

Opaque Pigment White

Barnes Products P/L

Chemwatch: 9848798

Version No: 6.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Issue Date: 09/02/2018

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L.GHS.AUS.EN

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

Product Identifier

Product name	Opaque Pigment White
Synonyms	BJB White
Other means of identification	Not Available

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

Relevant identified uses	Pigment.
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Details of the supplier of the safety data sheet

Registered company name	Barnes Products P/L
Address	5 Greenhills Avenue Moorebank NSW 2170 Australia
Telephone	+61 2 9793 7555
Fax	+61 2 9793 7091
Website	http://www.barnes.com.au/
Email	sales@barnes.com.au

Emergency telephone number

Association / Organisation	Barnes Products Pty Ltd
Emergency telephone numbers	+61 2 9793 7555 Business Hours
Other emergency telephone numbers	Poisons Information Centre 13 1126 after hours

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	1	1
Toxicity	1	1
Body Contact	2	2
Reactivity	1	1
Chronic	0	0

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

Poisons Schedule	Not Applicable
Classification	Not Applicable

Label elements

Hazard pictogram(s)	Not Applicable
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SIGNAL WORD	NOT APPLICABLE
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Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
13463-67-7	30-60	<u>titanium dioxide</u>
68515-49-1	30-60	<u>di-C9-11-alkyl phthalate, C10-rich</u>
21645-51-2	<5	<u>aluminium hydroxide</u>
7631-86-9	<5	<u>silica amorphous</u>
122-99-6	0.1-1	<u>ethylene glycol phenyl ether</u>
111-76-2	0.1-1	<u>ethylene glycol monobutyl ether</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none">▶ 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	<p>If skin contact occurs:</p> <ul style="list-style-type: none">▶ Immediately remove all contaminated clothing, including footwear.▶ Flush skin and hair with running water (and soap if available).▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none">▶ If fumes or combustion products are inhaled remove from contaminated area.▶ Lay patient down. Keep warm and rested.▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none">▶ If swallowed do NOT induce vomiting.▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.▶ Observe the patient carefully.▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

For acute or short term repeated exposures to ethylene glycol:

- ▶ Early treatment of ingestion is important. Ensure emesis is satisfactory.
- ▶ Test and correct for metabolic acidosis and hypocalcaemia.
- ▶ Apply sustained diuresis when possible with hypertonic mannitol.
- ▶ Evaluate renal status and begin haemodialysis if indicated. [I.L.O]
- ▶ Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- ▶ Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- ▶ Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- ▶ Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- ▶ Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.

Laitinen J., et al: *Occupational & Environmental Medicine* 1996; 53, 595-600

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Use water delivered as a fine spray to control fire and cool adjacent area. ‣ 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. ‣ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. <p>Combustion products include:</p> <ul style="list-style-type: none"> ‣ carbon dioxide (CO₂) ‣ other pyrolysis products typical of burning organic material. <p>May emit poisonous fumes. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

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	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb spill with sand, earth, inert material or vermiculite. ‣ Wipe up. ‣ Place in a suitable, labelled container for waste disposal.
Major Spills	<p>Environmental hazard - contain spillage. Moderate hazard.</p> <ul style="list-style-type: none"> ‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ No smoking, naked lights or ignition sources. ‣ Increase ventilation. ‣ Stop leak if safe to do so. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Absorb remaining product with sand, earth or vermiculite.

- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ 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 or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Packaging as recommended by manufacturer.
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	titanium dioxide	Titanium dioxide	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Fumed silica (respirable dust)	2 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Diatomaceous earth (uncalcined)	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Diatomaceous earth (uncalcined)	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica gel	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Fumed silica (respirable dust)	2 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica, fused	Not Available	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Precipitated silica	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Precipitated silica	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica gel	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	96.9 mg/m ³ / 20 ppm	242 mg/m ³ / 50 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
titanium dioxide	Titanium oxide; (Titanium dioxide)	30 mg/m3	330 mg/m3	2,000 mg/m3
aluminium hydroxide	Aluminum hydroxide	8.7 mg/m3	73 mg/m3	440 mg/m3
silica amorphous	Silica gel, amorphous synthetic	18 mg/m3	200 mg/m3	1,200 mg/m3
silica amorphous	Silica, amorphous fumed	18 mg/m3	100 mg/m3	630 mg/m3
silica amorphous	Siloxanes and silicones, dimethyl, reaction products with silica; (Hydrophobic silicon dioxide, amorphous)	120 mg/m3	1,300 mg/m3	7,900 mg/m3
silica amorphous	Silica, amorphous fume	45 mg/m3	500 mg/m3	3,000 mg/m3
silica amorphous	Silica amorphous hydrated	18 mg/m3	220 mg/m3	1,300 mg/m3
ethylene glycol phenyl ether	Phenoxyethanol, 2-; (Phenyl cellosolve)	1.5 ppm	16 ppm	97 ppm
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm

