COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY

Formula Marketing Limited

Chemwatch Hazard Alert Code: **4**

Version No: **2.2**
Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Issue Date: **15/07/2022**
Print Date: **18/07/2022**
L.GHS.NZL.EN

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

Product Identifier

Product name	COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY
Chemical Name	Not Applicable
Synonyms	CPA1792; CPS402
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	Application is by spray atomisation from a hand held aerosol pack

Details of the supplier of the safety data sheet

Registered company name	**Formula Marketing Limited**
Address	60B Cryers Road, East Tamaki Auckland 2013 New Zealand
Telephone	09 273 3600
Fax	Not Available
Website	www.formula.co.nz
Email	sales@formula.co.nz

Emergency telephone number

Association / Organisation	**NZ Poison Centre**
Emergency telephone numbers	0800 764 766
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation.
Classified as Dangerous Goods for transport purposes.

ChemWatch Hazard Ratings

		Min	Max
Flammability	4		
Toxicity	1		
Body Contact	3		
Reactivity	0		
Chronic	3		

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

Classification [1]	Hazardous to the Aquatic Environment Long-Term Hazard Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Serious Eye Damage/Eye Irritation Category 1, Reproductive Toxicity Category 2, Carcinogenicity Category 2, Aerosols Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*
Determined by Chemwatch using GHS/HSNO criteria	2.1.2A, 8.3A, 6.7B, 6.8B, 6.9B, 9.1B

Label elements

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

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Hazard statement(s)

H411	Toxic to aquatic life with long lasting effects.
H373	May cause damage to organs through prolonged or repeated exposure.
H318	Causes serious eye damage.
H361	Suspected of damaging fertility or the unborn child.
H351	Suspected of causing cancer.
H222+H229	Extremely flammable aerosol. Pressurized container: may burst if heated.

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.
P260	Do not breathe dust/fume.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.

Precautionary statement(s) Response

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.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P391	Collect spillage.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.

Precautionary statement(s) Disposal

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

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
67-64-1	20-35	acetone
123-86-4	8-15	n-butyl acetate
78-93-3	1-5	methyl ethyl ketone
108-88-3	1-5	toluene
763-69-9	1-5	ethyl-3-ethoxypropionate
1330-20-7	1-5	xylene
71-36-3	1-5	n-butanol
67-63-0	1-5	isopropanol
7779-90-0	1-5	zinc phosphate
1314-13-2	1-5	zinc oxide
108-65-6	1-5	2-Methoxy-1-methylethyl acetate
100-41-4	<1	ethylbenzene
106-97-8.	12-20	butane
74-98-6	3-10	propane
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 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.

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	▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▸ Generally not applicable.
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. ▸ Generally not applicable.
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. ▸ Generally not applicable.
Ingestion	▸ Not considered a normal route of entry. ▸ Generally not applicable. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:
- Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 50 mm Hg) 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.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

Treat symptomatically.
To treat poisoning by the higher aliphatic alcohols (up to C7):
- Gastric lavage with copious amounts of water.
- It may be beneficial to instill 60 ml of mineral oil into the stomach.
- Oxygen and artificial respiration as needed.
- Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5)

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 shock.
- Monitor and treat, where necessary, for pulmonary oedema.
- 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.
- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- 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.
- Acidosis may respond to hyperventilation and bicarbonate therapy.
- Haemodialysis might be considered in patients with severe intoxication.
- Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.
Symptomatic and supportive therapy is advised in managing patients.
For acute or short term repeated exposures to acetone:
- Symptoms of acetone exposure approximate ethanol intoxication.
- About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.

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- There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]
Management:
Measurement of serum and urine acetone concentrations may be useful to monitor the severity of ingestion or inhalation.
Inhalation Management:
- Maintain a clear airway, give humidified oxygen and ventilate if necessary.
- If respiratory irritation occurs, assess respiratory function and, if necessary, perform chest X-rays to check for chemical pneumonitis.
- Consider the use of steroids to reduce the inflammatory response.
- Treat pulmonary oedema with PEEP or CPAP ventilation.

Dermal Management:
- Remove any remaining contaminated clothing, place in double sealed, clear bags, label and store in secure area away from patients and staff.
- Irrigate with copious amounts of water.
- An emollient may be required.

Eye Management:
- Irrigate thoroughly with running water or saline for 15 minutes.
- Stain with fluorescein and refer to an ophthalmologist if there is any uptake of the stain.

Oral Management:
- No **GASTRIC LAVAGE OR EMETIC**
- Encourage oral fluids.

Systemic Management:
- Monitor blood glucose and arterial pH.
- Ventilate if respiratory depression occurs.
- If patient unconscious, monitor renal function.
- Symptomatic and supportive care.

The Chemical Incident Management Handbook:
Guy's and St. Thomas' Hospital Trust, 2000
BIOLOGICAL EXPOSURE INDEX
These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Sampling Time	Index	Comments
Acetone in urine	End of shift	50 mg/L	NS

NS: Non-specific determinant; also observed after exposure to other material
for simple esters:

BASIC TREATMENT

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

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
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- 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

SECTION 5 Firefighting measures

Extinguishing media

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

SMALL FIRE:
- Water spray, dry chemical or CO2

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.

