ColorSpec Tinter (Colorspec No Mix_E Basecoat)

Motor Active

Chemwatch Hazard Alert Code: 3

Chemwatch: **5678-14**
Version No: **3.2**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **02/05/2024**
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L.GHS.AUS.EN.E

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, E-02 Blue, E-03 Bright Blue, E-04 Bright Gold, E-05 Bright Red, E-06 Custard, E-07 Copper, E-08 Vivid Magenta, E-09 Raven Black, E-10 Deep Black, E-11 Cobalt Blue, E-12 Maroon,E-13 Deep Maroon, E-14 Garnet, E-15 Green, E-16 Green Blue, E-17 Green Gold, E-18 Grey Black, E-19 Empress Black, E-20 Light Red Oxide, E-21 Lime, E-22 Magenta, E-23 Midnight Blue, E-24 Red Gold, E-25 Red Maroon, E-26 Vivid Orange, E-27 Ruby, E-28 Turquoise, E-29 Midnight Black, E-30 Violet, E-31 White, E-32 Yellow Gold, E-33 Yellow Ochre, E-34 Special Violet, E-35 Port Wine Red,E-36 Deep Blue, E-37 Special Deep Black, E-38 Special Red Maroon, E-39 HS Special Red, E-40 Royal Blue, E-41 Reduced Black, E-42 Silk Silver, E-43 Silk Russet, E-44 Silk Gold, E-45 Silk Blue, E-46 Silk Red, E-47 Silk Green, E-48 Silk Copper, E-51 Red Yellow, E-52 Topaz, E-53 Organic Orange, 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, E-60 Stabiliser Additive, E-61 Effect White, E-62 HS Special White, E-63 HS Special Yellow, E-65 Fine Metallic, E-66 Medium Metallic,E-67 Coarse Metallic, E-68 Extra Fine Silver, E-69 Fine Silver, E-70 Silver, E-71 Medium Silver, E-72 Coarse Silver, E-74 Coarse Aluminium, E-75 Extra Coarse Aluminium, E-77 Fine White Pearl, E-78 White Sparkle Pearl, E-79 Bright Silver Pearl, E-80 Yellow Pearl, E-82 Fine Yellow Gold Pear,l E-83 Orange Pearl, E-86 Copper Pearl, E-87 Bright Russet Pearl, E-88 Fine Russet Pearl, E-89 Blue Russet Pearl, E-90 Red Blue Pearl, E-91 Fine Blue Pearl, E-92 Green Blue Pearl, E-93 Fine Green Pearl, E-95 Blue Green Pearl, E-96 Red Pearl, E-97 Fine Silver Pearl, E-98 Fine Violet Pearl, E-99 Metallic Raiser, E-101 Red Candy, E-102 Brandy Wine Candy, E-103 Yellow Candy, E-104 Green Candy, TU50ML Touch-Up Bottle 50ml, TL250ML Tinters 250ml, TL1L Tinters 1 Litre, TL2L Tinters 2 Litre, TL500 Tinters 500ml,CSA1000 Paint Pen.
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base)
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 manufacturer or 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	Not Available
Website	www.motoractive.com.au
Email	info@motoractive.com.au

Emergency telephone number

Association / Organisation	**MotorActive**
Emergency telephone number(s)	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
Other emergency telephone number(s)	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	1	
Body Contact	2	
Reactivity	1	
Chronic	3	

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

Poisons Schedule	S5
Classification [1]	Flammable Liquids Category 2, Acute Toxicity (Oral) Category 4, Aspiration Hazard Category 1, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Carcinogenicity Category 1B, Reproductive Toxicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Page 1 continued...

Label elements

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

Hazard statement(s)

H225	Highly flammable liquid and vapour.
H302	Harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H312	Harmful in contact with skin.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H350	May cause cancer.
H360D	May damage the unborn child.
H372	Causes damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.
AUH019	May form explosive peroxides.

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 and face protection.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting. If more than 15 mins from Doctor, INDUCE VOMITING (if conscious).
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
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.
P330	Rinse mouth.

Precautionary statement(s) Storage

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

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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
78-93-3	10-30	methyl ethyl ketone
108-65-6	10-30	propylene glycol monomethyl ether - mixture of isomers
13463-67-7	10-30	C.I. Pigment White 6
98-56-6	<10	4-chlorobenzotrifluoride
64742-95-6.	<10	naphtha petroleum, light aromatic solvent
763-69-9	<10	ethyl-3-ethoxypropionate
1309-37-1	<10	ferric oxide
5567-15-7	<10	C.I. Pigment Yellow 83
64742-94-5	<10	solvent naphtha petroleum, heavy aromatic
7429-90-5	<10	aluminium
12001-26-2	<10	mica
15680-42-9	<10	C.I. Pigment Yellow 129
1333-86-4	<10	carbon black
123-42-2	<10	diacetone alcohol
64742-48-9.	<10	naphtha petroleum, heavy, hydrotreated
7782-42-5	<10	graphite
100-41-4	<10	ethylbenzene
6358-30-1	<10	C.I. Pigment Violet 23
71-36-3	<1	n-butanol
872-50-4	<1	N-methyl-2-pyrrolidone
41556-26-7	<1	bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate
85408-46-4	<1	C.I. Solvent Yellow 88
80-62-6	<1	methyl methacrylate
107-98-2	<1	propylene glycol monomethyl ether - alpha isomer
Not Available	balance	Ingredients determined not to be hazardous

Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket.

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	▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives.
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	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ 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. ▸ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
Treat symptomatically.
For petroleum distillates
· In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
· Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
· Positive pressure ventilation may be necessary.
· Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
· After the initial episode,individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
· Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
· Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur.Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.
for copper intoxication:
▸ Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
▸ Administer egg white and other demulcents.
▸ Maintain electrolyte and fluid balances.
▸ Morphine or meperidine (Demerol) may be necessary for control of pain.
▸ If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
▸ Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
▸ If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.
▸ It is unlikely that methylene blue would be effective against the occassional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
▸ Institute measures for impending renal and hepatic failure.
[GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]
▸ A role for activated charcoals for emesis is, as yet, unproven.
▸ In severe poisoning CaNa2EDTA has been proposed.
[ELLENHORN & BARCELOUX: Medical Toxicology]
for simple esters:

BASIC TREATMENT

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

ADVANCED TREATMENT

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

EMERGENCY DEPARTMENT

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

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BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994
Periodic medical surveillance should be carried out on persons in occupations exposed to the manufacture or bulk handling of the product and this should include hepatic function tests and urinalysis examination. [ILO Encyclopaedia]

SECTION 5 Firefighting measures

Extinguishing media

At temperatures above 1500 C, carbon, graphite or graphene reacts with substances containing oxygen, including water and carbon dioxide. In case of intensely hot fires sand should be used to cover and isolate these materials.

- **DO NOT** use halogenated fire extinguishing agents.

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	Combustion products include: carbon dioxide (CO_2) hydrogen chloride phosgene nitrogen oxides (NOx) silicon dioxide (SiO_2) metal oxides other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions. When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles. A fire in bulk finely divided carbon may not be obviously visible unless the material is disturbed and sparks appear. A straw broom may be useful to produce the disturbance. Explosion and Ignition Behaviour of Carbon Black with Air Lower Limit for Explosion: 50 g/m3 (carbon black in air) Maximum Explosion Pressure: 10 bar Maximum Rate of Pressure Rise: 30-100 bar/sec Minimum Ignition Temperature: 315 deg. C. Ignition Energy: >1 kJ Glow Temperature: 500 deg. C. (approx.) Notes on Test Methods: Tests 1, 2 and 3 were conducted by Bergwerkeschaftliche Versuchstrecke, Dortmunde-Derne, using a 1 m3 vessel with two chemical igniters having an intensity of 5000 W.S. Tests 1 and 2 results are confirmed by information in the Handbook of Powder Technology, Vol. 4 (P. Field) In Test 4, a modified Godbert-Greenwald furnace was used. See U.S. Bureau of Mines, Report 5624, 1960, p.5, "Lab Equipment and Test Procedures". Test 5 used a 1 m3 vessel with chemical igniters of variable intensity. Test 6 was conducted in a laboratory oven. Active glowing appeared after 3 minutes exposure. (European Committee for Biological Effects of Carbon Black) (2/84) ‣ 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).
HAZCHEM	●3YE

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb small quantities with vermiculite or other absorbent material. ‣ Wipe up. ‣ Collect residues in a flammable waste container.
Major Spills	‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard.

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- 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

Graphite:
· is a good conductor of electricity; avoid contact with electrical circuitry.
· is a highly lubricious material and may present a slip hazard if spilled on pedestrian surfaces.
NOTE:
- Wet, activated carbon removes oxygen from the air thus producing a severe hazard to workers inside carbon vessels and in enclosed or confined spaces where activated carbons might accumulate.
- Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply.
- 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.