Ingredient	Original IDLH	Revised IDLH
titanium dioxide	5000 mg/m3	Not Available
di-C9-11-alkyl phthalate, C10-rich	Not Available	Not Available
aluminium hydroxide	Not Available	Not Available
silica amorphous	3000 mg/m3	Not Available
ethylene glycol phenyl ether	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.</p> <p>An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>										
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
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<p>Within each range the appropriate value depends on:</p>											
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	3: Intermittent, low production. 4: Large hood or large air mass in motion	3: High production, heavy use 4: Small hood-local control only
	<p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	
Personal protection		
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 	
Skin protection	See Hand protection below	
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> - frequency and duration of contact, - chemical resistance of glove material, - glove thickness and - dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> - When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. - Contaminated gloves should be replaced. <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> - Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. - Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <ul style="list-style-type: none"> ▶ Neoprene rubber gloves ▶ Nitrile rubber gloves 	
Body protection	See Other protection below	
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. 	
Thermal hazards	Not Available	

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

Opaque Pigment White

Material	CPI
BUTYL	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NEOPRENE	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

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

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	White viscous liquid with a slight odour; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	1.72 @25C
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	225 (PMCC)	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	0.19
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	3.270

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.

Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Inhalation hazard is increased at higher temperatures.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Phthalates (aromatic dicarboxylic acid esters), in general, exhibit low toxicity, partly because of poor absorption but mainly as a result of rapid metabolism in which the esters are saponified to phthalic acid (which is rapidly excreted) and the parent alcohol (which is subsequently metabolised). The pathology of these compounds seems to be related to the released alcohol and its biological effects. The rate of absorption of ingested phthalate esters is influenced by the content of dietary fat. Ingested phthalate esters may to a lesser degree be absorbed as the monoester derivatives or in the case of di(2-ethylhexyl)phthalate, as the diester. Cumulative toxicity of the phthalates has been observed on repeated administration. Both di-n-octyl phthalate and di(2-ethylhexyl)phthalate were found to have 22-28 times greater toxicity (based on LD50s) following repeated administration to animals. The liver has been shown to be the target organ affected by the phthalates. In general phthalates have induced liver enlargement; this increase in liver weight has been attributed to rapid cell division (hyperplasia) along with the detachment of cells (hypertrophy). The increase in liver weight caused by phthalates has been found to reverse to normal or even below normal levels on prolonged exposure. Exposure to phthalates, in general, has been found to be associated with a reduction in circulating cholesterol and serum triglyceride levels which accounted for a reduction in liver steroidogenesis. The phthalates also effect carbohydrate metabolism in the liver producing depleted glycogen electron transport inhibitors following interaction with mitochondria. Testicular atrophy produced in rats during feeding studies depends on the length and structure of the alcohol; in general the lower molecular weight esters produce the more severe effects. The toxicity of phthalic acid isomers decreases in the order o-phthalic acid, isophthalic acid and terephthalic acid. Phthalic acid is not metabolised but is excreted, unchanged, in the urine and faeces. Terephthalic acid appears to potentiate the biological effects of substances such as antibiotics, thiamine and sulfonamides.
Skin Contact	The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: <ul style="list-style-type: none"> ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but mild, 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 (non allergic). 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. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Limited evidence or practical experience suggests, that the material may cause moderate eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged exposure may cause moderate inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration
Chronic	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

Opaque Pigment White	TOXICITY	IRRITATION
	Not Available	Not Available
titanium dioxide	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2.28 mg/l4 h ^[1] Oral (rat) LD50: >2000 mg/kg ^[1]	Skin (human): 0.3 mg /3D (int)-mild *
di-C9-11-alkyl phthalate, C10-rich	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3160 mg/kg ^[1] Oral (rat) LD50: 30000 mg/kg ^[2]	Eye (rabbit): 500 mg/24h mild Skin (rabbit): 500 mg/24h mild

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

TITANIUM DIOXIDE

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

For titanium dioxide:

Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.

Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.

No data were available on genotoxic effects in titanium dioxide-exposed humans.

Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.

Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.

Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.

Animal carcinogenicity data

Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal 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.

