,250 mg/m3	Not Available
carbon black	1,750 mg/m3	Not Available
aluminium powder coated	Not Available	Not Available
mica	1,500 mg/m3	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available	Not Available
C.I. Pigment Violet 23	Not Available	Not Available
C.I. Pigment Yellow 83	Not Available	Not Available
n-butanol	1,400 ppm	Not Available
diacetone alcohol	1,800 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
4-chlorobenzotrifluoride	E	≤ 0.1 ppm
ethyl-3-ethoxypropionate	E	≤ 0.1 ppm
C.I. Pigment Yellow 83	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

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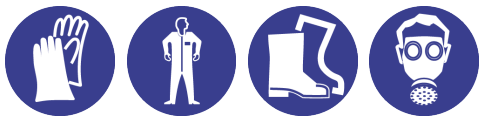
Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. ▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▸ Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▸ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▸ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▸ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▸ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.
Personal protection	
Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber For esters: ▸ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

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Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower. ▸ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▸ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▸ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
ColorSpec Tinter (Colorspec No Mix_E Basecoat)

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

Respiratory protection

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

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

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

Continued...

Information on basic physical and chemical properties

Appearance	Coloured viscous flammable liquid with a strong solvent odour; does not mix with water.

Physical state	Liquid	**Relative density (Water = 1)**	0.90-1.40
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	95-165	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	18 (CC)	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	8	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	8 @20C	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Applicable
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Inhalation hazard is increased at higher temperatures. 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 The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression , headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures. Prolonged exposure may cause headache, nausea and ultimately loss of consciousness.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Open cuts, abraded or irritated skin should not be exposed to this material
Eye	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.

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Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of: - appropriate long-term animal studies - other relevant information Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

ColorSpec Tinter (Colorspec No Mix_E Basecoat)	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

n-butyl acetate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >14100 mg/kg[2]	Eye (human): 300 mg
	Inhalation(Rat) LC50; =0.74 mg/l4hrs[2]	Eye (rabbit): 20 mg (open)-SEVERE
	Oral(Mouse) LD50; 0.006 mg/kg[2]	Eye (rabbit): 20 mg/24h - moderate
		Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating)[1]

4-chlorobenzotrifluoride	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2 mg/kg[2]	Not Available
	Inhalation(Rat) LC50; =33 mg/l4hrs[2]	
	Oral(Rat) LD50; 0.013 mg/kg[2]	

titanium dioxide	**TOXICITY**	**IRRITATION**
	dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50; >=2000 mg/kg[1]	Skin (human): 0.3 mg /3D (int)-mild *
		Skin: no adverse effect observed (not irritating)[1]

propylene glycol monomethyl ether acetate, alpha-isomer	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50; 5155 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

silica precipitated, crystalline free	**TOXICITY**	**IRRITATION**
	Not Available	Eye (rabbit) : 8.3 mg/48h

methyl ethyl ketone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >8.10 mg/kg[1]	Eye (human): 350 ppm -irritant
	Inhalation(Mouse) LC50; 32 mg/L4hrs[2]	Eye (rabbit): 80 mg - irritant
	Oral(Rat) LD50; 2054 mg/kg[1]	Skin (rabbit): 402 mg/24 hr - mild
		Skin (rabbit):13.78mg/24 hr open

xylene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1700 mg/kg[2]	Eye (human): 200 ppm irritant
	Inhalation(Rat) LC50; 5922 ppm4hrs[1]	Eye (rabbit): 5 mg/24h SEVERE
	Oral(Rat) LD50; 8.70 mg/kg[1]	Eye (rabbit): 87 mg mild
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):500 mg/24h moderate
		Skin: adverse effect observed (irritating)[1]

acetone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >7.426 mg/kg[1]	Eye (human): 500 ppm - irritant
	Inhalation(Mouse) LC50; 44 mg/L4hrs[2]	Eye (rabbit): 20mg/24hr -moderate
	Oral(Mouse) LD50; 0.003 mg/kg[2]	Eye (rabbit): 3.95 mg - SEVERE
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 500 mg/24hr - mild
		Skin (rabbit):395mg (open) - mild
		Skin: no adverse effect observed (not irritating)[1]

Continued...

ethyl-3-ethoxypropionate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4.076 mg/kg[1]	Eye (rabbit): 500mg/24h - mild
	Inhalation(Rat) LC50; 1250 ppm4hrs[2]	Skin (rabbit):10 mg/24h open mild
	Oral(Rat) LD50; ~3200-5000 mg/kg[2]	
methyl isobutyl ketone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >16 mg/kg[1]	Eye (human): 200 ppm/15m
	Inhalation(Rat) LC50; ~8.2-16.4 mg/l4hrs[2]	Eye (rabbit): 40 mg - SEVERE
	Oral(Rat) LD50; 0.002 mg/kg[1]	Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h - mild
naphtha petroleum, heavy, hydrotreated	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; 8.5 mg/L4hrs[2]	Skin: adverse effect observed (irritating)[1]
	Oral(Rat) LD50; >4500 mg/kg[1]	
naphtha petroleum, light aromatic solvent	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >5.2 mg/l4hrs[2]	Skin: adverse effect observed (irritating)[1]
	Oral(Rat) LD50; >4500 mg/kg[1]	
ferric oxide	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50; >26.20 mg/kg[1]	Not Available
graphite	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50; >2000 mg/kg[1]	Not Available
carbon black	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >0.003 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]
aluminium powder coated	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50; >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
mica	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
solvent naphtha petroleum, heavy aromatic	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (rabbit): Irritating
	Inhalation(Rat) LC50; >0.17 mg/l4hrs[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50; 512 mg/kg[1]	Skin: adverse effect observed (irritating)[1]
C.I. Pigment Violet 23	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50; 5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): Non-irritating *
		Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Yellow 83	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >3000 mg/kg[1]	Eye (rabbit): non-irritating
	Oral(Rat) LD50; >5000 mg/kg[2]	Skin (rabbit): non-irritating
n-butanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3.434 mg/kg[1]	Eye (human): 50 ppm - irritant
	Inhalation(Rat) LC50; >17.76 mg/l4hrs[2]	Eye (rabbit): 1.6 mg-SEVERE
	Oral(Rat) LD50; 0.001 mg/kg[2]	Eye (rabbit): 24 mg/24h-SEVERE
		Eye: adverse effect observed (irreversible damage)[1]
		Skin (rabbit): 405 mg/24h-moderate
		Skin: adverse effect observed (irritating)[1]

