ColorSpec Plastic Primer, 400g (Colorspec Plastic Primer)

Motor Active

Chemwatch Hazard Alert Code: **4**

Chemwatch: **40-9389**
Version No: **6.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **10/03/2023**
Print Date: **19/10/2023**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	ColorSpec Plastic Primer, 400g (Colorspec Plastic Primer)
Chemical Name	Not Applicable
Synonyms	Product Code: CSPP400G
Proper shipping name	AEROSOLS
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	Aerosol for industrial and commercial use. Application is by spray atomisation from a hand held aerosol pack 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 numbers	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
Other emergency telephone numbers	13 11 26 (In Case of Emergency contact: Poison Information Hotline)

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

		Min	Max
Flammability	4		
Toxicity	2		
Body Contact	2		
Reactivity	1		
Chronic	3		

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

Poisons Schedule	Not Applicable
Classification [1]	Aerosols Category 1, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Germ Cell Mutagenicity Category 2, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	

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Signal word	**Danger**

Hazard statement(s)

AUH044	Risk of explosion if heated under confinement.
H222+H229	Extremely flammable aerosol. Pressurized container: may burst if heated.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H341	Suspected of causing genetic defects.
H360Df	May damage the unborn child. Suspected of damaging fertility.
H412	Harmful to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

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
67-64-1	10-30	acetone
123-86-4	10-30	n-butyl acetate
1330-20-7	<10	xylene
14807-96-6	<10	talc
13463-67-7	<10	titanium dioxide
64-17-5	<10	ethanol
1317-65-3	<10	limestone

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CAS No	%[weight]	Name
108-88-3	<10	toluene
85-68-7	<1	butyl benzyl phthalate
Not Available	balance	Ingredients determined not to be hazardous
115-10-6	30-60	dimethyl ether
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 aerosols come in contact with the eyes: ▸ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ 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 solids or aerosol mists are deposited upon the skin: ▸ Flush skin and hair with running water (and soap if available). ▸ Remove any adhering solids with industrial skin cleansing cream. ▸ **DO NOT** use solvents. ▸ Seek medical attention in the event of irritation.
Inhalation	If aerosols, fumes or combustion products are inhaled: ▸ Remove to fresh air. ▸ 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. ▸ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, 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	▸ Avoid giving milk or oils. ▸ Avoid giving alcohol. ▸ Not considered a normal route of entry.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
for lower alkyl ethers:

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.
- A low-stimulus environment must be maintained.
- Monitor and treat, where necessary, for shock.
- Anticipate and treat, where necessary, for seizures.
- **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.

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 without signs of hypovolaemia may require vasopressors.
- 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.
- Ethers may produce anion gap acidosis. Hyperventilation and bicarbonate therapy might be indicated.
- Haemodialysis might be considered in patients with impaired renal function.
- Consult a toxicologist as necessary.

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

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ADVANCED TREATMENT

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

EMERGENCY DEPARTMENT

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

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

For acute or short term repeated exposures to xylene:

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

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

SECTION 5 Firefighting measures

Extinguishing media

SMALL FIRE:

- Water spray, dry chemical or CO_2

LARGE FIRE:

- Water spray or fog.

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. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ If safe, switch off electrical equipment until vapour fire hazard removed. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ **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	▸ Liquid and vapour are highly flammable. ▸ Severe fire hazard when exposed to heat or flame. ▸ Vapour forms an explosive mixture with air. ▸ Severe explosion hazard, in the form of vapour, when exposed to flame or spark. ▸ Vapour may travel a considerable distance to source of ignition. ▸ Heating may cause expansion or decomposition with violent container rupture. ▸ Aerosol cans may explode on exposure to naked flames. ▸ Rupturing containers may rocket and scatter burning materials. ▸ Hazards may not be restricted to pressure effects. ▸ May emit acrid, poisonous or corrosive fumes. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO_2) hydrogen chloride phosgene hydrogen fluoride nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions.
HAZCHEM	Not Applicable