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	▸ 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. Slight hazard when exposed to heat, flame and oxidisers.
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 monoxide (CO) , carbon dioxide (CO2) , metal oxides , other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions. Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place. Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard. **WARNING**: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.

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	▸ Clear area of all unprotected personnel and move upwind. ▸ Alert Emergency Authority and advise them of the location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear full body clothing with breathing apparatus. ▸ Prevent by any means available, spillage from entering drains and water-courses. ▸ Consider evacuation. ▸ Shut off all possible sources of ignition and increase ventilation. ▸ No smoking or naked lights within area. ▸ Use extreme caution to prevent violent reaction. ▸ Stop leak only if safe to so do. ▸ Water spray or fog may be used to disperse vapour. ▸ **DO NOT** enter confined space where gas may have collected. ▸ Keep area clear until gas has dispersed. ▸ Remove leaking cylinders to a safe place. ▸ Fit vent pipes. Release pressure under safe, controlled conditions ▸ Burn issuing gas at vent pipes. ▸ **DO NOT** exert excessive pressure on valve; **DO NOT** attempt to operate damaged 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.

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	▸ Clean up all spills immediately. ▸ Wear protective clothing, safety glasses, dust mask, gloves. ▸ Secure load if safe to do so. Bundle/collect recoverable product. ▸ Use dry clean up procedures and avoid generating dust. ▸ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▸ Water may be used to prevent dusting. ▸ Collect remaining material in containers with covers for disposal. ▸ Flush spill area with water.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	Natural gases contain a contaminant, radon-222, a naturally occurring radioactive gas. During subsequent processing, radon tends to concentrate in liquefied petroleum streams and in product streams having similar boiling points. Industry experience indicates that the commercial product may contain small amounts of radon-222 and its radioactive decay products (radon daughters). The actual concentration of radon-222 and radioactive daughters in process equipment (IE lines, filters, pumps and reactor units) may reach significant levels and produce potentially damaging levels of gamma radiation. A potential external radiation hazard exists at or near any pipe, valve or vessel containing a radon enriched stream or containing internal deposits of radioactive material. Field studies, however, have not shown that conditions exist that expose the worker to cumulative exposures in excess of general population limits. Equipment containing gamma-emitting decay products should be presumed to be internally contaminated with alpha-emitting decay products which may be hazardous if inhaled or ingested. During maintenance operations that require the opening of contaminated process equipment, the flow of gas should be stopped and a four hour delay enforced to allow gamma-radiation to drop to background levels. Protective equipment (including high efficiency particulate respirators (P3) suitable for radionucleotides or supplied air) should be worn by personnel entering a vessel or working on contaminated process equipment to prevent skin contamination or inhalation of any residue containing alpha-radiation. Airborne contamination may be minimised by handling scale and/or contaminated materials in a wet state. [*TEXACO*] The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid. ▸ 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. ▸ Store away from incompatible materials.

Conditions for safe storage, including any incompatibilities

Suitable container	Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler. ▸ Aerosol dispenser. ▸ Check that containers are clearly labelled.
Storage incompatibility	n-Butyl acetate: ▸ reacts with water on standing to form acetic acid and n-butyl alcohol ▸ reacts violently with strong oxidisers and potassium tert-butoxide ▸ is incompatible with caustics, strong acids and nitrates ▸ dissolves rubber, many plastics, resins and some coatings Xylenes: ▸ may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride ▸ attack some plastics, rubber and coatings ▸ may generate electrostatic charges on flow or agitation due to low conductivity. ▸ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ▸ Aromatics can react exothermically with bases and with diazo compounds. For alkyl aromatics: The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.

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- Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen
- Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.
- Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.
- Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.
- Alkali metals accelerate the oxidation while CO2 as co-oxidant enhances the selectivity.
- Microwave conditions give improved yields of the oxidation products.
- Photo-oxidation products may occur following reaction with hydroxyl radicals and NOx - these may be components of photochemical smogs.

Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007

Acetone:

- may react violently with chloroform, activated charcoal, aliphatic amines, bromine, bromine trifluoride, chlorotriazine, chromic(IV) acid, chromic(VI) acid, chromium trioxide, chromyl chloride, hexachloromelamine, iodine heptafluoride, iodoform, liquid oxygen, nitrosyl chloride, nitrosyl perchlorate, nitryl perchlorate, perchloromelamine, peroxomonosulfuric acid, platinum, potassium tert-butoxide, strong acids, sulfur dichloride, trichloromelamine, xenon tetrafluoride
- reacts violently with bromoform and chloroform in the presence of alkalies or in contact with alkaline surfaces.
- may form unstable and explosive peroxides in contact with strong oxidisers, fluorine, hydrogen peroxide (90%), sodium perchlorate, 2-methyl-1,3-butadiene
- can increase the explosive sensitivity of nitromethane on contact flow or agitation may generate electrostatic charges due to low conductivity
- dissolves or attacks most rubber, resins, and plastics (polyethylenes, polyester, vinyl ester, PVC, Neoprene, Viton)

Alcohols

- are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents.
- reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen
- react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium
- should not be heated above 49 deg. C. when in contact with aluminium equipment

Butane/ isobutane

- reacts violently with strong oxidisers
- reacts with acetylene, halogens and nitrous oxides
- is incompatible with chlorine dioxide, conc. nitric acid and some plastics
- may generate electrostatic charges, due to low conductivity, in flow or when agitated - these may ignite the vapour.