Continued...

diacetone alcohol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 13.630 mg/kg[1]	Eye (human): 100 ppm/15 mins.
	Oral(Rat) LD50; 2520 mg/kg[2]	Eye (rabbit): 5 mg SEVERE
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 500 mg open mild
		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*	

N-BUTYL ACETATE	Generally,linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic. The JEFCA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods **Internationl Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)** **Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998**
4-CHLOROBENZOTRIFLUORIDE	For 4-chlorobenzotrifluoride (PCBTF): **SUBCHRONIC DATA**: A 13-week inhalation study was conducted in rats exposed for 6 hours per day, 5 days a week at concentrations of 0, 10, 51, or 252 ppm. An increase in liver weights was seen in the high dose group. No macroscopic effects were noted. No adverse central nervous system effects were observed as measured by motor activity, functional observation battery, or neuropathology. In a separate study, rats were dosed daily via oral gavage for three months at 0, 10, 40, 150, or 500 mg/kg. Effects noted included initial decrease in body weight gain, decreased food consumption, and changes in biochemical parameters. Increases were noted in liver, kidney, and thyroid weights in both sexes in most treatment groups. Microscopic effects were also observed in these same organs. No overt physical signs of toxicity were observed during treatment. Effects similar to those described in the above two studies have also been observed in shorter inhalation and oral gavage testing. **REPRODUCTIVE TOXICITY**: In a two-generation reproduction study rats were exposed daily via oral gavage at doses of 0, 5, 15, and 45 mg/kg. Only limited reproductive effects were noted. **TERATOGENICITY (birth defects)**: No teratogenicity data are available on this material. **MUTAGENICITY**: This material was found to be negative in the following in vitro mutagenicity studies: chromosomal aberration study, cell transformation assay, DNA repair deficiency assay, and the mouse lymphoma forward mutation assay. In the in vitro Ames test, the compound was generally found to be negative; however two strains at the high dose produced positive results. In the in vitro sister chromatid exchange test, the compound produced positive results. In the in vivo cytogenetic assay in rats, the compound was found to be negative. **CHRONIC EFFECTS/CARCINOGENICITY**: There are no chronic effects or carcinogenicity data available on this material
TITANIUM DIOXIDE	* IUCLID Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. For titanium dioxide: 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. 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. No data were available on genotoxic effects in titanium dioxide-exposed humans. 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. 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. 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. **Animal carcinogenicity data** 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

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	administration in male mice and female rats. 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. 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. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I] *Shin-Etsu SDS for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I]
SILICA PRECIPITATED, CRYSTALLINE FREE	For silica amorphous: Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d. In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin. When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is

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	eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals. After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification. Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser. Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure. Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL. Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected. For Synthetic Amorphous Silica (SAS) Repeated dose toxicity Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet. Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) =1.3 mg/m3 based on mild reversible effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m3 based on reversible effects in the lungs and effects in the nasal cavity. For silane treated synthetic amorphous silica: Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested. There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.
METHYL ETHYL KETONE	Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities. Combinations with chloroform also show increase in toxicity
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.
ACETONE	for acetone: The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals. The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.
ETHYL-3-ETHOXYPROPIONATE	* Union Carbide ** Endura Manufacturing
METHYL ISOBUTYL KETONE	For methyl isobutyl ketone (MIBK): MIBK is primarily absorbed by the lungs in animals and humans; it can however be absorbed by the gastrointestinal system and through skin. In two cases involving individuals exposed to the vapour MIBK was found in the brain, liver, lung, vitreous fluid, kidney and blood. Experiments in guinea pigs show that MIBK is metabolised to 4-hydroxy-4-methyl-2-pentanone and 4-methyl-2-pentanol. Ketones are generally excreted rapidly in expired air. Small amounts of MIBK are also excreted in the urine. Humans excreted less than 0.1% of the dose as unmetabolised MIBK in the urine within the first 3 hours post exposure. Serum half-life in guinea pigs is about 55 minutes with a clearance time of 6 hours In animal studies, the acute systemic toxicity of MIBK, via the oral and inhalation routes of exposure, is low. In a 90-day gavage study on rats, a no-observed-effect level (NOEL) of 50 mg/kg per day was found. In 90-day inhalation studies on rats and mice, concentrations of up to 4100 mg/m3 (1000 ppm) did not result in significant toxicity, though compound-related reversible morphological changes were reported in the liver and kidney. Evidence of central nervous system depression was seen in animals exposed to a level of 4100 mg/m3 (1000 ppm). In a number of studies, exposure to MIBK concentrations as low as 1025 mg/m3 (250 ppm) resulted in an increase in liver size and induced hepatic microsomal metabolism. This may be responsible for the exacerbation of haloalkane toxicity and for the potentiation of the neurotoxicity of *n*-hexane. MIBK was also found to potentiate the cholestatic effects of manganese given with, or without, bilirubin. In 90-day studies on mice, rats, dogs, and monkeys, only male rats developed hyaline droplets in the proximal tubules of the kidney. Effects on behaviour were reported in baboons exposed for 7 days to 205 mg/m3 (50 ppm). At a concentration of 4100 mg/m3 (1000 ppm), MIBK was not embryotoxic, foetotoxic, or teratogenic in rats or mice. Foetotoxicity was only observed at concentrations of MIBK that caused maternal toxicity. MIBK did not induce gene mutations in *in vitro* bacterial test systems with, or without, metabolic activation. Negative results were also obtained *in vitro* with, or without, metabolic activation, in tests for mitotic gene conversion in yeast, and for gene mutation in cultured mammalian cells. The results of *in vitro* assays for unscheduled DNA synthesis in primary rat hepatocytes and for structural chromosome damage in cultured rat liver cells were negative. An *in vivo* micronucleus test on mice was negative. These data indicate that MIBK is not genotoxic. No long-term or carcinogenicity studies are available. The toxicity of MIBK for aquatic organisms and microorganisms is low.
NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe] For Low Boiling Point Naphthas (LBPNs): **Acute toxicity:** LBPNs generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000