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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	▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Wear protective clothing, impervious gloves and safety glasses. ▸ Shut off all possible sources of ignition and increase ventilation. ▸ Wipe up. ▸ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▸ Undamaged cans should be gathered and stowed safely.
Major Spills	▸ Remove leaking cylinders to a safe place if possible. ▸ Release pressure under safe, controlled conditions by opening the valve. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water courses ▸ 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. ▸ Absorb or cover spill with sand, earth, inert materials or vermiculite. ▸ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. ▸ Undamaged cans should be gathered and stowed safely. ▸ Collect residues and seal in labelled drums for disposal.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ **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. ▸ **DO NOT** incinerate or puncture aerosol cans. ▸ **DO NOT** spray directly on humans, exposed food or food utensils. ▸ 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 are maintained.
Other information	▸ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can ▸ Store in original containers in approved flammable liquid storage area. ▸ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped. ▸ No smoking, naked lights, heat or ignition sources. ▸ Keep containers securely sealed. Contents under pressure. ▸ Store away from incompatible materials. ▸ Store in a cool, dry, well ventilated area. ▸ Avoid storage at temperatures higher than 40 deg C. ▸ Store in an upright position. ▸ Protect containers against physical damage. ▸ Check regularly for spills and leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Aerosol dispenser. ▸ Check that containers are clearly labelled.
Storage incompatibility	▸ Avoid strong acids, bases. ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	acetone	Acetone	500 ppm / 1185 mg/m3	2375 mg/m3 / 1000 ppm	Not Available	Not Available
Australia Exposure Standards	n-butyl acetate	n-Butyl acetate	150 ppm / 713 mg/m3	950 mg/m3 / 200 ppm	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	talc	Talc, (containing no asbestos fibres)	2.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ethanol	Ethyl alcohol	1000 ppm / 1880 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	limestone	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	toluene	Toluene	50 ppm / 191 mg/m3	574 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	dimethyl ether	Dimethyl ether	400 ppm / 760 mg/m3	950 mg/m3 / 500 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
acetone	Not Available	Not Available	Not Available
n-butyl acetate	Not Available	Not Available	Not Available
xylene	Not Available	Not Available	Not Available
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3
ethanol	Not Available	Not Available	15000* ppm
limestone	45 mg/m3	210 mg/m3	1,300 mg/m3
toluene	Not Available	Not Available	Not Available
butyl benzyl phthalate	15 mg/m3	77 mg/m3	460 mg/m3
dimethyl ether	3,000 ppm	3800* ppm	7200* ppm

Ingredient	Original IDLH	Revised IDLH
acetone	2,500 ppm	Not Available
n-butyl acetate	1,700 ppm	Not Available
xylene	900 ppm	Not Available
talc	1,000 mg/m3	Not Available
titanium dioxide	5,000 mg/m3	Not Available
ethanol	3,300 ppm	Not Available
limestone	Not Available	Not Available
toluene	500 ppm	Not Available
butyl benzyl phthalate	Not Available	Not Available
dimethyl ether	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
butyl benzyl phthalate	D	> 0.1 to ≤ 1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

For talc (a form of magnesium silicate):
Most health problems associated with occupational exposure to talcs appear to evolve mostly from the nonplatiform content of the talc being mined or milled (being the asbestos-like amphiboles, serpentines (asbestiformes) and other minerals in the form of acicular, prismatic and fibrous crystals including, possibly, asbestos).
Because of severe health effects associated with exposures to asbestos, regulatory agencies tend to regard all elongate mineral crystal particles, whether prismatic, acicular, fibrous, as asbestos - the only provision is the particles have an aspect ratio (length to diameter) of 3:1 or greater.
Consideration is also given to their respirability, their width being less than or equal to 3 um. Only limited data, however, exists on the health effects of elongate mineral particles having prismatic, acicular or fibrous (non-asbestos) forms. Experimental evidence indicates that the carcinogen potential of mineral fibres is related to the size class with diameter of 8 um with shorter, thicker particles having little biological activity.
Dust of nonfibrous talc, consisting entirely of platiform talc crystals and containing no asbestos poses a relatively small respiratory hazard.
Difficulties exist, however, in the determination of asbestos as cleavage fragments of prismatic or acicular crystals, nonasbestos fibres and asbestos fibres are very similar.
Subject to an accurate determination of asbestos and crystalline silica, exposure at or below the recommended TLV-TWA, is thought to protect workers from the significant risk of nonmalignant respiratory effects associated with talc dusts.
for dimethyl ether:
The no-effect-level for dimethyl ether is somewhere between 2000 ppm (rabbits) and 50,000 ppm (humans) with possible cardiac sensitisation occurring around 200,000 ppm (dogs). The AIHA has adopted a safety factor of 100 in respect to the 50,000 ppm level in its recommendation for a workplace environmental exposure level (WEEL) which is thought to protect against both narcotic and sensitising effects. This level is consistent with the TLV-TWA of 400 ppm for diethyl ether and should be easily achievable using current technologies. The use of the traditionally allowable excursion of 1.25 to the level of 6.25 ppm is felt to be more than adequate as an upper safe limit of exposure.
Human data:
50,000 ppm (12 mins): Feelings of mild intoxication.