Contains low boiling substance:
Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.
- Check for bulging containers.
- Vent periodically
- Always release caps or seals slowly to ensure slow dissipation of vapours
- **DO NOT** allow clothing wet with material to stay in contact with skin

The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe
- **DO NOT** concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential.
- Any static discharge is also a source of hazard.
- Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina.
- Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage.
- Add inhibitor to any distillate as required.
- When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely.

The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example.
Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.
- A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date.
- The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date.
- Unopened containers received from the supplier should be safe to store for 18 months.
- Opened containers should not be stored for more than 12 months.
- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- **DO NOT** enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights, heat or ignition sources.
- When handling, **DO NOT** eat, drink or smoke.
- Vapour may ignite on pumping or pouring due to static electricity.
- **DO NOT** use plastic buckets.
- Earth and secure metal containers when dispensing or pouring product.
- Use spark-free tools when handling.
- Avoid contact with incompatible materials.
- Keep containers securely sealed.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- Use good occupational work practice.
- Observe manufacturer's storage and handling recommendations contained within this SDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.

Other information

- Store in original containers in approved flame-proof area.
- No smoking, naked lights, heat or ignition sources.
- **DO NOT** store in pits, depression, basement or areas where vapours may be trapped.
- Keep containers securely sealed.
- Store away from incompatible materials in a cool, dry well ventilated area.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storage and handling recommendations contained within this SDS.
- Tank storage: Tanks must be specifically designed for use with this product. Bulk storage tanks should be diked (bunded). Locate tanks away from heat and other sources of ignition. Cleaning, inspection and maintenance of storage tanks is a specialist operation, which requires the implementation of strict procedures and precautions.
- Keep in a cool place. Electrostatic charges will be generated during pumping. Electrostatic discharge may cause fire. Ensure electrical continuity by bonding and grounding (earthing) all equipment to reduce the risk. The vapours in the head space of the storage vessel may lie in the flammable/explosive range and hence may be flammable.

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	▸ For containers, or container linings use mild steel, stainless steel. Examples of suitable materials are: high density polyethylene (HDPE), polypropylene (PP), and Viton (FMK), which have been specifically tested for compatibility with this product. ▸ For container linings, use amine-adduct cured epoxy paint. ▸ For seals and gaskets use: graphite, PTFE, Viton A, Viton B. ▸ Unsuitable material: Some synthetic materials may be unsuitable for containers or container linings depending on the material specification and intended use. Examples of materials to avoid are: natural rubber (NR), nitrile rubber (NBR), ethylene propylene rubber (EPDM), polymethyl methacrylate (PMMA), polystyrene, polyvinyl chloride (PVC), polyisobutylene. However, some may be suitable for glove materials. ▸ Do not cut, drill, grind, weld or perform similar operations on or near containers. Containers, even those that have been emptied, can contain explosive vapours.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Glass container is suitable for laboratory quantities ▸ Packing as supplied by manufacturer. ▸ Plastic containers may only be used if approved for flammable liquid. ▸ Check that containers are clearly labelled and free from leaks. ▸ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▸ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▸ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▸ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▸ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▸ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	▸ Avoid 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	methyl ethyl ketone	Methyl ethyl ketone (MEK)	150 ppm / 445 mg/m3	890 mg/m3 / 300 ppm	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether - mixture of isomers	1-Methoxy-2-propanol acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether - mixture of isomers	Propylene glycol monomethyl ether	100 ppm / 369 mg/m3	553 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	C.I. Pigment White 6	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	ferric oxide	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric oxide	Rouge dust	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	aluminium	Aluminium (welding fumes) (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium, pyro powders (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	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	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	diacetone alcohol	Diacetone alcohol	50 ppm / 238 mg/m3	Not Available	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	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	ethylbenzene	Ethyl benzene	100 ppm / 434 mg/m3	543 mg/m3 / 125 ppm	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	N-methyl-2-pyrrolidone	1-Methyl-2-pyrrolidone	25 ppm / 103 mg/m3	309 mg/m3 / 75 ppm	Not Available	Not Available
Australia Exposure Standards	methyl methacrylate	Methyl methacrylate	50 ppm / 208 mg/m3	416 mg/m3 / 100 ppm	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether -	Propylene glycol monomethyl ether	100 ppm / 369 mg/m3	553 mg/m3 / 150 ppm	Not Available	Not Available

Continued...

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
	alpha isomer					

Ingredient	Original IDLH	Revised IDLH
n-butyl acetate	1,700 ppm	Not Available
methyl ethyl ketone	3,000 ppm	Not Available
propylene glycol monomethyl ether - mixture of isomers	Not Available	Not Available
C.I. Pigment White 6	5,000 mg/m3	Not Available
4-chlorobenzotrifluoride	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
ethyl-3-ethoxypropionate	Not Available	Not Available
ferric oxide	2,500 mg/m3	Not Available
C.I. Pigment Yellow 83	Not Available	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available	Not Available
aluminium	Not Available	Not Available
mica	1,500 mg/m3	Not Available
C.I. Pigment Yellow 129	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
diacetone alcohol	Not Available	Not Available
naphtha petroleum, heavy, hydrotreated	2,500 mg/m3	Not Available
graphite	1,250 mg/m3	Not Available
ethylbenzene	Not Available	Not Available
C.I. Pigment Violet 23	Not Available	Not Available
n-butanol	1,400 ppm	Not Available
N-methyl-2-pyrrolidone	Not Available	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available	Not Available
C.I. Solvent Yellow 88	Not Available	Not Available
methyl methacrylate	1,000 ppm	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available	Not Available

MATERIAL DATA

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.

Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air.
Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde]

Individual protection measures, such as personal protective equipment



Eye and face protection

- Safety glasses with side shields.

Continued...

	▸ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▸ 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].
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 **NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. For esters: ▸ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▸ 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:

Respiratory protection

Type AK-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.

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ColorSpec Tinter (Colorspec No Mix_E Basecoat)

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	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
SARANEX-23	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.

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	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-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

· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.
Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.
Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.
Filtration rate: Filters at least 99.95% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
· Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos
Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured highly flammable liquid with strong solvent like odour.

Physical state	Liquid	**Relative density (Water = 1)**	0.9-1.4
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	277
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	5714.3-8888.9
Initial boiling point and boiling range (°C)	79-165	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	-9 (OC)	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	13.8	**Surface Tension (dyn/cm or mN/m)**	Not Available

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Lower Explosive Limit (%)	0.7	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	10.5 @20C	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Presence of elevated temperatures. ▸ 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

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	There is sufficient evidence to classify this material as an aspiration hazard

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. The 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. Diacetone alcohol is primarily a narcotic and anticonvulsive. Inhalation exposure of mice rats, rabbits and cats at 2100 ppm for 1-3 hours produced somnolence after a period of restlessness and excitability and mucous membrane irritation. Symptoms of intoxication (narcosis) are rapidly decreasing respiration, marked decrease in blood pressure, relaxation of muscles and absence of conjunctival reflexes 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
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). 5
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. 511r6
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.

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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. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. 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 Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to establish a causal relationship between human exposure to the material and subsequent developmental toxic effects in the off-spring. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Many azo dyes have been found to be carcinogenic in laboratory animals, affecting the liver, urinary bladder and intestines. Specific toxicity effects in humans have not been established but some dyes are known to be mutagenic. The simplest azo dyes, which raise concern, have an exocyclic amino-group that is the key to any carcinogenicity for it is this group which undergoes biochemical N-oxidation and further reaction to reactive electrophiles. The DNA adducts formed by covalent binding through activated nitrogen have been identified. However not all azo compounds possess this activity and delicate alterations to structure vary the potential of carcinogenicity / acid, reduces or eliminates the effect. Complex azo dyes consisting of more than one azo (N=N) linkage may be metabolised to produce complexed carcinogenic aromatic amines such as benzidine Benzidine and its metabolic derivatives have been detected in the urine of workers exposed to Direct azo dyes. An epidemiological study of silk dyers and painters with multiple exposures to benzidine based and other dyes indicate a strong association with bladder cancer. Most organic azo dyes are potential skin sensitisers, the most important of which are para-phenylenediamine and its analogs. Water soluble azo dyes are more likely to cause clinical sensitisation than insoluble dyes. In addition to allergic eczematous contact dermatitis, color developing solutions have caused lichen planus like eruptions When administered in the diet, 3,3'-dichlorobenzidine induced hepatomas in male mice, increased the incidences of granulocytic leukemia and Zymbal gland carcinomas in male rats and mammary adenocarcinomas in rats of both sexes, induced transitional cell carcinomas of the urinary bladder in hamsters and female dogs and hepatocellular carcinomas in female dogs. Transplacental exposure increased the incidences of lymphoid leukemia in mice. In three retrospective epidemiological studies, no urinary bladder tumors were reported in men occupationally exposed to 3,3'-dichlorobenzidine Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats Which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer . beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production. Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure. When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity. Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction. There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs. Chromium(III) is considered an essential trace nutrient serving as a component of the "glucose tolerance factor" and a cofactor for insulin action. High concentrations of chromium are also found in RNA. Trivalent chromium is the most common form found in nature. Chronic inhalation of trivalent chromium compounds produces irritation of the bronchus and lungs, dystrophic changes to the liver and kidney, pulmonary oedema, and adverse effects on macrophages. Intratracheal administration of chromium(III) oxide, in rats, increased the incidence of sarcomas, and tumors and reticulum cell sarcomas of the lung. There is inadequate evidence of carcinogenicity of chromium(III) compounds in experimental animals and humans (IARC). Chronic exposure to hexavalent chromium compounds reportedly produces skin, eye and respiratory tract irritation, yellowing of the eyes and skin, allergic skin and respiratory reactions, diminished sense of smell and taste, blood disorders, liver and kidney damage, digestive disorders and lung damage. There is sufficient evidence of carcinogenicity of chromium(VI) compounds in experimental animals and humans to confirm these as Class 1 carcinogens (IARC).