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

* IUCLID

**DI-C9-11-ALKYL
PHTHALATE, C10-RICH**

High Molecular Weight Phthalate Esters (HMWPEs) Category as defined by the Phthalate Esters Panel HPV Testing Group (2001) and OECD (2004). The HMWPE group includes chemically similar substances produced from alcohols having backbone carbon lengths of ≥ 7 . Due to their similar chemical structure, category members are generally similar with respect to physicochemical, biological and toxicological properties or display an expected trend. Thus, read-across for toxicity endpoints is an appropriate approach to characterise selected endpoints for members of this category.

In some cases the substances have ester side group constituents that span two subcategories (i.e., transitional and high molecular weight constituents). If the level of C4 to C6 constituents in the substance exceeded 10%, the substance was conservatively placed in the transitional subcategory.

High molecular weight phthalates are used nearly exclusively as plasticisers of PVC.

They are very poorly soluble in water, and have very low vapor pressure. The extant database demonstrates that these substances have few biological effects. A notable exception to this generalisation is that hepatocarcinogenicity has been observed for diisononyl phthalate (DINP). The hepatocarcinogenicity effects of DINP are by a mechanism (peroxisomal proliferation) to which rodents are particularly sensitive. However, it does not appear to be relevant to humans.

The high molecular weight phthalates all demonstrate minimal acute toxicity, are not genotoxic, exhibit some liver and kidney effects at high doses, and are negative for reproductive and developmental effects. Further, the available data

indicate that the toxicological activity of these molecules diminishes with increasing molecular weight. Studies on HMWPEs indicate that they are rapidly metabolised in the gastrointestinal tract to the corresponding monoester, absorbed and excreted primarily in the urine.

Acute toxicity: The available data on phthalates spanning the carbon range from C8-C13 indicate that phthalate esters in the high molecular weight subcategory are not toxic by acute oral and dermal administration; LD50 values of all substances tested exceed the maximum amounts which can be administered to the animals. There are fewer data available on inhalation toxicity; only di-iso-nonyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) have been tested. However, the phthalates in the high molecular weight subcategory have extremely low vapor pressures, and exposure by inhalation at potentially hazardous levels is not anticipated.

Repeat dose toxicity. Several substances ranging from C8-C11 have been tested for repeated dose toxicity in studies ranging from 21 days to two years. Ditridecyl phthalate (CAS 119-06-2) has been studied by the Japan Ministry of Health and Welfare (unpublished report) and data for this substance is used as read-across data for DTDP*. In addition results from repeat dose studies examining DINP (CAS 685 15-48-0) and DIDP (CAS 68515-49-1) are used as read across for the di C9-C11 phthalates (CAS 68515-43-5). The principal effects found are those associated with peroxisomal proliferation, including liver enlargement and induction of peroxisomal enzymes. As shown for example in a comparative study of liver effects, the strongest inducers of peroxisomal proliferation were DEHP, DINP, and DIDP with substances of shorter and longer ester side chains (e.g., 610P*, 711P*, and diundecyl phthalate - DUP) showing less pronounced effects. Thus, it is reasonable to conclude that other members of this subcategory would show effects similar to but not more pronounced than those associated with DINP and DIDP. It should also be noted that the relevance of these findings to human health is, at best, questionable. It has been shown that these effects are mediated through the peroxisome proliferation-activated receptor alpha (PPAR α), and that levels of PPAR α are much higher in rodents than humans. Thus, one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence supporting this postulation is provided by studies in primates in which repeated administration of DEHP and DINP had no effects on liver, kidney or testicular parameters.

In this regard it should also be noted that kidney enlargement is also commonly observed but normally without any pathological changes. There is a component of the kidney changes which is also PPAR α -related. It has also been shown that in male rats, DINP induces an alpha 2u-globulin nephropathy which is male rat- specific but without relevance to humans. Thus, as was true for the liver changes, the relevance of the kidney changes to human health is also questionable

Finally, some of the lower molecular weight phthalates can induce testicular atrophy when administered to juvenile rats at high levels. However, the higher molecular weight phthalates including di-n-octyl phthalate (DnOP), DINP, DIDP, 610P, and 71 1P do not induce testicular atrophy. Further, the testis was not a target organ for DINP in either marmosets or cynomolgus monkeys. Thus, testicular atrophy is not an effect associated with phthalates in the high molecular weight subcategory

Reproductive toxicity: Reproductive toxicity tests in rats have been carried out with DINP, DIDP a linear C7-C9 phthalate (CAS 68515-41-3), a linear C9-C11 phthalate, and ditridecyl phthalate (Japan Ministry of Health and Welfare, unpublished report). None of these affected fertility or profoundly affected male reproductive development. A slight decrease in offspring viability was reported for both DIDP and ditridecyl phthalate at levels associated with maternal effects. DnOP was tested for effects on fertility in a continuous breeding protocol in mice, and, like the other members of this subcategory, did not reduce fertility. Thus, it can be concluded that the subcategory of high molecular weight phthalates do not affect fertility.