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mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure
Most LBPNs are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices.
Sensitisation:
LBPNs do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies
Repeat dose toxicity:
The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPN substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNs in the group. Most of the studies were carried out by the inhalation route of exposure.
Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNs, were considered species- and sex-specific These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.
Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNs. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3
No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats
No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNs and very few non-cancer chronic toxicity studies were identified for other LBPNs. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3
A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.
Genotoxicity:
Although few genotoxicity studies were identified for the site-restricted LBPNs, the genotoxicity of several other LBPN substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results.
For in vivo genotoxicity tests, LBPNs exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells.
For in vitro genotoxicity studies, LBPNs were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNs exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay . Mixed results were observed for UDS and the mouse lymphoma assay.
While the majority of in vivo genotoxicity results for LBPN substances are negative, the potential for genotoxicity of LBPNs as a group cannot be discounted based on the mixed in vitro genotoxicity results.
Carcinogenicity:
Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b).
Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect
s of human exposure to LBPN substances.
No inhalation studies assessing the carcinogenicity of the site-restricted LBPNs were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m3 of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously.Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPN group.
Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPN substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPN, as a Group 2B carcinogen (possibly carcinogenic to humans) and “occupational exposures in petroleum refining” as Group 2A carcinogens (probably carcinogenic to humans).
Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNs. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals’ lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light
straight-run naphtha and naphtha Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha
or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.
Reproductive/ Developmental toxicity:
No reproductive or developmental toxicity was observed for the majority of LBPN substances evaluated. Most of these studies were carried out by inhalation exposure in rodents.
NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m3 (CAS RN 8052-41-3) to 27 687 mg/m3 (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m3 to 27 059 mg/m3 for the site-restricted light catalytic cracked and

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full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m3, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted . For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13 .
For most LBPNs, no treatment-related developmental effects were observed by the different routes of exposure However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m3 delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.
Low Boiling Point Naphthas [Site-Restricted]
For trimethylbenzenes:
Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion . After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates . The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid . The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.
Acute Toxicity Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis . High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness . The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg) . Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels . No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days
Neurotoxicity 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes
Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.
Subchronic/Chronic Toxicity Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene
Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia .
Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella tymphimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation.
Developmental/Reproductive Toxicity: A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established Developmental toxicity, including possible develop- mental neurotoxicity, was evident in rats in a 3-generation reproductive study
No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation
For C9 aromatics (typically trimethylbenzenes - TMBs)
Acute Toxicity
Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50's range from 6,000 to 10,000 mg/m 3 for C9 aromatic naphtha and 18,000 to 24,000 mg/m3 for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.
Irritation and Sensitization
Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.
Repeated Dose Toxicity
Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m3). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m3, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m3) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.
The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m3. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m3). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m3 (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m3 for respiratory irritation and 250 ppm or 1230 mg/m3 for systemic effects.
Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of