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Exposure to chromium during chrome production and in the chrome pigment industry is associated with cancer of the respiratory tract. A slight increase in gastrointestinal cancer following exposure to chromium compounds has also been reported. The greatest risk is attributed to exposure to acid-soluble, water-insoluble hexavalent chromium which occurs in roasting and refining processes. Animal studies support the idea that the most potent carcinogenic compounds are the slightly soluble hexavalent compounds. The cells are more active in the uptake of the hexavalent forms compared to trivalent forms and this may explain the difference in occupational effect. It is the trivalent form, however, which is metabolically active and binds with nucleic acid within the cell suggesting that chromium mutagenesis first requires biotransformation of the hexavalent form by reduction.
Hexavalent chromes produce chronic ulceration of skin surfaces (quite independent of other hypersensitivity reactions exhibited by the skin). Water-soluble chromium(VI) compounds come close to the top of any published "hit list" of contact allergens (eczematogens) producing positive results in 4 to 10% of tested individuals. On the other hand only chromium(III) compounds can bind to high molecular weight carriers such as proteins to form a complete allergen (such as a hapten). Chromium(VI) compounds cannot. It is assumed that reduction must take place for such compounds to manifest any contact sensitivity. The apparent contradiction that chromium(VI) salts cause allergies to chromium(III) compounds but that allergy to chromium(III) compounds is difficult to demonstrate is accounted for by the different solubilities and skin penetration of these compounds. Water-soluble chromium(VI) salts penetrate the horny layer of the skin more readily than chromium(III) compounds which are bound by cross-linking in the horny layer ("tanning", as for leather) and therefore do not reach the cells involved in antigen processing.
The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compared to crystalline silicas and are considered to be nuisance dusts.
When heated to high temperature and a long time, amorphous silica can produce crystalline silica on cooling. Inhalation of dusts containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to develop. Discrepancies between various studies showing that fibrosis associated with chronic exposure to amorphous silica and those that do not may be explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is either weakly fibrogenic or nonfibrogenic and that fibrosis is due to contamination by crystalline silica content
Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.
Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.
After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.
At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.
Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.
Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.
Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).
Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [***Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995***]
the main target organs of aluminum are the central nervous system and bone. Aluminum binds with dietary phosphorus and impairs gastrointestinal absorption of phosphorus. The decreased phosphate body burden results in osteomalacia (softening of the bones due to defective bone mineralization) and rickets. Aluminum's neurotoxicity is believed to involve several mechanisms. Changes in cytoskeletal protein functions as a results of altered phosphorylation, proteolysis, transport, and synthesis are believed to be one cause. Aluminum may induce neurobehavioral effects by affecting permeability of the blood-brain barrier, cholinergic activity, signal transduction pathways, lipid peroxidation, and impair neuronal glutamate nitric oxide-cyclic GMP pathway, as well as interfere with metabolism of essential trace elements because of similar coordination chemistries and consequent competitive interactions. It has been suggested that aluminum's interaction with estrogen receptors , but studies have not been able to establish a clear link between aluminum and increased risk of breast cancer). Certain aluminum salts induce immune responses by activating inflammasomes.
Prolonged or repeated inhalation of dust may result in pneumoconiosis (lung disease caused by inhalation dust).
Graphite workers have reported symptoms of headaches, coughing, depression, low appetite, dyspnoea (difficult breathing) and black sputum.
A number of studies indicate that graphitosis is a progressive and disabling disease and that the presence of crystalline silica and some silicates as graphite impurities have a pronounced synergistic effect.
Workers suffering from graphite pneumoconiosis have generally worked in the industry for long periods, i.e. 10 years or more, although some cases have been reported after as little as four years.
Data indicate the higher the crystalline silica content of graphite the greater is the severity of the pneumoconiosis.

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Pre-employment and periodic examinations should be directed towards detecting significant respiratory disease through chest X-rays and pulmonary function tests
Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.
Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms.
Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:
No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar
naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.
Long-term exposure of mine workers to vermiculite (mica) dust showed no health hazards related to vermiculite with less than 1% silica and no asbestos. There is no evidence of mesothelioma caused by vermiculite. Continuous exposure, for several years, may produce fibrotic pneumoconiosis (lung scarring) which is readily detected by X-ray. When pneumoconiosis due to vermiculite alone has been demonstrated, signs and symptoms resemble those of silicosis, but X-ray patterns differ. Tuberculosis was not a complication of these workers (as is the case with classical silicosis). Some vermiculite ores contain silica which converts to the crystalline form when the ore is heated to make expanded vermiculites; this may in turn produce a form of silicosis amongst workers exposed to expanded forms.
Many cases of mica pneumoconiosis have been reported in the literature. A significant number of the cases suggest that pneumoconiosis may be caused by pure mica alone. In only a few cases was the diagnosis based on clinical examination, radiography, and lung biopsy or autopsy results. Several epidemiologic studies have been performed among mica-processing workers, and these studies are all cross-sectional. In addition many experimental investigations have been carried out. However, there are no controlled inhalation studies among them. The results from the intratracheal instillation studies do not give a unanimous conclusion as to whether pure mica is fibrogenic or not. Present knowledge suggests that pure mica is moderately toxic and may induce pneumoconiosis. Exposure to mica is usually associated with exposure to other minerals such as quartz and feldspar.
Two men developed pneumoconiosis after grinding and packing powdered mica in the course of their working life. The disease was characterised by progressive dyspnoea, a restrictive impairment of ventilation, a reduced transfer factor, and hypoxaemia. Radiographs showed widespread fine nodular and linear shadows. Progression occurred after cessation of exposure, but this was much more pronounced in the man who died from coronary artery disease. Postmortem examination showed widespread fine fibrosis and nodules measuring up to 1.5 cm in diameter, all related to the deposition of doubly refractile crystals. Mineral formed over 9% of dry tissue weight, and electron microscopy and x-ray analysis showed it to be muscovite. Other minerals were not found.
Chronic inhalation exposure of production workers has caused decreased pulmonary function ad myocardial dystrophy. There is suggestive but inconclusive evidence that carbon black containing polyaromatic hydrocarbons (PAHs) has been responsible for induction of skin cancers in exposed workers.
Long term inhalation of carbon black can cause cough, phlegm, tiredness,chest pain and headache. Dermal, mucosal, or inhalation exposure can cause irritation.
Inhalation of carbon black by mice,rats and monkeys caused thickened alveolar walls, increased pulmonary collagen, right atrial and ventricular strain, hypertrophy of the right atrial and ventricular septum and increased heart weights. Although carbon black itself did not cause cancer in treated animals, carbon black containing polyaromatic hydrocarbons (PAHs) did cause cancer following chronic administration by all routes tested.
Epidemiological studies of workers in the carbon black producing industries of North America and Western Europe show no significant health effect due to occupational exposure to carbon black. Several other studies provide conflicting evidence. Early studies in the former USSR and Eastern Europe report respiratory diseases amongst workers exposed to carbon black, including bronchitis, pneumonia, emphysema and rhinitis. These studies are of questionable validity due to inadequate study design and methodology, lack of appropriate controls for cigarette smoking and other confounding factors such as concurrent exposure to carbon dioxide, coal oil and petroleum vapours. Moreover, review of these studies indicates that the concentrations of carbon black were greater than current occupational standards.
Carbon black may cause adverse pulmonary changes following prolonged or repeated inhalation of the dust; these include oral mucosal lesions, bronchitis and pneumoconiosis which may lead to lung tumours.
The body of evidence of carcinogenicity in animal studies comes from two chronic inhalation studies and two intratracheal instillation studies in rats, which showed significantly elevated rates of lung cancer in exposed animals. An inhalation study was tested on mice, but did not show significantly elevated rates of lung cancer in exposed animals. Epidemiologic data comes from three different cohort studies of carbon black production workers. Two studies, from the United Kingdom and Germany, with over 1,000 workers in each study group, showed elevated mortality from lung cancer in the carbon black workers. Another study of over 5,000 workers in the United States did not show elevated mortality from lung cancer in the carbon black workers. Newer findings of increased lung cancer mortality in an update from the UK study may suggest that carbon black could be a late-stage carcinogen. However, a more recent and larger study from Germany did not confirm this hypothesis that carbon black acts as a late-stage carcinogen.
In studies employing channel and furnace black, hamsters, mice, guinea pigs, rabbits and monkeys exposed to dusts for 7 hours/day, 5 days/week, at concentrations of 87.4 mg/m3 for channel black and 56.5 mg/m3 for furnace black, no malignancies were observed in any of the animals. Channel black had little if any absorbed polyaromatic hydrocarbons (PAHs) (as benzene extractables) whilst furnace black had 0.28%.
Several findings have strengthened the association between inflammation and cancer and between the particle surface area dose of carbon black and other poorly soluble low toxicity (PSLT) particles and the pulmonary inflammation response in mice and the proinflammatory effects in lung cells in vitro. Other evidence suggests that in addition to a cancer mechanism involving indirect genotoxicity through inflammation and oxidative stress, nanoparticles may act as direct carcinogens .
Carbon black appears to act like PSLT particles, which can elicit lung tumours in rats following prolonged exposure to sufficiently high concentrations of particles. Particle surface area dose was found to be most predictive of pulmonary inflammation and tumour response in rats when comparing the dose-response relationships for various types and sizes of PSLT including carbon black. Compared to fine PSLT, much lower concentrations of ultrafine PSLT (e.g. 2.5, 6.5 or 11.5 mg/m3 carbon black and ~10 mg/m3 ultrafine titanium dioxide) were associated with impaired clearance, persistent inflammation, and malignant lung tumours in chronic inhalation studies in rats. Most evidence suggests that carbon black and other PSLT-elicited lung tumours occurs through a secondary genotoxic mechanism, involving chronic inflammation and oxidative stress. Experimental studies have shown that when the particle lung dose reaches a sufficiently high concentration (e.g.,mass dose of ~0.5 mg fine-sized PSLT/g lung in rats), the alveolar macrophage-medicated clearance process begins to