Developmental toxicity: Developmental toxicity tests in rats have been carried out with DINP; DIDP; C7-9 phthalate (CAS 68515-41-3); C9-11 phthalate (CAS 68515-43-5); and ditridecyl phthalate (CAS 119-06-2). None of the substances tested affected litter size, foetal survival or bodyweight, and none produced teratogenic effects. Increased frequencies of developmental variants including dilated renal pelvis, and supernumerary lumbar and cervical ribs were found at levels associated with maternal effects. The toxicological significance of these developmental variants is unclear. DnOP was not teratogenic in mice when tested at very high levels. Thus, it can be concluded that this subcategory of high molecular weight phthalates do not produce profound developmental effects in rodents

Genotoxicity: The majority of the substances in the subcategory of high molecular weight phthalates have been tested for genetic activity in the Salmonella assay, and all were inactive. One large program covering many of these substances was carried out by the National Institute of Environmental Health Sciences. Similarly, a range of substances covering the majority of the carbon numbers in this subcategory were found to be inactive in mouse lymphoma tests Chromosomal Aberrations. Two representative members of the subcategory of high molecular weight phthalates (DINP and DIDP) have been tested for chromosomal mutation in the mouse micronucleus test, and both were inactive. Ditricecyl phthalate (CAS 119-06-2) induced neither structural chromosomal aberrations nor polyploidy in CHL cells up to the limit concentration of 4.75 mg/ rnl, in the absence or presence of an exogenous metabolic activation system (Japan Ministry of Health and Welfare, unpublished report). Further, all of the low molecular weight and transitional phthalates that have been tested were inactive.

*610P - mixed decyl, hexyl and octyl esters (CAS Rn: 68648-93-1)
 *711P - C7,C11, branched and linear esters (CAS Rn: 111381-90-9)
 * DTDP - di-C11-14, C13 rich ester (CAS 68515-47-9)

The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens. However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

ALUMINIUM HYDROXIDE	No significant acute toxicological data identified in literature search.
SILICA AMORPHOUS	For silica amorphous: When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.

After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.

Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.

Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure.

Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m³ to 150 mg/m³. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m³. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m³. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL. Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic *in vitro*. No genotoxicity was detected in *in vivo* assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.

In humans, SAS is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.

There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.

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.
 Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]

**ETHYLENE GLYCOL
PHENYL ETHER**

The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles.

The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity.

At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin. The potential for eye irritation is minimal.

With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low. NOAELs for maternal and developmental toxicity are far in excess of current human exposure levels.

No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity *in vitro* bacterial assays, and *in vitro* mammalian cell assays. All *in vivo* micronucleus assays were negative.

It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients
 The Research Institute for Fragrance Materials (RIFM) Expert Panel
 Bacterial cell mutagen

**ETHYLENE GLYCOL
MONOBUTYL ETHER**

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):
 Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

Acute Toxicity: Oral LD₅₀ values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC₀ > 85 ppm (508 mg/m³) for EGHE, LC₅₀ > 400ppm (2620 mg/m³) for EGBEA to LC₅₀ > 2132 ppm (9061 mg/m³) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD₅₀ values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m³ and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m³), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m³), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m³) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m³ (rabbit-EGPE), 100 ppm or 425 mg/m³ (rat-EGPE), 50 ppm or 241 mg/m³ (rat EGBE) and 100 ppm or 483 mg/m³ (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m³ (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included

diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS

<p>TITANIUM DIOXIDE & DI-C9-11-ALKYL PHTHALATE, C10-RICH & ETHYLENE GLYCOL PHENYL ETHER & ETHYLENE GLYCOL MONOBUTYL ETHER</p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<p>ETHYLENE GLYCOL PHENYL ETHER & ETHYLENE GLYCOL MONOBUTYL ETHER</p>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>

<p>Acute Toxicity</p>	<p>☹</p>	<p>Carcinogenicity</p>	<p>☹</p>
<p>Skin Irritation/Corrosion</p>	<p>☹</p>	<p>Reproductivity</p>	<p>☹</p>
<p>Serious Eye Damage/Irritation</p>	<p>☹</p>	<p>STOT - Single Exposure</p>	<p>☹</p>
<p>Respiratory or Skin sensitisation</p>	<p>☹</p>	<p>STOT - Repeated Exposure</p>	<p>☹</p>
<p>Mutagenicity</p>	<p>☹</p>	<p>Aspiration Hazard</p>	<p>☹</p>