Segregate from nickel carbonyl in the presence of oxygen, heat (20-40 C)

- Esters react with acids to liberate heat along with alcohols and acids.
- Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products.
- Heat is also generated by the interaction of esters with caustic solutions.
- Flammable hydrogen is generated by mixing esters with alkali metals and hydrides.
- Esters may be incompatible with aliphatic amines and nitrates.

Ketones in this group:

- are reactive with many acids and bases liberating heat and flammable gases (e.g., H2).
- react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H2) and heat.
- are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides.
- react violently with aldehydes, HNO3 (nitric acid), HNO3 + H2O2 (mixture of nitric acid and hydrogen peroxide), and HClO4 (perchloric acid).
- may react with hydrogen peroxide to form unstable peroxides; many are heat- and shock-sensitive explosives.

A significant property of most ketones is that the hydrogen atoms on the carbons next to the carbonyl group are relatively acidic when compared to hydrogen atoms in typical hydrocarbons. Under strongly basic conditions these hydrogen atoms may be abstracted to form an enolate anion. This property allows ketones, especially methyl ketones, to participate in condensation reactions with other ketones and aldehydes. This type of condensation reaction is favoured by high substrate concentrations and high pH (greater than 1 wt% NaOH).

Propane:

- reacts violently with strong oxidisers, barium peroxide, chlorine dioxide, dichlorine oxide, fluorine etc.
- liquid attacks some plastics, rubber and coatings
- may accumulate static charges which may ignite its vapours
- Avoid strong acids, bases.
- Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	acetone	Acetone	500 ppm / 1185 mg/m3	2375 mg/m3 / 1000 ppm	Not Available	(bio)-Exposure can also be estimated by biological monitoring.
New Zealand Workplace Exposure Standards (WES)	n-butyl acetate	n-Butyl acetate	150 ppm / 713 mg/m3	950 mg/m3 / 200 ppm	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	methyl ethyl ketone	MEK (Methyl ethyl ketone, 2-Butanone)	150 ppm / 445 mg/m3	890 mg/m3 / 300 ppm	Not Available	(bio)-Exposure can also be estimated by biological monitoring.
New Zealand Workplace Exposure Standards (WES)	toluene	Toluene (Toluol)	50 ppm / 188 mg/m3	Not Available	Not Available	(skin)-Skin absorption
New Zealand Workplace Exposure Standards (WES)	xylene	Dimethylbenzene	50 ppm / 217 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	n-butanol	n-Butyl alcohol	Not Available	Not Available	50 ppm / 150 mg/m3	(skin)-Skin absorption
New Zealand Workplace Exposure Standards (WES)	isopropanol	Isopropyl alcohol	400 ppm / 983 mg/m3	1230 mg/m3 / 500 ppm	Not Available	Not Available

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	zinc phosphate	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	zinc phosphate	Particulates not otherwise classified respirable dust	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	zinc phosphate	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	zinc phosphate	Particulates not otherwise classified	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	zinc oxide	Zinc oxide fume respirable dust	3 mg/m3	10 mg/m3	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	zinc oxide	Zinc oxide Dust respirable dust	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	2-Methoxy-1-methylethyl acetate	Propylene glycol monomethyl ether	100 ppm / 369 mg/m3	553 mg/m3 / 150 ppm	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	ethylbenzene	Ethyl benzene	100 ppm / 434 mg/m3	543 mg/m3 / 125 ppm	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	butane	Butane	800 ppm / 1900 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	propane	Propane	Not Available	Not Available	Not Available	Simple asphyxiant - may present an explosion hazard

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
methyl ethyl ketone	Not Available	Not Available	Not Available
toluene	Not Available	Not Available	Not Available
ethyl-3-ethoxypropionate	1.6 ppm	18 ppm	110 ppm
xylene	Not Available	Not Available	Not Available
n-butanol	60 ppm	800 ppm	8000** ppm
isopropanol	400 ppm	2000* ppm	12000** ppm
zinc phosphate	12 mg/m3	36 mg/m3	220 mg/m3
zinc oxide	10 mg/m3	15 mg/m3	2,500 mg/m3
2-Methoxy-1-methylethyl acetate	100 ppm	160 ppm	660 ppm
2-Methoxy-1-methylethyl acetate	Not Available	Not Available	Not Available
ethylbenzene	Not Available	Not Available	Not Available
butane	Not Available	Not Available	Not Available
propane	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
acetone	2,500 ppm	Not Available
n-butyl acetate	1,700 ppm	Not Available
methyl ethyl ketone	3,000 ppm	Not Available
toluene	500 ppm	Not Available
ethyl-3-ethoxypropionate	Not Available	Not Available
xylene	900 ppm	Not Available
n-butanol	1,400 ppm	Not Available
isopropanol	2,000 ppm	Not Available
zinc phosphate	Not Available	Not Available
zinc oxide	500 mg/m3	Not Available
2-Methoxy-1-methylethyl acetate	Not Available	Not Available
ethylbenzene	800 ppm	Not Available
butane	Not Available	1,600 ppm
propane	2,100 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
ethyl-3-ethoxypropionate	E	≤ 0.1 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

IFRA Prohibited Fragrance Substance

Continued...