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be impaired (complete impairment occurs at ~10 mg/g lung. Overloading of lung clearance is accompanied by pulmonary inflammation, leading to increased production of reactive oxygen and nitrogen species, depletion of antioxidants and/or impairment of other defense mechanisms, cell injury, cell proliferation, fibrosis, and as seen in rats, induction of mutations and eventually cancer. Rats appear to be more sensitive to carbon black and other PSLT than other rodent species. Although studies in humans have not shown a direct link between inhaled PSLT and lung cancer, many of the steps in the mechanism observed in rats have also been observed in humans who work in dusty jobs, including increased particle lung retention and pulmonary inflammation in workers exposed to coal dust or crystalline silica and elevated lung cancer has been observed in some studies of workers exposed to carbon black, crystalline silica, and diesel exhaust particles
Monkeys exposed to channel black for 1000-1500 hours showed evidence of electrocardiac changes indicative of right atrial and right ventricular strain. These changes increased progressively until after 10,000 hours of exposure, when the changes were marked. The authors of this study concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of non-toxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours' exposure and marked atrial and right ventricular strain after 10,000 hours' exposure. The authors concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of nontoxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours exposure and marked atrial and right ventricular strain after 10,000 hours exposure.
Chromatographic fractions of oily material extracted from carbon black have been shown to be carcinogenic whilst the unfractionated extracts are not. The activity of some carcinogens appear to be inhibited by carbon black itself.
Repeated exposure to synthetic amorphous silicas may produce skin dryness and cracking.
Available data confirm the absence of significant toxicity by oral and dermal routes of exposure.
Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted 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. Differences in values may be due to particle size, and therefore the number of particles administered per unit dose. Generally, as particle size diminishes so does the NOAEL/ LOAEL. Exposure produced transient increases in lung inflammation, markers of cell injury and lung collagen content. There was no evidence of interstitial pulmonary fibrosis.
Animal testing shows that methyl ethyl ketone may have slight effects on the nervous system, liver, kidney and respiratory system; there may also be developmental effects and an increase in birth defects. However, there is limited information available on the long-term effects of methyl ethyl ketone in humans, and no information is available on whether it causes developmental or reproductive toxicity or cancer. It is generally considered to have low toxicity, but it is often used in combination with other solvents, and the toxic effects of the mixture may be greater than with either solvent alone. Combinations of n-hexane or methyl n-butyl ketone with methyl ethyl ketone may increase the rate of peripheral neuropathy, a progressive disorder of the nerves of the extremities. Combinations with chloroform also show increase in toxicity.
For copper and its compounds (typically copper chloride):
Acute toxicity: There are no reliable acute oral toxicity results available. Animal testing shows that skin in exposure to copper may lead to hardness of the skin, scar formation, exudation and reddish changes. Inflammation, irritation and injury of the skin were noted.
Repeat dose toxicity: Animal testing shows that very high levels of copper monochloride may cause anaemia.
Genetic toxicity: Copper monochloride does not appear to cause mutations in vivo, although chromosomal aberrations were seen at very high concentrations in vitro.
Cancer-causing potential: There was insufficient information to evaluate the cancer-causing activity of copper monochloride.
Chronic excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas. Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.
Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.
High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may be become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.
Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up.
[K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]
Repeated oral doses of 3 g/kg for propylene glycol monomethyl ether (PGME) produced minor changes in the liver and kidneys in rats. Repeated doses on the skin over a 90-day period resulted in absorption and anaesthetic death at 7-10 ml/kg/day. Mild narcosis was observed after topical application of 2-4 ml/kg/day.
Administration of 2% PGME in drinking water ad libitum to males for 25 days did not elicit significant changes in testes or seminal vesicle and coagulating gland weights or in peripheral leukocyte counts. No significant testicular toxicity was found in rats or rabbits that were exposed at up to 3000 PGME, 6 hours/day, 5 days/week for 13 weeks. Oral and parenteral administration to pregnant rabbits, mice and rats did not induce congenital malformations at concentrations up to 1800 mg/kg/day.
In a study on the teratogenic potential of the acetate of the beta-isomer (2-methoxy-1-propyl acetate), a significant increase in the number of litters with abnormal rats and rabbits was found after inhalation exposure by the mothers to 2700 ppm or 550 ppm, respectively, on days 6 to 15, or 6 to 18 of gestation. The rabbit inhalation no-observed-adverse effect concentration was 145 ppm. A similar embryotoxicity profile was seen after inhalation of 2-methoxy-1-propanol (beta-PGMA). In contrast to the alpha-isomer, beta-PGMA is oxidised in rats to 2-methoxypropionic acid.
Male dogs exposed to the beta-isomer, developed numerous spermiophages in epididymi. Administration of high doses of the beta-isomer to rats, by gavage, caused delayed ossification of the skull of rat foetus.
Whilst alpha-PGMA undergoes hepatic O-demethylation as the principal pathway, the beta-isomer is detoxified by alcohol/ aldehyde dehydrogenase. Commercial PGME contains low concentrations of the beta-isomer.
Long term exposure to the dusts of titanium and several of its compounds produces chronic lung disease (fibrosis) in animals. Radiological evidence exists amongst titanium dioxide workers suggesting chronic lung changes which resemble a slight form of silicosis. Workers chronically exposed to titanium or titanium dioxide dusts show a high incidence of chronic bronchitis (endobronchitis and peribronchitis). Early stages of this disease are characterised by impaired pulmonary respiration and ventilatory capacity and by reduced blood alkalinity. Cardiac changes characteristic of pulmonary disease (with hypertrophy of the right auricle) have also been observed amongst workers.
Titanium employed in implants has provoked immune responses which occur locally as metallosis and systemically as raised serum levels of activated T-lymphocytes. Some concern has been expressed about the potential for generating bone-resorbing mediators associated with titanium wear-debris.
The largest of the cohort studies was among white male production workers in the titanium dioxide industry in six European countries. The study indicated a slightly increased risk for lung cancer compared with the general population. However, there was no evidence of an exposure-response relationship within the cohort. No increase in the mortality rates for kidney cancer was found when the cohort was compared with the general population, but there was a suggestion of an exposure-response relationship in internal analyses. The other cohort studies, both of which were conducted in the USA, did not report an increased risk for lung cancer or cancer at any other site; no results for kidney cancer were reported, presumably because there were few cases.
One population-based case-control study conducted in Montreal did not indicate an increased risk for lung or kidney cancer.
In summary, the studies do not suggest an association between occupational exposure to titanium dioxide as it occurred in recent decades in western Europe and North America and risk for cancer.
All the studies had methodological limitations; misclassification of exposure could not be ruled out. None of the studies was designed to assess the impact of particle size (fine or ultrafine) or the potential effect of the coating compounds on the risk for lung cancer.
An increased incidence of lung adenomas in rats of both sexes and of cystic keratinising lesions, diagnosed as squamous cell carcinomas in female rats, was seen in animals subject to high doses of inhaled titanium dioxide. Intratracheal delivery of titanium dioxide in combination with benz[a]pyrene produced an increase in benign and malignant tumours of the larynx, trachea and lungs in hamsters.
Squamous cell carcinomas developed after exposure to 250 mg/m3 for 6 hours/day, 5 days/week for 2 years in rats; the type of carcinoma that developed was considered to be a unique experimentally induced tumour and to be of questionable relevance for extrapolation of the