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	repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers. Mutagenicity In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m3) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category Reproductive and Developmental Toxicity Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m3 , respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex/group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex/group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21. Systemic Effects on Parental Generations: The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m3). Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m3). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation,, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m3), which excludes analysis of the highest concentration due to excessive mortality. Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m3) based on the body weights reductions observed in the F3 offspring. Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported
C.I. PIGMENT VIOLET 23	No carcinogenic effects observed during a 43 day test animal feeding study on Pigment Violet 23. [Manufacturer]
C.I. PIGMENT YELLOW 83	For diarylide (disazo) pigments (3,3'-dichlorobenzidine-containing): The substances in this category do not present a hazard for human health due to their low hazard profile. Adequate screening-level data are available to characterise the human health hazard for the purposes of the OECD Cooperative Chemicals Assessment Programme. Diarylide pigments are synthesized by bis-diazotizing diamino-diphenyl derivatives, mainly 3,3'-dichlorobenzidine (DCB), and coupling with acetoacetarylides or arylsubstituted pyrazolones Studies indicate that essentially there is no potential for uptake via the oral and dermal routes. However, following repeated oral exposure at high dose levels, there is some evidence that a very limited uptake of the compound (or its impurities) could occur, based on observations of staining of the mucosal surfaces of internal organs (although the possibility of contamination during necropsy cannot be excluded). In an oral reproductive developmental screening study, staining of the pups could indicate a potential for limited placental transfer, again at a high dose level. Given that the Pigment Yellows are essentially not absorbed into the body,metabolism is not relevant. However, the presence of very low levels of 3,3'-dichlorobenzidine has been demonstrated in two studies using very sensitive techniques following oral administration of some yellow pigment compounds. It seems likely that this is due to the presence of a mono-azo impurity in some of the yellow pigment parent compounds, which is absorbed and subsequently metabolised. No DCB was found in the urine of experimental animals after exposure orally or via the lungs in long term studies. Following ingestion, the vast majority of the pigments are excreted unchanged in the faeces. Many diarylide pigments are derived from DCB. Therefore, the diarylide pigments on DCB basis have been tested toxicologically very extensively. Diarylide pigments with their LD50 values above 2 000 mg/kg show no acute toxicity according to the EU classification criteria. They are not irritating to the skin or mucous membranes. For acute dermal toxicity a single LD50 of >3,000 mg/kg bw is available for Pigment Yellow 13. No deaths or clinical signs of toxicity were observed following oral or dermal exposure. The inhalation LC50 available is >4,448 mg/m3 for Pigment Yellow 13. Tachypnoea, dyspnoea, exophthalmos, ruffled fur and curved or ventral body position were observed, although all animals recovered and no gross abnormalities were observed at necropsy. Based on the available data the pigments have a minimal to slight potential for eye irritation. There is no indication that they are sensitisers No adverse effects were seen after 4-7 weeks oral administration of Pigment Yellow 12 at 1000 mg/kg/day (NOAEL), the highest dose tested in a well conducted and reported test of repeated dose toxicity study. Furthermore, in the cases of Pigment Yellow 12 and 83, no toxicologically significant effects were observed in a range of chronic toxicity studies of lesser quality (in terms of reporting) in rats and mice at doses up to 6500 mg/kg/day. Based on the kinetics of the three pigments and the chemical similarities, it can be concluded that these findings can be