The International Fragrance Association (IFRA) Standards form the basis for the globally accepted and recognized risk management system for the safe use of fragrance ingredients and are part of the IFRA Code of Practice. This is the self-regulating system of the industry, based on risk assessments carried out by an independent Expert Panel
for zinc oxide:
Zinc oxide intoxication (intoxication zincale) is characterised by general depression, shivering, headache, thirst, colic and diarrhoea.
Exposure to the fume may produce metal fume fever characterised by chills, muscular pain, nausea and vomiting. Short-term studies with guinea pigs show pulmonary function changes and morphologic evidence of small airway inflammation. A no-observed-adverse-effect level (NOAEL) in guinea pigs was 2.7 mg/m3 zinc oxide. Based on present data, the current TLV-TWA may be inadequate to protect exposed workers although known physiological differences in the guinea pig make it more susceptible to functional impairment of the airways than humans.
These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:
D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

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)

For butane:
Odour Threshold Value: 2591 ppm (recognition)
Butane in common with other homologues in the straight chain saturated aliphatic hydrocarbon series is not characterised by its toxicity but by its narcosis-inducing effects at high concentrations. The TLV is based on analogy with pentane by comparing their lower explosive limits in air. It is concluded that this limit will protect workers against the significant risk of drowsiness and other narcotic effects.
Odour Safety Factor(OSF)
OSF=0.22 (n-BUTANE)

for propylene glycol monomethyl ether (PGME)
Odour Threshold: 10 ppm.
The TLV-TWA is protective against discomfort caused by odour, against eye and skin irritation, and chronic effects (including possible liver and kidney damage).
Individuals exposed to 100 ppm reported a transient unpleasant odour with slight eye irritation after about 1 or 2 hours. At 300 ppm, mild irritation of the eyes and nose developed within 5 minutes; some individuals found the irritation hardly bearable after about an hour. A concentration of 750 ppm was highly irritating. Signs of central nervous system depression developed at 1000 ppm. Neurological, clinical chemical and general medical examinations showed no other conspicuous toxicity.
Concentrations of the beta-isomer, 2-methoxy-1-propyl acetate are low in commercial grades of PGME and teratogenic effects associated with this isomer are expected to be absent.
Odour Safety Factor(OSF)
OSF=10 (propylene glycol monomethyl ether)
For methyl ethyl ketone:
Odour Threshold Value: Variously reported as 2 ppm and 4.8 ppm
Odour threshold: 2 ppm (detection); 5 ppm (recognition) 25 ppm (easy recognition); 300 ppm IRRITATING
Exposures at or below the recommended TLV-TWA are thought to prevent injurious systemic effects and to minimise objections to odour and irritation. Where synergism or potentiation may occur stringent control of the primary toxin (e.g. n-hexane or methyl butyl ketone) is desirable and additional consideration should be given to lowering MEK exposures.
Odour Safety Factor(OSF)
OSF=28 (METHYL ETHYL KETONE)

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.

Continued...

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 n-butanol:
Odour Threshold Value: 0.12-3.4 ppm (detection), 1.0-3.5 ppm (recognition)
NOTE: Detector tubes for n-butanol, measuring in excess of 5 ppm are commercially available.
Exposure at or below the TLV-TWA is thought to provide protection against hearing loss due to vestibular and auditory nerve damage in younger workers and to protect against the significant risk of headache and irritation.
25 ppm may produce mild irritation of the respiratory tract 50 ppm may produce headache and vertigo.
Higher concentrations may produce marked irritation, sore throat, coughing, nausea, shortness of breath, pulmonary injury and central nervous system depression characterised by headache, dizziness, dullness and drowsiness.
6000 ppm may produce giddiness, prostration, narcosis, ataxia, and death.
Odour Safety Factor (OSF)
OSF=60 (n-BUTANOL)

Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)
Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol
for ethyl benzene:
Odour Threshold Value: 0.46-0.60 ppm
NOTE: Detector tubes for ethylbenzene, measuring in excess of 30 ppm, are commercially available.
Ethyl benzene produces irritation of the skin and mucous membranes and appears to produce acute and chronic effects on the central nervous system. Animal experiments also suggest the effects of chronic exposure include damage to the liver, kidneys and testes. In spite of structural similarities to benzene, the material does not appear to cause damage to the haemopoietic system. The TLV-TWA is thought to be protective against skin and eye irritation. Exposure at this concentration probably will not result in systemic effects.
Subjects exposed at 200 ppm experienced transient irritation of the eyes; at 1000 ppm there was eye irritation with profuse lachrymation; at 2000 ppm eye irritation and lachrymation were immediate and severe accompanied by moderate nasal irritation, constriction in the chest and vertigo; at 5000 ppm exposure produced intolerable irritation of the eyes and throat.
Odour Safety Factor(OSF)
OSF=43 (ETHYL BENZENE)

For propane
Odour Safety Factor(OSF)
OSF=0.16 (PROPANE)

Exposure controls

Appropriate engineering controls

Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use.
Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.
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
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.