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results to humans. Given the extremely high level of dust in the lungs, the carcinomas were postulated to be the result of saturation of the normal pulmonary clearance mechanisms. At 50 mg/m3, massive accumulations of dust-laden macrophages, foamy dust cells and free particles were considered indicative of such overload.
Industrial workers exposed to 14 parts per million ethylbenzene experienced headaches, irritability and rapid fatigue. Some workers exposed for over 7 years showed nervous system disturbances, while other workers had enlarged livers.
Prolonged and repeated exposure may be harmful to the central nervous system (CNS), upper respiratory tract, and/or may cause liver disorders. It may also cause drying, scaling and blistering of the skin. Animal testing showed that chronic exposure to ethylbenzene may increase the incidence of tumours of the kidney, lung and liver.
4-Chlorobenzotrifluoride (PCBTF) has structural similarities with 4-chlorotrichlorotoluene, a carcinogen in mice, and to trifluralin (N,N-dipropyl-2,6-dinitro-4-trifluoromethylaniline), a rodent carcinogen.

ColorSpec Tinter (Colorspec No Mix_E Basecoat)

TOXICITY	IRRITATION
Not Available	Not Available

n-butyl acetate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 3200 mg/kg[2]	Eye (Human): 300ppm
Inhalation (Rat) LC50: 0.74 mg/l4h[2]	Eye (Rodent - rabbit): 100mg - Moderate
Oral (Rabbit) LD50; 3200 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: no adverse effect observed (not irritating)[1]

methyl ethyl ketone

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 6480 mg/kg[2]	Eye (Human): 350ppm
Inhalation (Mouse) LC50: 32 mg/L4h[2]	Eye (Rodent - rabbit): 80mg
Oral (Rat) LD50: 2054 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Skin (Rodent - rabbit): 14mg/24H - Mild
	Skin (Rodent - rabbit): 402mg/24H - Mild
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: no adverse effect observed (not irritating)[1]

propylene glycol monomethyl ether - mixture of isomers

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 100mg - Severe
Oral (Rat) LD50: 3739 mg/kg[2]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Eye: no adverse effect observed (not irritating)[1]
	Skin (Rodent - rabbit): 500mg - Mild
	Skin: no adverse effect observed (not irritating)[1]

C.I. Pigment White 6

TOXICITY	IRRITATION
dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: >2.28 mg/l4h[1]	Skin (Human): 300ug/3D (intermittent) - Mild
Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

4-chlorobenzotrifluoride

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: >32.03 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
Oral (Mouse) LD50; 11500 mg/kg[2]	

naphtha petroleum, light aromatic solvent

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye (Rodent - rabbit): 100uL/24H - Mild
Inhalation (Rat) LC50: >4.42 mg/L4h[1]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50: >4500 mg/kg[1]	Skin: adverse effect observed (irritating)[1]

ethyl-3-ethoxypropionate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 4076 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: 1250 ppm4h[2]	Skin (Rodent - rabbit): 500mg/24H - Mild
Oral (Rat) LD50: ~3200-5000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

ferric oxide

TOXICITY	IRRITATION
Oral (Rat) LD50: >5000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Skin: no adverse effect observed (not irritating)[1]

C.I. Pigment Yellow 83

TOXICITY	IRRITATION
dermal (rat) LD50: >3000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: >0.23 mg/L4h[1]	Skin (Rodent - rabbit): 500mg - Mild

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	Oral (Rat) LD50: >5000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
solvent naphtha petroleum, heavy aromatic	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 100uL/24H - Moderate
	Inhalation (Rat) LC50: >0.003 mg/L4h[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin (Rodent - rabbit): 500uL/24H - Mild
		Skin (Rodent - rabbit): 500uL/24H - Moderate
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
aluminium	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >2.3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
mica	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
C.I. Pigment Yellow 129	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: ~1.825 mg/L4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >5000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
carbon black	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
diacetone alcohol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 13500 mg/kg[2]	Eye (Human): 100ppm/15M
	Oral (Rat) LD50: 2520 mg/kg[2]	Eye (Rodent - rabbit): 100uL/24H - Severe
		Eye (Rodent - rabbit): 20mg - Severe
		Eye: adverse effect observed (irritating)[1]
		Skin (Rodent - rabbit): 10mg/24H
		Skin (Rodent - rabbit): 500mg - Mild
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
naphtha petroleum, heavy, hydrotreated	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >4.42 mg/L4h[1]	Skin: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: >4500 mg/kg[1]	
graphite	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >2 mg/L4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >200 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
ethylbenzene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 17800 mg/kg[2]	Eye (Rodent - rabbit): 500mg - Severe
	Inhalation (Rat) LC50: 17.2 mg/l4h[2]	Skin (Rodent - rabbit): 15mg/24H - Mild
	Oral (Rat) LD50: 3500 mg/kg[2]	
C.I. Pigment Violet 23	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
n-butanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3400 mg/kg[2]	Eye (Human): 50ppm
	Inhalation (Rat) LC50: 8000 ppm4h[2]	Eye (Human): 990ppm/1H
	Oral (Rat) LD50: 790 mg/kg[2]	Eye (Rodent - rabbit): 0.005mL - Severe
		Eye (Rodent - rabbit): 0.1mL
		Eye (Rodent - rabbit): 1.62mg - Severe
		Eye (Rodent - rabbit): 2mg/24H - Severe

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		Eye: adverse effect observed (irreversible damage)[1]
		Skin (Human): 20uL/20M
		Skin (Rodent - rabbit): 20mg/24H - Moderate
		Skin: adverse effect observed (irritating)[1]
N-methyl-2-pyrrolidone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 8000 mg/kg[2]	Eye (Human): 530ppm/30M - Mild
	Inhalation (Rat) LC50: 3.1-8.8 mg/l4h[2]	Eye (Rodent - rabbit): 0.1mL
	Oral (Rat) LD50: 3914 mg/kg[2]	Eye (Rodent - rabbit): 100mg - Moderate
		Eye: adverse effect observed (irritating)[1]
		Skin: adverse effect observed (irritating)[1]
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 3100 mg/kg[2]	Not Available
C.I. Solvent Yellow 88	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
methyl methacrylate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >5000 mg/kg[2]	Eye (Rodent - rabbit): 150mg
	Inhalation (Rat) LC50: 29.8 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 7872 mg/kg[2]	Skin (Human - woman): 2%/48H
		Skin (Rodent - rabbit): 10gm
		Skin: adverse effect observed (irritating)[1]
propylene glycol monomethyl ether - alpha isomer	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Inhalation (Rat) LC50: >6 mg/l4h[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 3739 mg/kg[1]	Skin (Rodent - rabbit): 500mg - Mild
		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**
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
PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS	NOTE: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Fetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
C.I. PIGMENT WHITE 6	Substance has been investigated as a mutagen, tumorigen and primary irritant. 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.

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	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 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.
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
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 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

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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 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

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	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 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.
ETHYL-3-ETHOXYPROPIONATE	* Union Carbide ** Endura Manufacturing
C.I. PIGMENT YELLOW 83	For diarylide (disazo) pigments (3,3'-dichlorobenzidine-containing):

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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 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).