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75,000 ppm (12 mins): As above plus slight lack of attenuation.
82,000 ppm (12 mins): Some incoordination, slight blurring of vision
(30 mins): As above plus analgesia of the face and rushing of blood to the face.
100,000 ppm (10-20 mins): Narcotic symptoms; (64 mins): Sickness (assumed to be nausea)
144,000 ppm (36 mins):Unconsciousness

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)
Saturation vapour concentration: 237000 ppm @ 20 C
NOTE: Detector tubes measuring in excess of 40 ppm, are available.
Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.
Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.
A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.
Odour Safety Factor(OSF)
OSF=38 (ACETONE)

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

Animals exposed by inhalation to 10 mg/m3 titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.
· The label on a package containing 1% or more of titanium oxide with aerodynamic diameter equal or below 10 microns shall bear the following statement: EUH211 "Warning! Hazardous respirable droplets may be formed when sprayed. Do NOT breathe spray or mist
· The label on the packaging of solid mixtures containing 1% or more of titanium dioxide shall bear the following statement: EUH212" "Warning! Hazardous respirable dust may be formed when used. Do not breathe dust".
In addition, the label on the packaging of liquid and solid mixtures not intended for the general public and not classified as hazardous which are labelled EUH211 or EU212 shall bear statement EUH210: "Safety data sheet available on request."
For toluene:
Odour Threshold Value: 0.16-6.7 (detection), 1.9-69 (recognition)
NOTE: Detector tubes measuring in excess of 5 ppm, are available.
High concentrations of toluene in the air produce depression of the central nervous system (CNS) in humans. Intentional toluene exposure (glue-sniffing) at maternally-intoxicating concentration has also produced birth defects. Foetotoxicity appears at levels associated with CNS narcosis and probably occurs only in those with chronic toluene-induced kidney failure. Exposure at or below the recommended TLV-TWA is thought to prevent transient headache and irritation, to provide a measure of safety for possible disturbances to human reproduction, the prevention of reductions in cognitive responses reported amongst humans inhaling greater than 40 ppm, and the significant risks of hepatotoxic, behavioural and nervous system effects (including impaired reaction time and incoordination). Although toluene/ethanol interactions are well recognised, the degree of protection afforded by the TLV-TWA among drinkers is not known.
Odour Safety Factor(OSF)
OSF=17 (TOLUENE)

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

For ethanol:
Odour Threshold Value: 49-716 ppm (detection), 101 ppm (recognition)
Eye and respiratory tract irritation do not appear to occur at exposure levels of less than 5000 ppm and the TLV-TWA is thought to provide an adequate margin of safety against such effects. Experiments in man show that inhalation of 1000 ppm caused slight symptoms of poisoning and 5000 ppm caused strong stupor and morbid sleepiness. Subjects exposed to 5000 ppm to 10000 ppm experienced smarting of the eyes and nose and coughing. Symptoms disappeared within minutes. Inhalation also causes local irritating effects to the eyes and upper respiratory tract, headaches, sensation of heat intraocular tension, stupor, fatigue and a need to sleep. At 15000 ppm there was continuous lachrymation and coughing.

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.

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.
Provide adequate ventilation in warehouse or closed storage areas.
Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Speed:
aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s

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	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion) 1-2.5 m/s (200-500 f/min.) Within each range the appropriate value depends on: Lower end of the range / Upper end of the range 1: Room air currents minimal or favourable to capture / 1: Disturbing room air currents 2: Contaminants of low toxicity or of nuisance value only. / 2: Contaminants of high toxicity 3: Intermittent, low production. / 3: High production, heavy use 4: Large hood or large air mass in motion / 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Individual protection measures, such as personal protective equipment	
Eye and face protection	▸ Safety glasses with side shields. ▸ 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]. ▸ Close fitting gas tight goggles **DO NOT** wear contact lenses. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens 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 1337.1, EN166 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	For esters: ▸ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. ▸ No special equipment needed when handling small quantities. ▸ **OTHERWISE:** ▸ For potentially moderate exposures: ▸ Wear general protective gloves, eg. light weight rubber gloves. ▸ For potentially heavy exposures: ▸ Wear chemical protective gloves, eg. PVC. and safety footwear.
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. **OTHERWISE:** ▸ Overalls. ▸ Skin cleansing cream. ▸ Eyewash unit. ▸ Do not spray on hot surfaces. ▸ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton. ▸ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. BRETHERICK: Handbook of Reactive Chemical Hazards.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
ColorSpec Plastic Primer, 400g (Colorspec Plastic Primer)

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C

Respiratory protection

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

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

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

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur

Continued...

NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/BUTYL	C
VITON/CHLOROBUTYL	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.

Ansell Glove Selection

Glove — *In order of recommendation*
AlphaTec® 38-612
BioClean™ Ultimate BUPS
AlphaTec® 58-005
BioClean™ N-Plus BNPS
MICROFLEX® MidKnight® XTRA 93-862
AlphaTec® 53-001
BioClean™ Emerald BENS
BioClean™ Extra BLAS
BioClean™ Fusion (Sterile) S-BFAP
MICROFLEX® LifeStar EC™ 93-868

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

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Thin grey highly flammable liquid with a strong solvent odour. Supplied as an aerosol pack. Contents under **PRESSURE**. Contains highly flammable ether propellant.

Physical state	Liquid	**Relative density (Water = 1)**	0.80-0.90
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	354
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	14-18 secs B4 Cup @25C
Initial boiling point and boiling range (°C)	-25 to 145	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	-41 (OC)	**Taste**	Not Available
Evaporation rate	Not Availa0.40-10.00 BuAC = 1	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	19	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	510 @20C	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (1%)**	Not Available

Continued...

Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Elevated temperatures. ▸ Presence of open flame. ▸ 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 may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur. Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body. Common, generalised symptoms associated with toxic gas inhalation include: ▸ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures; ▸ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest; ▸ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest; ▸ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain. Ethers produce narcosis following inhalation. Inhalation of lower alkyl ethers may result in central nervous system depression or stimulation, intoxication, headache, dizziness, weakness, blurred vision, seizures and possible coma. Cardiovascular involvement may produce hypotension, bradycardia and cardiovascular collapse, whilst respiratory symptoms might include irritation of nose and throat, cough, laryngeal spasm, pharyngitis, irregular respiration, depression, pulmonary oedema and respiratory arrest. Nausea, vomiting and salivation might also indicate overexposure. Convulsions, respiratory distress or paralysis, asphyxia, pneumonitis, and unconsciousness are all serious manifestations of poisoning. Fatalities have been reported. Kidney and liver damage with interstitial cystitis may result from massive exposures. Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. 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 **WARNING**:Intentional misuse by concentrating/inhaling contents may be lethal. Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue. Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression , headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures. Prolonged exposure may cause headache, nausea and ultimately loss of consciousness.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments 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.

Continued...

	Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Ingestion of alkyl ethers may produce symptoms similar to those produced following inhalation.
Skin Contact	Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. The material may produce moderate skin irritation; limited evidence or practical experience suggests, 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. Spray mist may produce discomfort Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression. Open cuts, abraded or irritated skin should not be exposed to this material
Eye	Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures.. Eye contact with alkyl ethers (vapours or liquid) may produce irritation, redness and lachrymation.
Chronic	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. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Principal route of occupational exposure to the gas is by inhalation. Chronic exposure to alkyl ethers may result in loss of appetite, excessive thirst, fatigue, and weight loss Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mix ed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms. Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers. Xylene has been classed as a developmental toxin in some jurisdictions. Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex. Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents. Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size. Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1). (1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996 On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Workers exposed to 700 ppm acetone for 3 hours/day for 7-15 years showed inflammation of the respiratory tract, stomach and duodenum, attacks of giddiness and loss of strength. Exposure to acetone may enhance liver toxicity of chlorinated solvents.

ColorSpec Plastic Primer, 400g (Colorspec Plastic Primer)	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

acetone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 20000 mg/kg[2]	Eye (human): 500 ppm - irritant
	Inhalation(Mouse) LC50; 44 mg/L4h[2]	Eye (rabbit): 20mg/24hr -moderate
	Oral (Rat) LD50: 5800 mg/kg[2]	Eye (rabbit): 3.95 mg - SEVERE
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 500 mg/24hr - mild
		Skin (rabbit):395mg (open) - mild
		Skin: no adverse effect observed (not irritating)[1]

n-butyl acetate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3200 mg/kg[2]	Eye (human): 300 mg * [PPG]

Continued...