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Personal protection	
Eye and face protection	▸ 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 1336 or national equivalent] No special equipment for minor exposure i.e. when handling small quantities. **OTHERWISE:** For potentially moderate or heavy exposures: ▸ Safety glasses with side shields. ▸ **NOTE:** Contact lenses pose a special hazard; soft lenses may absorb irritants and **ALL** lenses concentrate them. No special equipment required due to the physical form of the product. ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	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. No special equipment required due to the physical form of the product.
Body protection	See Other protection below
Other protection	▸ 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. No special equipment needed when handling small quantities. **OTHERWISE:** ▸ Overalls. ▸ Skin cleansing cream. ▸ Eyewash unit. ▸ Do not spray on hot surfaces. No special equipment required due to the physical form of the product.

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:
COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C

Respiratory protection

Type AX 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	-	AX-PAPR-AUS / Class 1
up to 50 x ES	-	AX-AUS / Class 1	-
up to 100 x ES	-	AX-2	AX-PAPR-2 ^

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

Respiratory protection not normally required due to the physical form of the product.
▸ Generally not applicable.
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.

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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.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Grey,Aerosol

Physical state	article	**Relative density (Water = 1)**	0.8
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	431
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	-81	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	10	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1.5	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (Not Available%)**	Not Available
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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as

Continued...

	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. No health effects were seen in humans exposed at 1,000 ppm isobutane for up to 8 hours or 500 ppm for 8 hours/day for 10 days. Isobutane can have anaesthetic and asphyxiant effects at high concentrations, well above the lower explosion limit of 1.8% (18,000 ppm). Butane is a simple asphyxiant and is mildly anaesthetic at high concentrations (20-25%). 10000 ppm for 10 minutes causes drowsiness. Narcotic effects may be accompanied by exhilaration, dizziness, headache, nausea, confusion, incoordination and unconsciousness in severe cases The paraffin gases C1-4 are practically nontoxic below the lower flammability limit, 18,000 to 50,000 ppm; above this, low to moderate incidental effects such as CNS depression and irritation occur, but are completely reversible upon cessation of the exposure. Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well. 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 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. 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. 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. **WARNING**:Intentional misuse by concentrating/inhaling contents may be lethal. The odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal. 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. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms. Systemic effects of acetone inhalation exposure include central nervous system depression, light-headedness, incoherent speech, ataxia, stupor, hypotension, tachycardia, metabolic acidosis, hyperglycaemia and ketosis. Rarely, convulsions and tubular necrosis may be evident. Other symptoms of exposure may include restlessness, headache, vomiting, low blood-pressure and rapid and irregular pulse, eye and throat irritation, weakness of the legs and dizziness. Inhalation of high concentrations may produce dryness of the mouth and throat, nausea, uncoordinated movement, loss of coordinated speech, drowsiness and, in severe cases, coma. Inhalation of acetone vapours over long periods causes irritation of the respiratory tract, coughing and headache. Rats exposed to 52200 ppm vapour for 1 hour showed clear signs of narcosis; fatalities occurred at 126600 ppm.
Ingestion	Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased. Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema. Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in he case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments Swallowing 10 millilitres of isopropanol may cause serious injury; 100 millilitres may be fatal if not properly treated. The adult single lethal dose is

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	approximately 250 millilitres. Isopropanol is twice as poisonous as ethanol, and the effects caused are similar, except that isopropanol does not cause an initial feeling of well-being. Swallowing may cause nausea, vomiting and diarrhea; vomiting and stomach inflammation is more prominent with isopropanol than with ethanol. Animals given near-lethal doses also showed inco-ordination, lethargy, inactivity and loss of consciousness. There is evidence that a slight tolerance to isopropanol may be acquired. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Accidental ingestion of the material may be damaging to the health of the individual.
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. Spray mist may produce discomfort Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. 511ipa The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: ▸ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non allergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. 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.. Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage. Eye contact may cause tearing or blurring of vision.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility 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. 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, or repeated exposure of isopropanol may cause inco-ordination and tiredness. Repeated inhalation exposure to isopropanol may produce sleepiness, inco-ordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in adult animals. Isopropanol does not cause genetic damage. There are inconclusive reports of human sensitisation from skin contacts with isopropanol. Chronic alcoholics are more tolerant of the whole-body effects of isopropanol. Animal testing showed the chronic exposure did not produce reproductive effects. NOTE: Commercial isopropanol does not contain "isopropyl oil", which caused an excess incidence of sinus and throat cancers in isoproanol production workers in the past. "Isopropyl oil" is no longer formed during production of isopropanol. 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.

COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY	**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

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		Skin: no adverse effect observed (not irritating)[1]
n-butyl acetate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3200 mg/kg[2]	Eye (human): 300 mg
	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]
methyl ethyl ketone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 6480 mg/kg[2]	Eye (human): 350 ppm -irritant
	Inhalation(Mouse) LC50; 32 mg/L4h[2]	Eye (rabbit): 80 mg - irritant
	Oral (Rat) LD50; 2054 mg/kg[1]	Skin (rabbit): 402 mg/24 hr - mild
		Skin (rabbit):13.78mg/24 hr open
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]
ethyl-3-ethoxypropionate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4076 mg/kg[2]	Eye (rabbit): 500mg/24h - mild
	Inhalation(Rat) LC50; 1250 ppm4h[2]	Skin (rabbit):10 mg/24h open mild
	Oral (Rat) LD50; ~3200-5000 mg/kg[2]	
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]
n-butanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3400 mg/kg[2]	Eye (human): 50 ppm - irritant
	Inhalation(Rat) LC50; 8000 ppm4h[2]	Eye (rabbit): 1.6 mg-SEVERE
	Oral (Rat) LD50; 790 mg/kg[2]	Eye (rabbit): 24 mg/24h-SEVERE
		Eye: adverse effect observed (irreversible damage)[1]
		Skin (rabbit): 405 mg/24h-moderate
		Skin: adverse effect observed (irritating)[1]
isopropanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 12800 mg/kg[2]	Eye (rabbit): 10 mg - moderate
	Inhalation(Mouse) LC50; 53 mg/L4h[2]	Eye (rabbit): 100 mg - SEVERE
	Oral (Mouse) LD50; 3600 mg/kg[2]	Eye (rabbit): 100mg/24hr-moderate
		Skin (rabbit): 500 mg - mild
zinc phosphate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; >5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]