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	extrapolated to most if not all diarylide pigments. For the inhalation route the effects seen are related to the deposition of dust particles in the lungs, leading to Pigment Yellow 13 related effects even at the lowest exposure concentration of 54 mg/m3 (local LOAEL). Systemically no effects were observed at the highest concentration tested, 410 mg/m3 (systemic NOAEL). All three pigments are not genotoxic in bacterial tests. Pigment Yellow 12 did not induce clastogenic effects in mammalian cells. Based on the chemical similarities between the three pigments, it is predicted that all three Yellow Pigments will not induce chromosomal changes in mammalian cells. There are no in vitro data to suggest that the pigments are genotoxic in vivo. No increased tumour incidence after treatment with Pigment Yellow 12 and 83 were observed in several long-term studies in rats and mice (NOAEL (rat) > 630 mg/kg; NOAEL (mouse) > 1,960 mg/kg). Based on chemical similarity it can be concluded that the pigments are not carcinogenic. It can be concluded that Pigment Yellow 12 does not have any adverse effects on reproductive parameters. There was no evidence of teratogenicity. The NOAEL for maternal and reproductive toxicity is >1,000 mg/kg bw. Supporting evidence is also available from the fact that no changes on the reproductive organs were observed in the studies of repeat dose toxicity and carcinogenicity study with Pigment Yellow 83. In view of the structural similarities and similar kinetics no effects on reproduction or development are expected from pigments of this class. In studies of the bioavailability of several representatives of this group of pigments, no carcinogenic cleavage product was released in detectable amounts after oral, inhalative or intratracheal application on rats. One further study of the bioavailability of DCB (DCB haemoglobin adduct) has been performed with the diarylide pigments C.I. Pigment Yellow 13 and C.I. Pigment Yellow 17. In this study, no release of carcinogenic DCB from the pigments has been detected. This indicates the absence of metabolism to DCB under the test conditions. In summary then, according to the known studies, diarylide pigments do not represent any health risk although risks might attach to contaminants introduced during synthesis. Colourants for Food Contact Plastics - Aspects of Product Safety; Responsible Care initiative of the European Chemical Industry Council. For 3,3'-dichlorobenzidine: Various tumours developed after oral or subcutaneous administration of 3,3'-dichlorobenzidine to mice, rats, hamsters and dogs. Tumours have not yet been identified in persons exposed to the substance alone. The substance can be absorbed through the skin in dangerous quantities. Increases in temperature and relative humidity promote dermal absorption. Upper respiratory infection and sore throat were listed among several principal reasons for visits to a company's medical clinic by workers handling 3,3'-dichlorobenzidine dihydrochloride However, there is no conclusive evidence that these effects were due to inhalation of 3,3'-dichlorobenzidine dihydrochloride. No adverse health effects were observed in male rats exposed by inhalation to 3,3'- dichlorobenzidine free base (23,700 mg/m3) 2 hours per day for 7 days . In another study, 10 rats were exposed to an unspecified concentration of 3,3'-dichlorobenzidine dihydrochloride dust particles for 1 hour and then observed for 14 days. Slight-to-moderate pulmonary congestion and one pulmonary abscess were observed upon necropsy . The effects observed in the study using the ionized (hydrochloride) form of 3,3'-dichlorobenzidine may have been due to the irritative properties of hydrochloric acid released from the salt in combination with particulate toxicity. Gastrointestinal upset was one of the symptoms reported by employees who worked with 3,3'-dichlorobenzidine dihydrochloride. However, there is no conclusive evidence that the gastrointestinal effects, or other symptoms reported by employees, resulted specifically from inhalation of 3,3'-dichlorobenzidine dihydrochloride. The only relevant information regarding neurological effects in humans exposed to 3,3'-dichlorobenzidine was found in an early study which reported that headache and dizziness were among several principal reasons why employees working with 3,3'-dichlorobenzidine in a chemical manufacturing plant visited the company medical clinic. However, there is no conclusive evidence that these symptoms were caused specifically by 3,3'-dichlorobenzidine since there was exposure to other chemicals as well. In a 3,3'-dichlorobenzidine carcinogenicity study, 1 of 6 dogs exhibited convulsions after 21, 28, or 42 months of oral treatment with 10.4 mg/kg/day over a period of 3.5 years **Carcinogenicity:** Several epidemiological studies have investigated cancer incidences among workers occupationally exposed to 3,3'-dichlorobenzidine . Exposure may have been by both inhalation and dermal routes. Due, in part, to structure-activity considerations, epidemiological studies of potential cancer effects of occupational exposure to 3,3'-dichlorobenzidine have been particularly concerned with bladder tumors, since 3,3'-dichlorobenzidine is structurally similar to benzidine, a chemical which is known to be a human bladder carcinogen. No bladder tumors were found in a group of 35 workers who handled only 3,3'-dichlorobenzidine; in the same dyestuff plant, bladder tumors occurred in 3 out of 14 workers exposed to both benzidine and 3,3'-dichlorobenzidine. The investigator reported a total exposure time of 68,505 hours, equivalent to nearly 140 full-time working years. No cases of bladder tumors were found in an epidemiology study of 259 workers exposed to dry and sernidry 3,3'-dichlorobenzidine base and hydrochloride. Workers were exposed to an average of less than 16 years each to 3,3'-dichlorobenzidine, which means that an adequate exposure duration and/or the latent period following exposure may not have been reached for tumor expression. In a retrospective epidemiological study of workers employed in a dye and pigment manufacturing plant that used 3,3'-dichlorobenzidine as chemical precursor, no bladder tumors were observed in a cohort of 207 workers, most of whom had been exposed for up to 15 years. Limitations of this study included using data from a very small and incomplete sample of workers; focusing solely on the occurrence of bladder tumors; and using data that may have been misleading and, at times, apparently inaccurate. A statistically significant increased incidence of hepatomas was observed in male ICR/JCL mice exposed to 0.1% 3,3'-dichlorobenzidine in the diet (170 mg/kg/day) at 6 months (8 of 8 treated as opposed to 0 of 5 controls) and 12 months (18 of 18 treated as opposed to 2 of 2 1 controls). Hepatic tumors were observed in 4/l 8 strain D mice exposed to 11.2-l 1.9 mg 3,3'-dichlorobenzidine/kg/day in the diet for 10 months No bladder carcinomas were observed in rats exposed to 0.03% 3,3'-dichlorobenzidine in the diet (27 mg/kg/day) for 4 or 40 weeks , nor were any mammary tumors observed in rats administered approximately 49 mg 3,3'-dichlorobenzidine dihydrochloride/kg/day by gavage once every 3 days over a 30-day period and sacrificed 8 months later. In a study in which rats were exposed to 10-20 mg 3,3'-dichlorobenzidine per day (120 mg/kg/day) in feed 6 days per week for 12 months, tumors were observed at a variety of sites, including the Zymbal gland (7 of 29 animals), mammary gland (7/29), bladder (3/29), hematopoietic system (3/29), skin (3/29), ileum (2/29), connective tissue (2/29), salivary gland (2/29), liver (l/29), and thyroid (l/29). In another rat study, 3,3'-dichlorobenzidine was administered to 50 male (70 mg/kg/day) and 50 female (80 mg/kg/day) Sprague-Dawley rats, in a standard diet for up to 16 months . In rats fed 3,3'-dichlorobenzidine in the diet for a total of 349 days (females) and 353 days (males), histopathological evaluations revealed mammary adenocarcinoma (16% incidence), malignant lymphoma (14%) granulocytic leukemia (20%), carcinoma of the Zymbal gland (18%) in males, and mammary adenocarcinoma (59%) in females. The authors noted that most of these tumors appeared to arise in the bone marrow and haematopoietic foci in the spleen and liver with subsequent metastasis to other organs. **Haematological Effects**. Although haematological effects may not be sensitive indicators for 3,3'-dichlorobenzidine toxicity, haemoglobin adducts have been detected in female Wistar rats orally administered single 127 or 253 mg/kg doses of 3,3'-dichlorobenzidine or with repeated doses between 0.3 and 5.8 mg/kg/day . It was suggested that metabolically formed nitroso derivatives and the formation of a sulfinic acid amide with cysteine residues in haemoglobin may be the mechanism of adduct formation. **Hepatic Effects**. Limited animal evidence suggests that chronic-duration oral exposure to 3,3'-dichlorobenzidine results in mild-to-moderate liver injury. **Genotoxic effects:** Genotoxic effects have been reported in animals treated with 3,3'-dichlorobenzidine. A single dose of 3,3'-dichlorobenzidine (1,000 mg/kg) administered to male and pregnant female mice induced micronuclei in polychromatic erythrocytes in the bone marrow of the males and in the liver of the foetuses, but not in bone marrow of the dams. In another study, an increase in unscheduled deoxyribonucleic acid synthesis (UDS) was observed in cultured liver cells from male mice previously pretreated orally with single doses of . 500 mg/kg 3,3'-dichlorobenzidine; no response was observed at a dose of .200 mg/kg. 3,3'-Dichlorobenzidine was also shown to bind extensively to tissue deoxyribonucleic acid (DNA) in rats and mice
N-BUTANOL	for n-butanol **Acute toxicity:** n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC0 of 8000 ppm (24000 mg/m3) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately