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	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
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported
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].
ETHYLBENZENE	Liver changes, utheral tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded. Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylgloxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances**.** Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys. Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures ***(400 ppm and greater)*** of ethylbenzene In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncitial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland. In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.
C.I. PIGMENT VIOLET 23	No carcinogenic effects observed during a 43 day test animal feeding study on Pigment Violet 23. [Manufacturer]
N-BUTANOL	for n-butanol **Acute toxicity:** n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC0 of 8000 ppm (24000 mg/m3) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA s localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser. The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed. **Repeat dose toxicity:** An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAc) to BA. Hydrolysis of BAc in blood and brain was estimated to be 99 percent complete within 2.7 minutes (elimination t1/2 = 0.41 minute). Thus, organisms exposed to BAc can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAc can be used as supplemental, surrogate data to provide information on the toxicity of BA. A thirteen-week, subchronic exposure to BAc, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m3) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled

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	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.
N-METHYL-2-PYRROLIDONE	for N-methyl-2-pyrrolidone (NMP): **Acute toxicity:** In rats, NMP is absorbed rapidly after inhalation, oral, and dermal administration, distributed throughout the organism, and eliminated mainly by hydroxylation to polar compounds, which are excreted via urine. About 80% of the administered dose is excreted as NMP and NMP metabolites within 24 h. A probably dose-dependent yellow coloration of the urine in rodents is observed. The major metabolite is 5-hydroxy-***N***-methyl-2-pyrrolidone. Studies in humans show comparable results. Dermal penetration through human skin has been shown to be very rapid. NMP is rapidly biotransformed by hydroxylation to 5-hydroxy-***N***-methyl-2-pyrrolidone, which is further oxidized to ***N***-methylsuccinimide; this intermediate is further hydroxylated to 2-hydroxy-***N***-methylsuccinimide. These metabolites are all colourless. The excreted amounts of NMP metabolites in the urine after inhalation or oral intake represented about 100% and 65% of the administered doses, respectively. NMP has a low potential for skin irritation and a moderate potential for eye irritation in rabbits. Repeated daily doses of 450 mg/kg body weight administered to the skin caused painful and severe haemorrhage and eschar formation in rabbits. These adverse effects have not been seen in workers occupationally exposed to pure NMP, but they have been observed after dermal exposure to NMP used in cleaning processes. No sensitisation potential has been observed. In acute toxicity studies in rodents, NMP showed low toxicity. Uptake of oral, dermal, or inhaled acutely toxic doses causes functional disturbances and depressions in the central nervous system. Local irritation effects were observed in the respiratory tract when NMP was inhaled and in the pyloric and gastrointestinal tracts after oral administration. In humans, there was no irritative effect in the respiratory system after an 8-h exposure to 50 mg/m3. **Repeat dose toxicity:** There is no clear toxicity profile of NMP after multiple administration. In a 28-day dietary study in rats, a compound-related decrease in body weight gain was observed in males at 1234 mg/kg body weight and in females at 2268 mg/kg body weight. Testicular degeneration and atrophy in males and thymic atrophy in females were observed at these dose levels. The no-observed-adverse-effect level (NOAEL) was 429 mg/kg body weight in males and 1548 mg/kg body weight in females. In a 28-day intubation study in rats, a dose-dependent increase in relative liver and kidney weights and a decrease in lymphocyte count in both sexes were observed at 1028 mg/kg body weight. The NOAEL in this study was 514 mg/kg body weight. In another rat study, daily dietary intake for 90 days caused decreased body weights at doses of 433 and 565 mg/kg body weight in males and females, respectively. There were also neurobehavioural effects at these dose levels. The NOAELs in males and females were 169 and 217 mg/kg body weight, respectively. The toxicity profile after exposure to airborne NMP depends strongly on the ratio of vapour to aerosol and on the area of exposure (i.e., head-only or whole-body exposure). Because of higher skin absorption for the aerosol, uptake is higher in animals exposed to aerosol than in those exposed to vapour at similar concentrations. Studies in female rats exposed head only to 1000 mg/m3 showed only minor nasal irritation, but massive mortality and severe effects on major organs were observed when the females were whole-body exposed to the same concentration of coarse droplets at high relative humidity. Several studies in rats following repeated exposure to NMP at concentrations between 100 and 1000 mg/m3 have shown systemic toxicity effects at the lower dose levels. In most of the studies, the effects were not observed after a 4-week observation period. In rats, exposure to 3000 mg NMP/m3 (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight gain, an increase in erythrocytes, haemoglobin, haematocrit, and mean corpuscular volume, decreased absolute testis weight, and cell loss in the germinal epithelium of the testes. The NOAEL was 500 mg/m3. There are no data in humans after repeated-dose exposure. **Carcinogenicity:** NMP did not show any clear evidence for carcinogenicity in rats exposed to concentrations up to 400 mg/m3 in a long-term inhalation study. **Genotoxicity:** The mutagenic potential of NMP is weak. Only a slight increase in the number of revertants was observed when tested in a ***Salmonella*** assay with base-pair substitution strains. NMP has been shown to induce aneuploidy in yeast ***Saccharomyces cerevisiae*** cells. No investigations regarding mutagenicity in humans were available. **Reproductive toxicity:** In a two-generation reproduction study in rats, whole-body exposure of both males and females to 478 mg/m3 of NMP vapour for 6 h/day, 7 days/week, for a minimum of 100 days (pre-mating, mating, gestation, and lactation periods) resulted in a 7% decrease in fetal weight in the F1 offspring. A 4-11% transient, non-dose-dependent decrease was observed in the average pup weight at all exposure levels tested (41, 206, and 478 mg/m3). **Developmental toxicity:** When NMP was administered dermally, developmental toxicity was registered in rats at 750 mg/kg body weight. The observed effects were increased preimplantation losses, decreased fetal weights, and delayed ossification. The NOAEL for both developmental effects and maternal toxicity (decreased body weight gain) was 237 mg/kg body weight. Inhalation studies in rats (whole-body exposure) demonstrated developmental toxicity as increased preimplantation loss without significant effect on implantation rate or number of live fetuses at 680 mg/m3 and behavioural developmental toxicity at 622 mg/m3. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m3, and the NOAEL for developmental effects was 360 mg/m3. A tolerable inhalation concentration, 0.3 mg/m3, based on mortality and organ damage, is expected to be protective against any possible reproductive toxicity. Similarly, an oral tolerable intake of 0.6 mg/kg body weight per day, based on a 90-day study, is expected to provide adequate protection against possible reproductive effects. Because of non-existent data on the exposure of the general population and very limited information on occupational exposure, no meaningful risk characterisation can be performed A substance (or part of a group of chemical substances) of very high concern (SVHC) - or product containing an SVHC: It is proposed that use within the European Union be subject to authorisation under the REACH Regulation.Indeed, listing of a substance as an SVHC by the European Chemicals Agency (ECHA) is the first step in the procedure for authorisation or restriction of use of a chemical. The criteria are given in article 57 of the REACH Regulation. A substance may be proposed as an SVHC if it meets one or more of the following criteria: ▸ it is carcinogenic *; ▸ it is mutagenic *; ▸ it is toxic for reproduction *; ▸ it is persistent, bioaccumulative and toxic (PBT substances); ▸ it is very persistent and very bioaccumulative (vPvB substances); ▸ there is "scientific evidence of probable serious effects to human health or the environment which give rise to an equivalent level of concern"; such substances are identified on a case-by-case basis. * Collectively described as CMR substances The "equivalent concern" criterion is significant because it is this classification which allows substances which are, for example, neurotoxic, endocrine-disrupting or otherwise present an unanticipated environmental health risk to be regulated under REACH] Simply because a substance meets one or more of the criteria does not necessarily mean that it will be proposed as an SVHC. Many such substances are already subject to restrictions on their use within the European Union, such as those in Annex XVII of the REACH Regulation SVHCs are substances for which the current restrictions on use (where these exist) might be insufficient. There are three priority groups for assessment: ▸ PBT substances and vPvB substances; ▸ substances which are widely dispersed during use; ▸ substances which are used in large quantities.
C.I. SOLVENT YELLOW 88	Not mutagenic in Ames assay * * Ciba Speciality Chemicals MSDS
METHYL METHACRYLATE	Inhalation (human) TCLo: 60 mg/m3(15 ppm) [* Manuf. Rohm & Haas] **For methyl methacrylate:**