	Inhalation(Rat) LC50: 0.74 mg/l4h[2]	Eye (rabbit): 20 mg (open)-SEVERE
	Oral (Rabbit) LD50; 3200 mg/kg[2]	Eye (rabbit): 20 mg/24h - moderate
		Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating)[1]
xylene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1700 mg/kg[2]	Eye (human): 200 ppm irritant
	Inhalation(Rat) LC50: 5000 ppm4h[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (Mouse) LD50; 2119 mg/kg[2]	Eye (rabbit): 87 mg mild
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):500 mg/24h moderate
		Skin: adverse effect observed (irritating)[1]
talc	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: >2.1 mg/l4h[1]	Skin (human): 0.3 mg/3d-I mild
	Oral (Rat) LD50: >5000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
titanium dioxide	**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): 0.3 mg /3D (int)-mild *
	Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
ethanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 17100 mg/kg[1]	Eye (rabbit): 500 mg SEVERE
	Inhalation(Rat) LC50: 64000 ppm4h[2]	Eye (rabbit):100mg/24hr-moderate
	Oral (Rat) LD50: 7060 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):20 mg/24hr-moderate
		Skin (rabbit):400 mg (open)-mild
		Skin: no adverse effect observed (not irritating)[1]
limestone	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 6450 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating)[1]
toluene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 12124 mg/kg[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation(Rat) LC50: >13350 ppm4h[2]	Eye (rabbit):0.87 mg - mild
	Oral (Rat) LD50: 636 mg/kg[2]	Eye (rabbit):100 mg/30sec - mild
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
butyl benzyl phthalate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >10000 mg/kg[2]	Not Available
	Inhalation(Rat) LC50: >6.7 mg/l4h[2]	
	Oral (Rat) LD50: 2330 mg/kg[2]	
dimethyl ether	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50: >20000 ppm4h[1]	Not Available
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*	

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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**
XYLENE	Reproductive effector in rats
TALC	For talc (a form of magnesium silicate) The overuse of talc in nursing infants has resulted in pulmonary oedema, pneumonia and death within hours of inhaling talcum powder.The powder dries the mucous membranes of the bronchioles, disrupts pulmonary clearance, clogs smaller airways. Victims display wheezing, rapid or difficult breathing, increased pulse, cyanosis, fever. Mild exposure may cause relatively minor inflammatory lung disease. Long term exposure may show wheezing, weakness, productive cough, limited chest expansion, scattered rales, cyanosis.
TITANIUM DIOXIDE	* IUCLID Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. **Animal carcinogenicity data** Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
LIMESTONE	Eye (rabbit) 0.75: mg/24h - No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
TOLUENE	For toluene: **Acute Toxicity** Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies. **Humans -** Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.

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	Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days. Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death Toluene can also strip the skin of lipids causing dermatitis **Animals -** The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days **Subchronic/Chronic Effects:** Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm. **Humans -** Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin. Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L **Animals -** The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) . **Developmental/Reproductive Toxicity** Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals. **Humans -** Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy **Animals** - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m3 toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m3 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m3 toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m3. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring. **Absorption** - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor. Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene . **Distribution** - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues . **Metabolism** - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites **Excretion -** Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.
BUTYL BENZYL PHTHALATE	Reproductive effector in rats. 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. for benzyl butyl phthalate: **Repeat dose toxicity:** The repeated-dose toxicity of BBP has been well investigated in studies, primarily in the rat, in which dose-response was well characterised. Effects observed consistently have been decreases in body weight gain (often accompanied by decreases in food consumption) and increases in organ to body weight ratios, particularly for the kidney and liver. Histopathological effects on the pancreas and kidney and haematological effects have also been observed. At higher doses, degenerative effects on the testes and, occasionally, histopathological effects on the liver have been reported. In specialised investigations, peroxisomal proliferation in the liver has been observed, although potency in this regard was less than that for other phthalates, such as bis(2-ethylhexyl) phthalate (DEHP). **Reproductive Toxicity and Teratology Studies** Groups of male F344/N rats given 20, 200, or 2200 mg/kg body weight butyl benzyl phthalate daily in feed for 10 weeks resulted in significantly decreased prostate gland, right cauda, right epididymis, and right testis weights at the highest dose versus those of the controls (NTP, 1997). Additionally, the epididymal spermatozoal concentrations in males given the 200 and 2200 mg/kg levels were significantly less than the controls. Females mated to 20 and 200 mg/kg males exhibited maternal body weights similar to those of females mated to control males. Litter data between the two dose groups and controls were also similar. Females mated to 2200 mg/kg males were initially found to be sperm positive; however, at necropsy, none of the females were pregnant. The fertility indices of the males and females were observed to be significantly lower than those of the controls. **Developmental Toxicity:** In several well-conducted studies in rats and mice, BBP has induced marked developmental effects, but only at dose levels that induce significant maternal toxicity. **Carcinogenicity Studies** In a 2-year study, groups of male F344/N rats were given 120, 240, or 500 mg/kg body weight butyl benzyl phthalate daily in feed and females were given 300, 600, or 1200 mg/kg/day (NTP, 1997). At the highest dose, the incidences of pancreatic acinar cell adenoma and adenoma or carcinoma (combined) were significantly greater in males than those in the controls. In females, the incidence of transitional epithelial hyperplasia was significantly greater than that in the controls. Specifically, two transitional epithelial papillomas in the urinary bladder were seen. It was concluded that there was "some evidence" of carcinogenicity in male rats, based on an increased incidence of pancreatic tumours, and equivocal evidence in female rats, based on marginal increases in pancreatic and bladder tumours. Dietary restriction prevented full expression of the pancreatic tumours and delayed appearance of the bladder tumours. There was no evidence of carcinogenicity in mice. **Genotoxicity Studies** At concentrations up to 11,550 ug/plate butyl benzyl phthalate in *Salmonella typhimurium* strains TA98, TA100, TA1535, and TA1537, no mutagenic response was obtained, in the presence or absence of metabolic activation (S9) (NTP, 1997). *In vitro* studies with L5178Y mouse lymphoma cells and cultured Chinese hamster ovary cells, both conducted with and without S9, were also negative. In germ cells of male *Drosophila melanogaster*, no induction of sex-linked recessive lethal mutations was observed. In contrast to these results, butyl benzyl phthalate gave positive responses in two *in vivo* mouse studies. In one experiment, sister chromatid exchanges were weakly positive at 23