Legend: ✘ – Data available but does not fill the criteria for classification
✔ – Data available to make classification
☹ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Opaque Pigment White	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
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	Not Available	Not Available	Not Available	Not Available	Not Available
titanium dioxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	155mg/L	2
	EC50	48	Crustacea	>10mg/L	2
	EC50	72	Algae or other aquatic plants	5.83mg/L	4
	EC20	72	Algae or other aquatic plants	1.81mg/L	4
	NOEC	336	Fish	0.089mg/L	4
di-C9-11-alkyl phthalate, C10-rich	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>0.37mg/L	2
	EC50	48	Crustacea	>0.18mg/L	1
	EC50	96	Algae or other aquatic plants	>1.3mg/L	1
	NOEC	504	Crustacea	0.0034mg/L	2
aluminium hydroxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.2262mg/L	2
	EC50	48	Crustacea	0.7364mg/L	2
	EC50	96	Algae or other aquatic plants	0.0054mg/L	2
	NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2
silica amorphous	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	ca.2000mg/L	1
	EC50	48	Crustacea	ca.7600mg/L	1
	EC50	72	Algae or other aquatic plants	440mg/L	1
	EC10	72	Algae or other aquatic plants	140mg/L	1
	NOEC	72	Algae or other aquatic plants	60mg/L	1
ethylene glycol phenyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	154mg/L	2
	EC50	48	Crustacea	>500mg/L	1
	EC50	72	Algae or other aquatic plants	>500mg/L	1
	NOEC	504	Crustacea	9.43mg/L	2
ethylene glycol monobutyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1250mg/L	4
	EC50	48	Crustacea	>1000mg/L	4
	NOEC	96	Crustacea	1000mg/L	4
Legend:	<i>Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data</i>				

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
titanium dioxide	HIGH	HIGH
silica amorphous	LOW	LOW
ethylene glycol phenyl ether	LOW	LOW
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
titanium dioxide	LOW (BCF = 10)

di-C9-11-alkyl phthalate, C10-rich	HIGH (BCF = 3500)
silica amorphous	LOW (LogKOW = 0.5294)
ethylene glycol phenyl ether	LOW (LogKOW = 1.16)
ethylene glycol monobutyl ether	LOW (BCF = 2.51)

Mobility in soil

Ingredient	Mobility
titanium dioxide	LOW (KOC = 23.74)
silica amorphous	LOW (KOC = 23.74)
ethylene glycol phenyl ether	LOW (KOC = 12.12)
ethylene glycol monobutyl ether	HIGH (KOC = 1)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

TITANIUM DIOXIDE(13463-67-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

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

DI-C9-11-ALKYL PHTHALATE, C10-RICH(68515-49-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

ALUMINIUM HYDROXIDE(21645-51-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

SILICA AMORPHOUS(7631-86-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

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 4

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

ETHYLENE GLYCOL PHENYL ETHER(122-99-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Part 2, Section Seven - Appendix I

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

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

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (ethylene glycol phenyl ether; aluminium hydroxide; ethylene glycol monobutyl ether; di-C9-11-alkyl phthalate, C10-rich)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (di-C9-11-alkyl phthalate, C10-rich)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	<i>Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)</i>

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
titanium dioxide	13463-67-7, 1317-70-0, 1317-80-2, 12188-41-9, 1309-63-3, 100292-32-8, 101239-53-6, 116788-85-3, 12000-59-8, 12701-76-7, 12767-65-6, 12789-63-8, 1344-29-2, 185323-71-1, 185828-91-5, 188357-76-8, 188357-79-1, 195740-11-5, 221548-98-7, 224963-00-2, 246178-32-5, 252962-41-7, 37230-92-5, 37230-94-7, 37230-95-8, 37230-96-9, 39320-58-6, 39360-64-0, 39379-02-7, 416845-43-7, 494848-07-6, 494848-23-6, 494851-77-3, 494851-98-8, 55068-84-3, 55068-85-4, 552316-51-5, 62338-64-1, 767341-00-4, 97929-50-5, 98084-96-9
aluminium hydroxide	21645-51-2, 1330-44-5, 1302-29-0, 12252-70-9, 51330-22-4
silica amorphous	7631-86-9, 112945-52-5, 67762-90-7, 68611-44-9, 68909-20-6, 112926-00-8, 61790-53-2, 60676-86-0, 91053-39-3, 69012-64-2, 844491-94-7
ethylene glycol phenyl ether	122-99-6, 37220-49-8, 134367-25-2, 18249-17-7, 200260-63-5, 79586-53-1, 9004-78-8, 56257-90-0, 1219804-65-5

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average

PC—STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit.

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index

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