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	irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BAs localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser. The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed. **Repeat dose toxicity:** An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAc) to BA. Hydrolysis of BAc in blood and brain was estimated to be 99 percent complete within 2.7 minutes (elimination t1/2 = 0.41 minute). Thus, organisms exposed to BAc can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAc can be used as supplemental, surrogate data to provide information on the toxicity of BA. A thirteen-week, subchronic exposure to BAc, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m3) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOAEL) of 500 ppm (2395 mg/m3) was reported for systemic effects in rats, and a NOAEL of 3000 ppm (14370 mg/m3) was reported for post exposure neurotoxicity in rats. **Reproductive toxicity:** Several studies indicate that BA is not a reproductive toxicant. Female rats exposed to 6000 ppm (18000 mg/m3) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m3) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity. **Developmental toxicity:** BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m3) throughout gestation. **Genotoxicity:** An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic. **Carcinogenicity:** Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.
DIACETONE ALCOHOL	Inhalation (human) TCLo: 400 ppm resp.effect For diacetone alcohol (DAA): **Acute toxicity:** Oral LD50 of diacetone alcohol is more than 4,000 mg/kg. The lowest reported toxic concentration for human is 0.475 g/m3, although the reliability is not sure because of too old study and no detailed information. This chemical is moderately irritating to skin and irritating to eyes but there is no available data for sensitisation. **Repeat dose toxicity:** In oral rat study by an OECD combined repeated dose and reproductive/developmental toxicity screening test [TG 422] at doses of 0, 30, 100, 300 and 1,000 mg/kg/day for at least 44 days, decreased locomotor activity and less response to stimulation by knocking sounds or palpation were observed in males and females of the 300 and 1,000 mg/kg groups. Histopathological examination revealed increases of deposition of hyaline droplets in the proximal tubular epithelium at doses of 100 mg/kg or more, basophilic tubules at doses of 300 and 1,000 mg/kg and dilatation of the distal tubules at dose of 1,000 mg/kg in male kidneys. Slight but no significant increases of dilated distal tubules and fatty degeneration of the proximal tubular epithelium were observed in female kidneys at doses of 300 and 1,000 mg/kg. Furthermore, hepatocellular hypertrophy was evident in both sexes of the 1,000 mg/kg group, and vacuolization of the cells of the zona fasciculata in the adrenals of males receiving 1,000 mg/kg. Based on renal toxicity in male, NOAEL by oral administration was considered 30 mg/kg/day. An inhalation rat study conducted for 6 hr/day, 6 day/week, 6 weeks at doses of 0.232, 1.035 and 4.494 g/m3 demonstrated the histologic changes in the proximal tubules of the kidneys toxicity in males at the highest dose. As only liver weight was increased at mid dose, NOAEL was considered at 1.035 g/m3 for 6 hr/day, 6 day/week. The daily intake is roughly calculated as 156 mg/kg/day. **Reproductive and developmental toxicity:** In reproductive /developmental toxicity study [OECD TG 422], there were no statistically significant adverse effects noted at any dose. However, the composite of data at the 1,000 mg/kg suggest there may be chemically related adverse effects such as decreased tendency in the fertility index, number of implantations, implantation index and birth index with two mothers ability not to normally carry the litter. Therefore, a NOAEL for reproductive/developmental toxicity was considered to be 300 mg/kg/day. **Genotoxicity:** Evidence of malformations was not observed at any dose. This chemical was not genotoxic in bacterial test and chromosomal aberration test *in vitro* [OECD TG 471 & 473].
N-BUTYL ACETATE & XYLENE & N-BUTANOL & DIACETONE ALCOHOL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
N-BUTYL ACETATE & METHYL ETHYL KETONE & XYLENE & N-BUTANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
4-CHLOROBENZOTRIFLUORIDE & TITANIUM DIOXIDE & SILICA PRECIPITATED, CRYSTALLINE FREE & METHYL ETHYL KETONE & METHYL ISOBUTYL KETONE & FERRIC OXIDE & GRAPHITE & MICA & N-BUTANOL	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
TITANIUM DIOXIDE & GRAPHITE & CARBON BLACK & ALUMINIUM POWDER COATED & MICA & DIACETONE ALCOHOL	No significant acute toxicological data identified in literature search.
TITANIUM DIOXIDE & ACETONE & ETHYL-3-ETHOXYPROPIONATE & METHYL ISOBUTYL KETONE & DIACETONE ALCOHOL	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.
TITANIUM DIOXIDE & METHYL ISOBUTYL KETONE & CARBON BLACK	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
NAPHTHA PETROLEUM, HEAVY, HYDROTREATED & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & SOLVENT NAPHTHA PETROLEUM, HEAVY	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in

Continued...