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	and 42 hours. In the other study, chromosomal aberrations were induced in bone marrow cells 17 hours after intraperitoneal injection of 5000 mg/kg of the compound. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).
ColorSpec Plastic Primer, 400g (Colorspec Plastic Primer) & N-BUTYL ACETATE & XYLENE & LIMESTONE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ColorSpec Plastic Primer, 400g (Colorspec Plastic Primer) & N-BUTYL ACETATE & XYLENE & ETHANOL & LIMESTONE & TOLUENE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
ColorSpec Plastic Primer, 400g (Colorspec Plastic Primer) & ACETONE	For acetone: The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals. The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.
ACETONE & TITANIUM DIOXIDE	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.
XYLENE & TALC & BUTYL BENZYL PHTHALATE	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.
TALC & TITANIUM DIOXIDE	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. No significant acute toxicological data identified in literature search.

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

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

SECTION 12 Ecological information

Toxicity

ColorSpec Plastic Primer, 400g (Colorspec Plastic Primer)

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

acetone

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	3744.6-5000.7mg/L	4
NOEC(ECx)	12h	Fish	0.001mg/L	4
EC50	72h	Algae or other aquatic plants	5600-10000mg/l	4
EC50	48h	Crustacea	6098.4mg/L	5
EC50	96h	Algae or other aquatic plants	9.873-27.684mg/l	4

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n-butyl acetate

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

xylene

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

talc

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	7202.7mg/l	2
LC50	96h	Fish	89581.016mg/l	2
NOEC(ECx)	720h	Algae or other aquatic plants	918.089mg/l	2

titanium dioxide

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

ethanol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	275mg/l	2
EC50	48h	Crustacea	2mg/l	4
EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
LC50	96h	Fish	42mg/l	4
EC50(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	4

limestone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>14mg/l	2
NOEC(ECx)	1h	Fish	4-320mg/l	4
LC50	96h	Fish	>165200mg/L	4

toluene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	>376.71mg/L	4
EC50	72h	Algae or other aquatic plants	12.5mg/l	4
EC50	48h	Crustacea	3.78mg/L	5
LC50	96h	Fish	5-35mg/l	4
NOEC(ECx)	168h	Crustacea	0.74mg/L	5

butyl benzyl phthalate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.5mg/l	1
EC50	48h	Crustacea	0.97mg/l	1
EC50	96h	Algae or other aquatic plants	>2.69mg/l	1
ErC50	72h	Algae or other aquatic plants	0.325mg/L	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.1mg/l	1
LC50	96h	Fish	0.46-0.55mg/l	4

dimethyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>4400mg/L	2
EC50	96h	Algae or other aquatic plants	154.917mg/l	2
LC50	96h	Fish	1783.04mg/l	2
NOEC(ECx)	48h	Crustacea	>4000mg/l	1