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zinc oxide	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) : 500 mg/24 h - mild
	Inhalation(Rat) LC50; >1.79 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg[1]	Skin (rabbit) : 500 mg/24 h- mild
		Skin: no adverse effect observed (not irritating)[1]
2-Methoxy-1-methylethyl acetate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) 230 mg mild
	Oral (Rat) LD50; 3739 mg/kg[2]	Eye (rabbit) 500 mg/24 h. - mild
		Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit) 500 mg open - mild
		Skin: no adverse effect observed (not irritating)[1]
ethylbenzene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 17800 mg/kg[2]	Eye (rabbit): 500 mg - SEVERE
	Inhalation(Rat) LC50; 17.2 mg/l4h[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; 3500 mg/kg[2]	Skin (rabbit): 15 mg/24h mild
		Skin: no adverse effect observed (not irritating)[1]
butane	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; 658 mg/l4h[2]	Not Available
propane	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >13023 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*	

COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY	Data demonstrate that during inhalation exposure,aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure. Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism. The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.
METHYL ETHYL KETONE	Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities. Combinations with chloroform also show increase in toxicity
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. 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

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	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.
ETHYL-3-ETHOXYPROPIONATE	* Union Carbide ** Endura Manufacturing
XYLENE	Reproductive effector in rats
N-BUTANOL	for n-butanol **Acute toxicity:** n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC0 of 8000 ppm (24000 mg/m3) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA s localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser. The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed. **Repeat dose toxicity:** An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAc) to BA. Hydrolysis of BAc in blood and brain was estimated to be 99 percent complete within 2.7 minutes (elimination t1/2 = 0.41 minute). Thus, organisms exposed to BAc can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAc can be used as supplemental, surrogate data to provide information on the toxicity of BA. A thirteen-week, subchronic exposure to BAc, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m3) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOAEL) of 500 ppm (2395 mg/m3) was reported for systemic effects in rats, and a NOAEL of 3000 ppm (14370 mg/m3) was reported for post exposure neurotoxicity in rats. **Reproductive toxicity:** Several studies indicate that BA is not a reproductive toxicant. Female rats exposed to 6000 ppm (18000 mg/m3) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m3) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity. **Developmental toxicity:** BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m3) throughout gestation. **Genotoxicity:** An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic. **Carcinogenicity:** Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.
ISOPROPANOL	For isopropanol (IPA): **Acute toxicity:** Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat. Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred. **Repeat dose studies**: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney. **Reproductive toxicity**: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful. **Developmental toxicity:** The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity **Genotoxicity:** All genotoxicity assays reported for isopropanol have been negative **Carcinogenicity:** rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for

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	interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment
2-METHOXY-1-METHYLETHYL ACETATE	NOTE: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Fetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ETHYLBENZENE	Liver changes, utheral tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded. Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylgloxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances. Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys. Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures *(400 ppm and greater)* of ethylbenzene In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncitial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland. In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY & METHYL ETHYL KETONE & N-BUTANOL & ISOPROPANOL & 2-METHOXY-1-METHYLETHYL ACETATE	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.
COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY & 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**
COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY & 2-METHOXY-1-METHYLETHYL ACETATE	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is

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	>2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.
COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY & 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 & ETHYL-3-ETHOXYPROPIONATE & ISOPROPANOL & ZINC OXIDE & 2-METHOXY-1-METHYLETHYL ACETATE & ETHYLBENZENE	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.
N-BUTYL ACETATE & XYLENE & N-BUTANOL & ETHYLBENZENE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
N-BUTYL ACETATE & METHYL ETHYL KETONE & TOLUENE & XYLENE & N-BUTANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
XYLENE & ISOPROPANOL	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.
2-METHOXY-1-METHYLETHYL ACETATE & PROPANE	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

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COLORPAK PRO SERIES AEROSOL PRIMER SURFACER GREY

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
NOEC(ECx)	12h	Fish	0.001mg/L	4
EC50	48h	Crustacea	6098.4mg/L	5
EC50	96h	Algae or other aquatic plants	9.873-27.684mg/l	4
LC50	96h	Fish	3744.6-5000.7mg/L	4

n-butyl acetate

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

methyl ethyl ketone

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	48h	Crustacea	68mg/l	2
EC50	72h	Algae or other aquatic plants	1972mg/l	2
EC50	48h	Crustacea	308mg/l	2
EC50	96h	Algae or other aquatic plants	>500mg/l	4
LC50	96h	Fish	>324mg/L	4

toluene

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

ethyl-3-ethoxypropionate

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

xylene

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

n-butanol

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

isopropanol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>1000mg/l	1
EC50(ECx)	24h	Algae or other aquatic plants	0.011mg/L	4
EC50	48h	Crustacea	7550mg/l	4
EC50	96h	Algae or other aquatic plants	>1000mg/l	1
LC50	96h	Fish	4200mg/l	4

zinc phosphate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	24h	Crustacea	0.22mg/l	2
EC50	48h	Crustacea	>1.08mg/l	2

zinc oxide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.036-0.049mg/l	4
BCF	1344h	Fish	19-110	7