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	Acute toxicity: MMA is rapidly absorbed after oral or inhalatory administration. ***In vitro*** skin absorption studies in human skin indicate that MMA can be absorbed through human skin. After inhalation to rats 10 to 20% of the substance is deposited in the upper respiratory tract where it is metabolised by local tissue esterases. Acute toxicity of MMA by the oral, dermal, and inhalative routes is low as judged by tests with different species: The oral LD50 for rats, mice, and rabbits is found to exceed 5000 mg/kg bw. Acute inhalation toxicity for rats and mice is described by LC50 values of > 25 mg/l/4 hours. Acute dermal toxicity is reported for rabbits to exceed 5000 mg/kg bw. Skin and respiratory irritation are reported for subjects exposed to monomeric MMA. The substance has been shown to produce severe skin irritation when tested undiluted on rabbit skin. There are indications from studies in animals that MMA can be irritating to the respiratory system. In contact with eyes MMA has shown only weak irritation of the conjunctivae. MMA has a moderate to strong sensitising potential in experimental animals**.** Cases of contact dermatitis have been reported for workers exposed to the monomeric chemical. There is no convincing evidence that MMA is a respiratory sensitiser in humans. The lead effect caused by MMA is a degeneration of the olfactory region of the nose being the most sensitive target tissue. For this effect a NOAEC of 25 ppm (104 mg/m3) in a two-year inhalation study in rats was identified but only slight effects on the olfactory tissues have been observed at 100 ppm. Concerning systemic effects, two different valid studies have been considered for identifying a N(L)OAEL. Due to different dose selections, different values for N(L)OEALs are available. The LOEALs and the NOEALs for female rats ranges between 400 and 500 ppm and from 100 to 250 ppm respectively. In subchronic inhalation studies systemic toxic effects were seen in rats >1000 ppm, respectively in mice >500 ppm, including degenerative and necrotic lesions in liver, kidney, brain, and atrophic changes in spleen and bone marrow. These effects were not seen in chronic studies up to 1000 ppm. Oral administration to rats resulted in a NOAEL of 200 mg/kg bw/d. MMA has ***in vitro*** the potential for induction of mutagenic effects, especially clastogenicity. However, this potential is limited to high doses with strong toxic effects. Furthermore, the negative ***in vivo*** micronucleus test and the negative dominant lethal assay indicate that this potential is not expressed ***in vivo***. There is no relevant concern on carcinogenicity of MMA in humans and animals. Epidemiology data on increased tumour rates in exposed cohorts are of limited reliability and cannot be related to MMA as the solely causal agent. MMA did not reveal an effect on male fertility when animals had been exposed to up to 9000 ppm. From the available developmental toxicity investigations, including an inhalation study according to OECD Guideline 414, no teratogenicity, embryotoxicity or foetotoxicity has been observed at exposure levels up to and including 2028 ppm (8425 mg/m3). The available human data on sexual disorders in male and female workers cannot be considered to conclude on reproductive toxicity effects of MMA due to the uncertain validity of the studies Where no "official" classification for acrylates and methacrylates exists, there has been cautious attempts to create classifications in the absence of contrary evidence. For example Monalkyl or monoarylesters of acrylic acids should be classified as R36/37/38 and R51/53 Monoalkyl or monoaryl esters of methacrylic acid should be classified as R36/37/38 Based on the available oncogenicity data and without a better understanding of the carcinogenic mechanism the Health and Environmental Review Division (HERD), Office of Toxic Substances (OTS), of the US EPA previously concluded that all chemicals that contain the acrylate or methacrylate moiety (CH2=CHCOO or CH2=C(CH3)COO) should be considered to be a carcinogenic hazard unless shown otherwise by adequate testing. This position has now been revised and acrylates and methacrylates are no longer ***de facto*** carcinogens.
PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER	NOTE: For PGE - mixed isomers: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.
N-BUTYL ACETATE & DIACETONE ALCOHOL & ETHYLBENZENE & N-BUTANOL	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 & PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS & ETHYL-3-ETHOXYPROPIONATE & DIACETONE ALCOHOL & ETHYLBENZENE & N-BUTANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
METHYL ETHYL KETONE & PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS & 4-CHLOROBENZOTRIFLUORIDE & FERRIC OXIDE & MICA & GRAPHITE & N-BUTANOL & N-METHYL-2-PYRROLIDONE & METHYL METHACRYLATE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS & PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER	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

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	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.
PROPYLENE GLYCOL MONOMETHYL ETHER - MIXTURE OF ISOMERS & ALUMINIUM & MICA & C.I. PIGMENT YELLOW 129 & CARBON BLACK & DIACETONE ALCOHOL & GRAPHITE	No significant acute toxicological data identified in literature search.
C.I. PIGMENT WHITE 6 & METHYL METHACRYLATE	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.
NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC & NAPHTHA PETROLEUM, HEAVY, HYDROTREATED	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.
CARBON BLACK & ETHYLBENZENE	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
BIS(1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL)SEBACATE & METHYL METHACRYLATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

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***

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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
EC50	48h	Crustacea	32mg/l	1
EC50	72h	Algae or other aquatic plants	246mg/l	2
EC50(ECx)	96h	Fish	18mg/l	2
LC50	96h	Fish	17-19mg/L	4

methyl ethyl ketone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	308mg/l	2
EC50	72h	Algae or other aquatic plants	1220mg/l	2
NOEC(ECx)	48h	Crustacea	68mg/l	2
EC50	96h	Algae or other aquatic plants	>500mg/L	4
LC50	96h	Fish	>324mg/L	4

propylene glycol monomethyl ether - mixture of isomers

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	373mg/l	2
EC50	72h	Algae or other aquatic plants	>1000mg/l	2
EC50	96h	Algae or other aquatic plants	>1000mg/l	2
NOEC(ECx)	336h	Fish	47.5mg/l	2
LC50	96h	Fish	100-180mg/l	2

C.I. Pigment White 6

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<1.1-9.6	7
EC50	48h	Crustacea	1.9mg/l	2
EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
EC50	96h	Algae or other aquatic plants	179.05mg/l	2
NOEC(ECx)	672h	Fish	>=0.004mg/L	2
LC50	96h	Fish	1.85-3.06mg/l	4

4-chlorobenzotrifluoride

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

naphtha petroleum, light aromatic solvent

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

ethyl-3-ethoxypropionate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	970mg/l	1
EC50(ECx)	48h	Crustacea	970mg/l	1
EC50	72h	Algae or other aquatic plants	>114.86mg/l	2
LC50	96h	Fish	45.3mg/l	2

ferric oxide

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

C.I. Pigment Yellow 83

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>0.1mg/l	2
EC50	48h	Crustacea	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2

Continued...

	NOEC(ECx)	24h	Fish	>=0.1mg/l	2

solvent naphtha petroleum, heavy aromatic

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.95mg/l	1
EC50	72h	Algae or other aquatic plants	<1mg/l	1
LC50	96h	Fish	0.58mg/l	2
EC50	96h	Algae or other aquatic plants	11.7mg/l	2
EC50(ECx)	48h	Crustacea	0.95mg/l	1

aluminium

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.736mg/L	2
NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
EC50	72h	Algae or other aquatic plants	0.017mg/L	2
EC50	96h	Algae or other aquatic plants	0.005mg/L	2
LC50	96h	Fish	0.078-0.108mg/l	2

mica

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

C.I. Pigment Yellow 129

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	5.54mg/l	2
EC10(ECx)	72h	Algae or other aquatic plants	1.81mg/l	2
LC50	96h	Fish	>100mg/l	2

carbon black

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	33.076-41.968mg/l	4
EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
NOEC(ECx)	24h	Crustacea	3200mg/l	1
LC50	96h	Fish	>100mg/l	2

diacetone alcohol

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

naphtha petroleum, heavy, hydrotreated

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>0.002mg/l	2
EC50	96h	Algae or other aquatic plants	64mg/l	2
EC50(ECx)	48h	Crustacea	>0.002mg/l	2

graphite

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

ethylbenzene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.37-4.4mg/l	4
EC50	72h	Algae or other aquatic plants	2.4-9.8mg/L	4
EC50(ECx)	24h	Algae or other aquatic plants	0.02-938mg/L	4
EC50	96h	Algae or other aquatic plants	1.7-7.6mg/L	4
LC50	96h	Fish	3.381-4.075mg/L	4

C.I. Pigment Violet 23

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

n-butanol

Endpoint	Test Duration (hr)	Species	Value	Source

Continued...