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

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(Japan) - Bioconcentration Data 8. Vendor Data

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
For Aromatic Substances Series:
Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.
Atmospheric Fate: PAHs are 'semi-volatile substances" which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.
Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes >naphthalenes. Anthrcene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.
Most ethers are very resistant to hydrolysis, and the rate of cleavage of the carbon-oxygen bond by abiotic processes is expected to be insignificant.
Direct photolysis will not be an important removal process since aliphatic ethers do not absorb light at wavelengths >290 nm
For Xylenes:
log Koc : 2.05-3.08; Koc : 25.4-204; Half-life (hr) air : 0.24-42; Half-life (hr) H2O surface water : 24-672; Half-life (hr) H2O ground : 336-8640; Half-life (hr) soil : 52-672; Henry's Pa m3 /mol : 637-879; Henry's atm m3 /mol - 7.68E-03; BOD 5 if unstated - 1.4,1%; COD - 2.56,13% ThOD - 3.125 : BCF : 23; log BCF : 1.17-2.41.
Environmental Fate: Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. Soil - Xylenes are expected to have moderate mobility in soil evaporating rapidly from soil surfaces. The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. Xylene can remain below the soil surface for several days and may travel through the soil profile and enter groundwater. Soil and water microbes may transform it into other, less harmful compounds, although this happens slowly. It is not clear how long xylene remains trapped deep underground in soil or groundwater, but it may be months or years.
Atmospheric Fate: Xylene evaporates quickly into the air from surface soil and water and can remain in the air for several days until it is broken down by sunlight into other less harmful chemicals. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylene may contribute to photochemical smog formation. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzylnitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.
Aquatic Fate: p-xylene may adsorb to suspended solids and sediment in water and is expected to volatilise from water surfaces. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. Measurements taken from goldfish, eels and clams indicate that bioconcentration in aquatic organisms is low. Photo-oxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. p-Xylene is biodegradable and has been observed to degrade in pond water however; it is unclear if it degrades in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high. Ecotoxicity: Xylenes are slightly toxic to fathead minnow, rainbow trout and bluegill and not acutely toxic to water fleas. For Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/L. and Gammarus lacustris LC50 (48 h): 0.6 mg/L.
For Ketones: Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds.
Aquatic Fate: Hydrolysis of ketones in water is thermodynamically favourable only for low molecular weight ketones. Reactions with water are reversible with no permanent change in the structure of the ketone substrate. Ketones are stable to water under ambient environmental conditions. When pH levels are greater than 10, condensation reactions can occur which produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavourable. Based on its reactions in air, it seems likely that ketones undergo photolysis in water.
Terrestrial Fate: It is probable that ketones will be biodegraded by micro-organisms in soil and water.
Ecotoxicity: Ketones are unlikely to bioconcentrate or biomagnify.
DO NOT discharge into sewer or waterways.
For n-Butyl Acetate:
Koc: ~200;
log Kow: 1.78;
Half-life (hr) air: 144;
Half-life (hr) H2O surface water: 178 - 27156;
Henry's atm: m3 /mol: 3.20E-04
BOD 5 if unstated: 0.15-1.02,7%;
COD: 78%;
ThOD: 2.207;
BCF : 4-14.
Environmental Fate: Terrestrial Fate - Butyl acetate is expected to have moderate mobility in soil. Volatilization of n-butyl acetate is expected from moist and dry soil surfaces. n-Butyl acetate may biodegrade in soil. Aquatic Fate: n-Butyl acetate is not expected to adsorb to suspended solids and sediment in water. Butyl acetate is expected to volatilize from water surfaces. Estimated half-lives for a model river and model lake are 7 and 127 hours respectively. Hydrolysis may be an important environmental fate for this compound. Atmospheric Fate: n-Butyl acetate is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase n-butyl acetate is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 4 days.
Ecotoxicity: It is expected that bioconcentration in aquatic organisms is low. n-Butyl acetate is not acutely toxic to fish specifically, island silverside, bluegill sunfish, fathead minnow, and water fleas and has low toxicity to algae.
for acetone:
log Kow: -0.24
Half-life (hr) air: 312-1896
Half-life (hr) H2O surface water: 20
Henry's atm m3 /mol: 3.67E-05
BOD 5: 0.31-1.76,46-55%
COD: 1.12-2.07
ThOD: 2.2
BCF: 0.69
Environmental fate:
Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water
In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.
Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.
Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.
Acetone does not concentrate in the food chain.
Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period
Drinking Water Standard: none available.
Soil Guidelines: none available.
Air Quality Standards: none available.
Ecotoxicity:
Testing shows that acetone exhibits a low order of toxicity
Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l
Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l