AROMATIC	the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. for petroleum: Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic. This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents **Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans. **Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays. **Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed. **Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in 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

ColorSpec Tinter (Colorspec No Mix_E Basecoat)

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

n-butyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	-17-19mg/L	4
EC50	48	Crustacea	32mg/L	2
EC50	72	Algae or other aquatic plants	246mg/L	2
EC0	192	Algae or other aquatic plants	=21mg/L	1
NOEC	504	Crustacea	23.2mg/L	2

4-chlorobenzotrifluoride

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	3mg/L	2
EC50	48	Crustacea	=3.68mg/L	1
EC50	72	Algae or other aquatic plants	>0.41mg/L	2
NOEC	504	Crustacea	=0.03mg/L	1

titanium dioxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	-1.85-3.06mg/L	4
EC50	48	Crustacea	1.9mg/L	2
EC50	72	Algae or other aquatic plants	-3.75-7.58mg/L	4
BCF	24	Crustacea	0.66mg/L	4
NOEC	552	Not Available	0.01-mg/L	4

propylene glycol monomethyl ether acetate, alpha-isomer

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	373mg/L	2
EC50	72	Algae or other aquatic plants	>1000mg/L	2
NOEC	336	Fish	47.5mg/L	2

Continued...

silica precipitated, crystalline free

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	1033.016mg/L	2

methyl ethyl ketone

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>400mg/L	4
EC50	48	Crustacea	308mg/L	2
EC50	96	Algae or other aquatic plants	>500-mg/L	4
EC0	48	Crustacea	136mg/L	2
NOEC	48	Crustacea	68mg/L	2

xylene

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	0.0013404-mg/L	4
EC50	48	Crustacea	1.8mg/L	2
EC50	72	Algae or other aquatic plants	3.2mg/L	2
NOEL	72	Not Available	0.01-mg/L	4

acetone

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>100mg/L	4
EC50	48	Crustacea	6098.4mg/L	5
EC50	96	Algae or other aquatic plants	-9.873-27.684mg/L	4
NOEC	96	Not Available	<0.000000005-=mg/L	4

ethyl-3-ethoxypropionate

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	45.3mg/L	2
EC50	48	Crustacea	>95mg/L	1
EC50	72	Algae or other aquatic plants	>114.86mg/L	2
NOEC	48	Crustacea	=9.5mg/L	1

methyl isobutyl ketone

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>179mg/L	2
EC50	48	Crustacea	=170mg/L	1
EC50	96	Algae or other aquatic plants	=400mg/L	1
NOEC	Not coded	Crustacea	-7.8-39mg/L	4

naphtha petroleum, heavy, hydrotreated

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	4.1mg/L	2
EC50	48	Crustacea	4.5mg/L	2
EC50	72	Algae or other aquatic plants	3.1mg/L	2
NOEL	72	Algae or other aquatic plants	0.1mg/L	2

naphtha petroleum, light aromatic solvent

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	4.1mg/L	2
EC50	48	Crustacea	3.2mg/L	2
EC50	72	Algae or other aquatic plants	3.1mg/L	2
NOEL	72	Algae or other aquatic plants	0.1mg/L	2

ferric oxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	0.05mg/L	2
EC50	48	Crustacea	>100mg/L	2
EC50	72	Algae or other aquatic plants	18mg/L	2
NOEC	504	Fish	0.52mg/L	2

graphite

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	>100mg/L	2
EC50	72	Algae or other aquatic plants	>100mg/L	2
NOEC	72	Algae or other aquatic plants	>=100mg/L	2

carbon black

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	-33.076-41.968mg/L	4

Continued...

	EC50	72	Algae or other aquatic plants	>0.2mg/L	2
	EC10	72	Algae or other aquatic plants	>10000mg/L	2
	NOEC	24	Not Available	0.05mg/L	4

aluminium powder coated					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	0.078242mg/L	2
	EC50	48	Crustacea	0.7364mg/L	2
	EC50	96	Algae or other aquatic plants	0.0054mg/L	2
	BCF	360	Not Available	9mg/L	4
	NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2

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

solvent naphtha petroleum, heavy aromatic					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	0.58mg/L	2
	EC50	48	Crustacea	0.76mg/L	2
	EC50	72	Algae or other aquatic plants	0.79mg/L	2
	NOEC	96	Algae or other aquatic plants	0.12mg/L	2

C.I. Pigment Violet 23					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>100mg/L	2
	EC0	48	Crustacea	>=100mg/L	2
	NOEL	504	Crustacea	2.5mg/L	2

C.I. Pigment Yellow 83					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	>0.1mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>100mg/L	2
	NOEC	24	Fish	>=0.1mg/L	2

n-butanol					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	-100-500mg/L	4
	EC50	48	Crustacea	>500mg/L	1
	EC50	96	Algae or other aquatic plants	225mg/L	2
	BCF	24	Fish	921-mg/L	4
	EC10	168	Algae or other aquatic plants	<20-mg/L	4
	NOEC	504	Crustacea	4.1mg/L	2

diacetone alcohol					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>1000mg/L	2
	EC50	72	Algae or other aquatic plants	>1000mg/L	2
	NOEC	504	Crustacea	100mg/L	2

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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
n-butyl acetate	LOW	LOW
4-chlorobenzotrifluoride	HIGH	HIGH
titanium dioxide	HIGH	HIGH
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
silica precipitated, crystalline free	LOW	LOW
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
ethyl-3-ethoxypropionate	LOW	LOW
methyl isobutyl ketone	HIGH (Half-life = 7001 days)	LOW (Half-life = 1.9 days)
C.I. Pigment Yellow 83	HIGH	HIGH
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
diacetone alcohol	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
n-butyl acetate	LOW (BCF = 14)
4-chlorobenzotrifluoride	LOW (BCF = 202)
titanium dioxide	LOW (BCF = 10)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
silica precipitated, crystalline free	LOW (LogKOW = 0.5294)
methyl ethyl ketone	LOW (LogKOW = 0.29)
xylene	MEDIUM (BCF = 740)
acetone	LOW (BCF = 0.69)
ethyl-3-ethoxypropionate	LOW (LogKOW = 1.0809)
methyl isobutyl ketone	LOW (LogKOW = 1.31)
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)
C.I. Pigment Yellow 83	LOW (LogKOW = 8.6648)
n-butanol	LOW (BCF = 0.64)
diacetone alcohol	LOW (LogKOW = -0.3376)