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	NOEC(ECx)	72h	Algae or other aquatic plants	0.005mg/l	2
	EC50	48h	Crustacea	0.301-0.667mg/l	4
	EC50	96h	Algae or other aquatic plants	0.3mg/l	2
	LC50	96h	Fish	0.927-2.589mg/l	4

2-Methoxy-1-methylethyl acetate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>1000mg/l	2
	NOEC(ECx)	336h	Fish	47.5mg/l	2
	EC50	48h	Crustacea	373mg/l	2
	EC50	96h	Algae or other aquatic plants	>1000mg/l	2
	LC50	96h	Fish	100mg/l	1

ethylbenzene	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	4.6mg/l	1
	NOEC(ECx)	720h	Fish	0.381mg/L	4
	EC50	48h	Crustacea	1.37-4.4mg/l	4
	EC50	96h	Algae or other aquatic plants	3.6mg/l	2
	LC50	96h	Fish	3.381-4.075mg/L	4

butane	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
	EC50	96h	Algae or other aquatic plants	7.71mg/l	2
	LC50	96h	Fish	24.11mg/l	2

propane	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
	EC50	96h	Algae or other aquatic plants	7.71mg/l	2
	LC50	96h	Fish	24.11mg/l	2

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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT 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.
When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the
oxygen transfer between the air and the water
Oils of any kind can cause:

- drowning of water-fowl due to lack of buoyancy, loss of insulating capacity of feathers, starvation and vulnerability to predators due to lack of mobility
- lethal effects on fish by coating gill surfaces, preventing respiration
- asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom and
- adverse aesthetic effects of fouled shoreline and beaches

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.
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.
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

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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.
For butane:
log Kow: 2.89
Koc: 450-900
BCF: 1.9
Environmental Fate
Terrestrial Fate: An estimated Koc value of 900, determined from a log Kow of 2.89 indicates that n-butane is expected to have low mobility in soil. Volatilisation of n-butane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 0.95 atm-cu m/mole, derived from its vapor pressure, 1820 mm Hg and water solubility, 61.2 mg/l. The potential for volatilisation of n-butane from dry soil surfaces may exist based upon its vapor pressure. While volatilistion from soil surfaces is expected to be the predominant fate process of n-butane released to soil, this compound is also susceptible to biodegradation. In one soil, a biodegradation rate of 1.8 mgC/day/kg dry soil was reported.
Aquatic fate: The estimated Koc value indicates that n-butane may adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 2.2 hours and 3 days, respectively. An estimated BCF of 33 derived from the log Kow suggests the potential for bioconcentration in aquatic organisms is moderate. While volatilisation from water surfaces is expected to be the major fate process for n-butane released to water, biodegradation of this compound is also expected to occur. In a screening study, complete biodegradation was reported in 34 days. In a second study using a defined microbial culture, it was reported that n-butane was degraded to 2-butanone and 2-butanol. Photolysis or hydrolysis of n-butane in aquatic systems is not expected to be important.
Atmospheric fate: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and the vapour pressure, n-butane, is expected to exist solely as a gas in the ambient atmosphere. Gas-phase n-butane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 6.3 days, calculated from its rate constant of 2.54x10-12 cu cm/molecule-sec at 25 deg. Based on data for iso-octane and n-hexane, n-butane is not expected to absorb UV light in the environmentally significant range, >290 nm and probably will not undergo direct photolysis in the atmosphere. Experimental data showed that 7.7% of the n-butane fraction in a dark chamber reacted with nitrogen oxide to form the corresponding alkyl nitrate, suggesting nighttime reactions with radical species and nitrogen oxides may contribute to the atmospheric transformation of n-butane.
For Propane: Koc 460. log
Kow 2.36.
Henry's Law constant of 7.07x10-1 atm-cu m/mole, derived from its vapour pressure, 7150 mm Hg, and water solubility, 62.4 mg/L. Estimated BCF: 13.1.
Terrestrial Fate: Propane is expected to have moderate mobility in soil. Volatilization from moist soil surfaces is expected to be an important fate process. Volatilization from dry soil surfaces is based vapor pressure. Biodegradation may be an important fate process in soil and sediment.
Aquatic Fate: Propane is expected to adsorb to suspended solids and sediment. Volatilization from water surfaces is expected and half-lives for a model river and model lake are estimated to be 41 minutes and 2.6 days, respectively. Biodegradation may not be an important fate process in water.
Ecotoxicity: The potential for bioconcentration in aquatic organisms is low.
Atmospheric Fate: Propane is expected to exist solely as a gas in the ambient atmosphere. Gas-phase propane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 14 days and is not expected to be susceptible to direct photolysis by sunlight.
DO NOT discharge into sewer or waterways.
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
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.