	EC50	48h	Crustacea	>500mg/l	1
	NOEC(ECx)	504h	Crustacea	4.1mg/l	2
	EC50	72h	Algae or other aquatic plants	>500mg/l	1
	EC50	96h	Algae or other aquatic plants	225mg/l	2
	LC50	96h	Fish	100-500mg/l	4

N-methyl-2-pyrrolidone					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	ca.4897mg/l	1
	EC50	72h	Algae or other aquatic plants	>500mg/l	1
	NOEC(ECx)	504h	Crustacea	12.5mg/l	2
	LC50	96h	Fish	464mg/l	1

bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC0(ECx)	24h	Crustacea	<10mg/l	1
	LC50	96h	Fish	0.34mg/l	1

C.I. Solvent Yellow 88					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	10mg/l	Not Available

methyl methacrylate					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	69mg/l	1
	EC50	72h	Algae or other aquatic plants	>110mg/l	2
	EC50	96h	Algae or other aquatic plants	170mg/l	1
	EC0(ECx)	48h	Crustacea	48mg/l	1
	LC50	96h	Fish	>79mg/l	2

propylene glycol monomethyl ether - alpha isomer					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	23300mg/l	1
	EC50	72h	Algae or other aquatic plants	>500mg/l	2
	EC50(ECx)	168h	Algae or other aquatic plants	>1000mg/l	1
	EC50	96h	Algae or other aquatic plants	>1000mg/l	2
	LC50	96h	Fish	>=1000mg/l	2

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

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

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
n-butyl acetate	LOW	LOW
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
propylene glycol monomethyl ether - mixture of isomers	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
C.I. Pigment White 6	HIGH	HIGH
4-chlorobenzotrifluoride	HIGH	HIGH
ethyl-3-ethoxypropionate	LOW	LOW
C.I. Pigment Yellow 83	HIGH	HIGH
diacetone alcohol	HIGH	HIGH
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
N-methyl-2-pyrrolidone	LOW	LOW
methyl methacrylate	LOW	LOW
propylene glycol monomethyl ether - alpha isomer	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
n-butyl acetate	LOW (BCF = 14)
methyl ethyl ketone	LOW (LogKOW = 0.29)
propylene glycol monomethyl ether - mixture of isomers	LOW (BCF = 2)
C.I. Pigment White 6	LOW (BCF = 10)
4-chlorobenzotrifluoride	LOW (BCF = 202)
ethyl-3-ethoxypropionate	LOW (LogKOW = 1.08)

Continued...

Ingredient	Bioaccumulation
C.I. Pigment Yellow 83	LOW (LogKOW = 8.6648)
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)
aluminium	LOW (LogKOW = 0.33)
diacetone alcohol	LOW (LogKOW = -0.14)
ethylbenzene	LOW (BCF = 79.43)
n-butanol	LOW (BCF = 0.64)
N-methyl-2-pyrrolidone	LOW (BCF = 0.16)
methyl methacrylate	LOW (BCF = 6.6)
propylene glycol monomethyl ether - alpha isomer	LOW (BCF = 2)

Mobility in soil

Ingredient	Mobility
n-butyl acetate	LOW (Log KOC = 20.86)
methyl ethyl ketone	MEDIUM (Log KOC = 3.827)
propylene glycol monomethyl ether - mixture of isomers	HIGH (Log KOC = 1)
C.I. Pigment White 6	LOW (Log KOC = 23.74)
4-chlorobenzotrifluoride	LOW (Log KOC = 1912)
ethyl-3-ethoxypropionate	LOW (Log KOC = 10)
C.I. Pigment Yellow 83	LOW (Log KOC = 1126000)
diacetone alcohol	HIGH (Log KOC = 1)
ethylbenzene	LOW (Log KOC = 517.8)
n-butanol	MEDIUM (Log KOC = 2.443)
N-methyl-2-pyrrolidone	LOW (Log KOC = 20.94)
methyl methacrylate	LOW (Log KOC = 10.14)
propylene glycol monomethyl ether - alpha isomer	HIGH (Log 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.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3YE

Land transport (ADG)

14.1. **UN number or ID number**	1263
14.2. **UN proper shipping name**	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base)
14.3. **Transport hazard class(es)**	Class: 3

Continued...

	Subsidiary Hazard: Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 163 367 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	1263
14.2. **UN proper shipping name**	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 3 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 3L
14.4. **Packing group**	II
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **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)

14.1. **UN number**	1263
14.2. **UN proper shipping name**	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base)
14.3. **Transport hazard class(es)**	IMDG Class: 3 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	II
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-E , S-E Special provisions: 163 367 Limited Quantities: 5 L

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
n-butyl acetate	Not Available
methyl ethyl ketone	Not Available
propylene glycol monomethyl ether - mixture of isomers	Not Available
C.I. Pigment White 6	Not Available
4-chlorobenzotrifluoride	Not Available
naphtha petroleum, light aromatic solvent	Not Available
ethyl-3-ethoxypropionate	Not Available
ferric oxide	Not Available
C.I. Pigment Yellow 83	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
aluminium	Not Available
mica	Not Available
C.I. Pigment Yellow 129	Not Available
carbon black	Not Available
diacetone alcohol	Not Available
naphtha petroleum, heavy, hydrotreated	Not Available

Continued...

Product name	Group
graphite	Not Available
ethylbenzene	Not Available
C.I. Pigment Violet 23	Not Available
n-butanol	Not Available
N-methyl-2-pyrrolidone	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available
C.I. Solvent Yellow 88	Not Available
methyl methacrylate	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
n-butyl acetate	Not Available
methyl ethyl ketone	Not Available
propylene glycol monomethyl ether - mixture of isomers	Not Available
C.I. Pigment White 6	Not Available
4-chlorobenzotrifluoride	Not Available
naphtha petroleum, light aromatic solvent	Not Available
ethyl-3-ethoxypropionate	Not Available
ferric oxide	Not Available
C.I. Pigment Yellow 83	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
aluminium	Not Available
mica	Not Available
C.I. Pigment Yellow 129	Not Available
carbon black	Not Available
diacetone alcohol	Not Available
naphtha petroleum, heavy, hydrotreated	Not Available
graphite	Not Available
ethylbenzene	Not Available
C.I. Pigment Violet 23	Not Available
n-butanol	Not Available
N-methyl-2-pyrrolidone	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available
C.I. Solvent Yellow 88	Not Available
methyl methacrylate	Not Available
propylene glycol monomethyl ether - alpha isomer	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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
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)

propylene glycol monomethyl ether - mixture of isomers 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

C.I. Pigment White 6 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

Continued...

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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

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

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

Australian Inventory of Industrial Chemicals (AIIC)

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 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

C.I. Pigment Yellow 83 is found on the following regulatory lists

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

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 - Not Classified as Carcinogenic

aluminium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

mica is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

C.I. Pigment Yellow 129 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)

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)

diacetone alcohol is found on the following regulatory lists

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

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

graphite is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ethylbenzene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

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

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

n-butanol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Continued...

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)

N-methyl-2-pyrrolidone 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)
Chemical Footprint Project - Chemicals of High Concern List

bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate is found on the following regulatory lists

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

C.I. Solvent Yellow 88 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

methyl methacrylate 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 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

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

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

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (n-butyl acetate; methyl ethyl ketone; C.I. Pigment White 6; 4-chlorobenzotrifluoride; naphtha petroleum, light aromatic solvent; ethyl-3-ethoxypropionate; ferric oxide; C.I. Pigment Yellow 83; solvent naphtha petroleum, heavy aromatic; aluminium; mica; C.I. Pigment Yellow 129; carbon black; diacetone alcohol; naphtha petroleum, heavy, hydrotreated; graphite; ethylbenzene; C.I. Pigment Violet 23; n-butanol; N-methyl-2-pyrrolidone; bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; C.I. Solvent Yellow 88; methyl methacrylate; propylene glycol monomethyl ether - alpha isomer)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (mica)
Japan - ENCS	No (aluminium; mica; graphite)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	TSCA Inventory 'Active' substance(s) (n-butyl acetate; methyl ethyl ketone; propylene glycol monomethyl ether - mixture of isomers; C.I. Pigment White 6; 4-chlorobenzotrifluoride; naphtha petroleum, light aromatic solvent; ethyl-3-ethoxypropionate; ferric oxide; C.I. Pigment Yellow 83; solvent naphtha petroleum, heavy aromatic; aluminium; carbon black; diacetone alcohol; naphtha petroleum, heavy, hydrotreated; graphite; ethylbenzene; C.I. Pigment Violet 23; n-butanol; N-methyl-2-pyrrolidone; bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; C.I. Solvent Yellow 88; methyl methacrylate; propylene glycol monomethyl ether - alpha isomer); No (mica; C.I. Pigment Yellow 129)
Taiwan - TCSI	Yes
Mexico - INSQ	No (4-chlorobenzotrifluoride; C.I. Pigment Yellow 83; C.I. Pigment Yellow 129; C.I. Solvent Yellow 88)
Vietnam - NCI	Yes
Russia - FBEPH	No (C.I. Pigment Yellow 129; C.I. Solvent Yellow 88)
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	02/05/2024
Initial Date	30/04/2024

SDS Version Summary

Version	Date of Update	Sections Updated
2.2	02/05/2024	Identification of the substance / mixture and of the company / undertaking - Synonyms
3.1	02/05/2024	Name
3.2	16/04/2025	Identification of the substance / mixture and of the company / undertaking - Synonyms, Name

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
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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