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Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l
Aquatic invertebrate 2100 - 16700 mg/l
Aquatic plant NOEC: 5400-7500 mg/l
Daphnia magna chronic NOEC 1660 mg/l
Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephestia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m3. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.
The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
n-butyl acetate	LOW	LOW
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
titanium dioxide	HIGH	HIGH
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
butyl benzyl phthalate	HIGH (Half-life = 180 days)	LOW (Half-life = 2.5 days)
dimethyl ether	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
acetone	LOW (BCF = 0.69)
n-butyl acetate	LOW (BCF = 14)
xylene	MEDIUM (BCF = 740)
titanium dioxide	LOW (BCF = 10)
ethanol	LOW (LogKOW = -0.31)
toluene	LOW (BCF = 90)
butyl benzyl phthalate	MEDIUM (BCF = 663)
dimethyl ether	LOW (LogKOW = 0.1)

Mobility in soil

Ingredient	Mobility
acetone	HIGH (KOC = 1.981)
n-butyl acetate	LOW (KOC = 20.86)
titanium dioxide	LOW (KOC = 23.74)
ethanol	HIGH (KOC = 1)
toluene	LOW (KOC = 268)
butyl benzyl phthalate	LOW (KOC = 9359)
dimethyl ether	HIGH (KOC = 1.292)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Consult State Land Waste Management Authority for disposal. ▸ Discharge contents of damaged aerosol cans at an approved site. ▸ Allow small quantities to evaporate. ▸ **DO NOT** incinerate or puncture aerosol cans. ▸ Bury residues and emptied aerosol cans at an approved site.

Continued...

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG)

14.1. **UN number or ID number**	1950
14.2. **UN proper shipping name**	AEROSOLS
14.3. **Transport hazard class(es)**	Class: 2.1 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: 63 190 277 327 344 381 Limited quantity: 1000ml

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	1950
14.2. **UN proper shipping name**	Aerosols, flammable; Aerosols, flammable (engine starting fluid)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 2.1 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 10L
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: A145 A167 A802; A1 A145 A167 A802 Cargo Only Packing Instructions: 203 Cargo Only Maximum Qty / Pack: 150 kg Passenger and Cargo Packing Instructions: 203; Forbidden Passenger and Cargo Maximum Qty / Pack: 75 kg; Forbidden Passenger and Cargo Limited Quantity Packing Instructions: Y203; Forbidden Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G; Forbidden

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	1950
14.2. **UN proper shipping name**	AEROSOLS
14.3. **Transport hazard class(es)**	IMDG Class: 2.1 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	Not Applicable
14.5 **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	EMS Number: F-D, S-U Special provisions: 63 190 277 327 344 381 959 Limited Quantities: 1000 ml

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
acetone	Not Available

Continued...

Product name	Group
n-butyl acetate	Not Available
xylene	Not Available
talc	Not Available
titanium dioxide	Not Available
ethanol	Not Available
limestone	Not Available
toluene	Not Available
butyl benzyl phthalate	Not Available
dimethyl ether	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
acetone	Not Available
n-butyl acetate	Not Available
xylene	Not Available
talc	Not Available
titanium dioxide	Not Available
ethanol	Not Available
limestone	Not Available
toluene	Not Available
butyl benzyl phthalate	Not Available
dimethyl ether	Not Available

SECTION 15 Regulatory information

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

acetone is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

n-butyl acetate is found on the following regulatory lists

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

xylene is found on the following regulatory lists

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

talc 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 - Group 2B: Possibly carcinogenic to humans
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

titanium dioxide 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 - Group 2B: Possibly carcinogenic to humans
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ethanol is found on the following regulatory lists

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

limestone 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)

toluene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Chemical Footprint Project - Chemicals of High Concern List
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 - Not Classified as Carcinogenic

butyl benzyl phthalate is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

dimethyl ether 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)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (acetone; n-butyl acetate; xylene; talc; ethanol; toluene; butyl benzyl phthalate; dimethyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	15/01/2014

SDS Version Summary

Version	Date of Update	Sections Updated
5.1	10/12/2021	Classification change due to full database hazard calculation/update.
6.1	10/03/2023	Classification change due to full database hazard calculation/update.

Other information

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

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

Definitions and abbreviations

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

Continued...

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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