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

Continued...

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.

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
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
ethyl-3-ethoxypropionate	LOW	LOW
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
2-Methoxy-1-methylethyl acetate	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
butane	LOW	LOW
propane	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
acetone	LOW (BCF = 0.69)
n-butyl acetate	LOW (BCF = 14)
methyl ethyl ketone	LOW (LogKOW = 0.29)
toluene	LOW (BCF = 90)
ethyl-3-ethoxypropionate	LOW (LogKOW = 1.0809)
xylene	MEDIUM (BCF = 740)
n-butanol	LOW (BCF = 0.64)
isopropanol	LOW (LogKOW = 0.05)
zinc oxide	LOW (BCF = 217)
2-Methoxy-1-methylethyl acetate	LOW (BCF = 2)
ethylbenzene	LOW (BCF = 79.43)
butane	LOW (LogKOW = 2.89)
propane	LOW (LogKOW = 2.36)

Mobility in soil

Ingredient	Mobility
acetone	HIGH (KOC = 1.981)
n-butyl acetate	LOW (KOC = 20.86)
methyl ethyl ketone	MEDIUM (KOC = 3.827)
toluene	LOW (KOC = 268)
ethyl-3-ethoxypropionate	LOW (KOC = 10)
n-butanol	MEDIUM (KOC = 2.443)
isopropanol	HIGH (KOC = 1.06)
2-Methoxy-1-methylethyl acetate	HIGH (KOC = 1)
ethylbenzene	LOW (KOC = 517.8)
butane	LOW (KOC = 43.79)
propane	LOW (KOC = 23.74)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. ▸ **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...

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.
The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.
DO NOT deposit the hazardous substance into or onto a landfill or a sewage facility.
Burning the hazardous substance must happen under controlled conditions with no person or place exposed to
(1) a blast overpressure of more than 9 kPa; or
(2) an unsafe level of heat radiation.
The disposed hazardous substance must not come into contact with class 1 or 5 substances.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	Not Applicable

Land transport (UN)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	Class: 2.1 Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 63; 190; 277; 327; 344; 381 Limited quantity: 1000ml

Air transport (ICAO-IATA / DGR)

UN number	1950
UN proper shipping name	Aerosols, flammable
Transport hazard class(es)	ICAO/IATA Class: 2.1 ICAO / IATA Subrisk: Not Applicable ERG Code: 10L
Packing group	Not Applicable
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A145 A167 A802 Cargo Only Packing Instructions: 203 Cargo Only Maximum Qty / Pack: 150 kg Passenger and Cargo Packing Instructions: 203 Passenger and Cargo Maximum Qty / Pack: 75 kg Passenger and Cargo Limited Quantity Packing Instructions: Y203 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	IMDG Class: 2.1 IMDG Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Marine Pollutant

Continued...

Special precautions for user	EMS Number	F-D, S-U
	Special provisions	63 190 277 327 344 381 959
	Limited Quantities	1000 ml

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

Not Applicable

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

Product name	Group
acetone	Not Available
n-butyl acetate	Not Available
methyl ethyl ketone	Not Available
toluene	Not Available
ethyl-3-ethoxypropionate	Not Available
xylene	Not Available
n-butanol	Not Available
isopropanol	Not Available
zinc phosphate	Not Available
zinc oxide	Not Available
2-Methoxy-1-methylethyl acetate	Not Available
ethylbenzene	Not Available
butane	Not Available
propane	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
acetone	Not Available
n-butyl acetate	Not Available
methyl ethyl ketone	Not Available
toluene	Not Available
ethyl-3-ethoxypropionate	Not Available
xylene	Not Available
n-butanol	Not Available
isopropanol	Not Available
zinc phosphate	Not Available
zinc oxide	Not Available
2-Methoxy-1-methylethyl acetate	Not Available
ethylbenzene	Not Available
butane	Not Available
propane	Not Available

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002517	Aerosols Flammable Carcinogenic Group Standard 2020

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

acetone is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls	New Zealand Inventory of Chemicals (NZIoC)
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals	New Zealand Workplace Exposure Standards (WES)
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data	

n-butyl acetate is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls	New Zealand Inventory of Chemicals (NZIoC)
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals	New Zealand Workplace Exposure Standards (WES)
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data	

methyl ethyl ketone is found on the following regulatory lists

Continued...

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

toluene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

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

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)

xylene is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

n-butanol is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

isopropanol is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

zinc phosphate is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

zinc oxide is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

2-Methoxy-1-methylethyl acetate is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

ethylbenzene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

butane is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

propane is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

Continued...

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantity (Closed Containers)	Quantity (Open Containers)
2.1.2A	3 000 L (aggregate water capacity)	3 000 L (aggregate water capacity)

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
2.1.2A				1L (aggregate water capacity)

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (acetone; n-butyl acetate; methyl ethyl ketone; toluene; ethyl-3-ethoxypropionate; xylene; n-butanol; isopropanol; ethylbenzene; butane; propane)
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	No (zinc phosphate)
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	15/07/2022
Initial Date	14/07/2022

SDS Version Summary

Version	Date of Update	Sections Updated
1.2	14/07/2022	Chronic Health, Classification, Ingredients, Personal Protection (hands/feet)

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

Continued...

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

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end of SDS