Mobility in soil

Ingredient	Mobility
n-butyl acetate	LOW (KOC = 20.86)
4-chlorobenzotrifluoride	LOW (KOC = 1912)
titanium dioxide	LOW (KOC = 23.74)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
silica precipitated, crystalline free	LOW (KOC = 23.74)
methyl ethyl ketone	MEDIUM (KOC = 3.827)
acetone	HIGH (KOC = 1.981)
ethyl-3-ethoxypropionate	LOW (KOC = 10)
methyl isobutyl ketone	LOW (KOC = 10.91)
C.I. Pigment Yellow 83	LOW (KOC = 1126000)
n-butanol	MEDIUM (KOC = 2.443)
diacetone alcohol	HIGH (KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ **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.

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SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3YE

Land transport (ADG)

UN number	1263
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Transport hazard class(es)	Class: 3 Subrisk: Not Applicable
Packing group	II
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 163 367 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

UN number	1263
UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base)
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Packing group	II
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A3 A72 A192 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L Passenger and Cargo Packing Instructions: 353 Passenger and Cargo Maximum Qty / Pack: 5 L Passenger and Cargo Limited Quantity Packing Instructions: Y341 Passenger and Cargo Limited Maximum Qty / Pack: 1 L

Sea transport (IMDG-Code / GGVSee)

UN number	1263
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk: Not Applicable
Packing group	II
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-E , S-E Special provisions: 163 367 Limited Quantities: 5 L

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

Not Applicable

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

Product name	Group

Continued...

Product name	Group
n-butyl acetate	Not Available
4-chlorobenzotrifluoride	Not Available
titanium dioxide	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
silica precipitated, crystalline free	Not Available
methyl ethyl ketone	Not Available
xylene	Not Available
acetone	Not Available
ethyl-3-ethoxypropionate	Not Available
methyl isobutyl ketone	Not Available
naphtha petroleum, heavy, hydrotreated	Not Available
naphtha petroleum, light aromatic solvent	Not Available
ferric oxide	Not Available
graphite	Not Available
carbon black	Not Available
aluminium powder coated	Not Available
mica	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
C.I. Pigment Violet 23	Not Available
C.I. Pigment Yellow 83	Not Available
n-butanol	Not Available
diacetone alcohol	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
n-butyl acetate	Not Available
4-chlorobenzotrifluoride	Not Available
titanium dioxide	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
silica precipitated, crystalline free	Not Available
methyl ethyl ketone	Not Available
xylene	Not Available
acetone	Not Available
ethyl-3-ethoxypropionate	Not Available
methyl isobutyl ketone	Not Available
naphtha petroleum, heavy, hydrotreated	Not Available
naphtha petroleum, light aromatic solvent	Not Available
ferric oxide	Not Available
graphite	Not Available
carbon black	Not Available
aluminium powder coated	Not Available
mica	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
C.I. Pigment Violet 23	Not Available
C.I. Pigment Yellow 83	Not Available
n-butanol	Not Available
diacetone alcohol	Not Available

SECTION 15 Regulatory information

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

n-butyl acetate is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

4-chlorobenzotrifluoride is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
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

titanium dioxide 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)

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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

silica precipitated, crystalline free is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

methyl ethyl ketone 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)

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

acetone 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)

ethyl-3-ethoxypropionate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

methyl isobutyl ketone 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

naphtha petroleum, heavy, hydrotreated 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

naphtha petroleum, light aromatic solvent 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

ferric oxide is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
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

graphite is found on the following regulatory lists

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)

Continued...

aluminium powder coated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

mica is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

solvent naphtha petroleum, heavy aromatic is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

C.I. Pigment Violet 23 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment Yellow 83 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 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

n-butanol 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)

diacetone alcohol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (n-butyl acetate; 4-chlorobenzotrifluoride; propylene glycol monomethyl ether acetate, alpha-isomer; silica precipitated, crystalline free; methyl ethyl ketone; xylene; acetone; ethyl-3-ethoxypropionate; methyl isobutyl ketone; naphtha petroleum, heavy, hydrotreated; naphtha petroleum, light aromatic solvent; ferric oxide; graphite; carbon black; aluminium powder coated; mica; solvent naphtha petroleum, heavy aromatic; C.I. Pigment Violet 23; C.I. Pigment Yellow 83; n-butanol; diacetone alcohol)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (silica precipitated, crystalline free)
Japan - ENCS	No (silica precipitated, crystalline free; naphtha petroleum, heavy, hydrotreated; graphite; aluminium powder coated; mica; solvent naphtha petroleum, heavy aromatic)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (silica precipitated, crystalline free; mica)
Taiwan - TCSI	Yes
Mexico - INSQ	No (4-chlorobenzotrifluoride; C.I. Pigment Yellow 83)
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	09/02/2021
Initial Date	03/01/2013

SDS Version Summary

Version	Issue Date	Sections Updated
9.1.1.1	03/09/2020	Classification change due to full database hazard calculation/update.
10.1.1.1	09/02/2021	Classification, Synonyms

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.

Continued...

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index