COLORPAK PRO SERIES AEROSOL ACRYLIC SATIN BLACK

Formula Marketing Limited

Chemwatch Hazard Alert Code: **4**

Version No: **1.1**
Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Issue Date: **22/07/2022**
Print Date: **22/07/2022**
L.GHS.NZL.EN

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

Product Identifier

Product name	COLORPAK PRO SERIES AEROSOL ACRYLIC SATIN BLACK
Chemical Name	Not Applicable
Synonyms	CPA1096; CPS422
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	2		
Body Contact	2		
Reactivity	0		
Chronic	4		

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

Classification [1]	Specific Target Organ Toxicity - Repeated Exposure Category 2, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2, Reproductive Toxicity Category 2, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3, 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, 6.3A, 6.4A, 6.5B (contact), 6.8B, 6.9B, 9.1C

Label elements

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

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Hazard statement(s)

H373	May cause damage to organs through prolonged or repeated exposure.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H361	Suspected of damaging fertility or the unborn child.
H317	May cause an allergic skin reaction.
H412	Harmful to aquatic life with long lasting effects.
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.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P314	Get medical advice/attention if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

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	30-45	acetone
78-93-3	10-20	methyl ethyl ketone
108-88-3	10-20	toluene
108-65-6	8-15	2-Methoxy-1-methylethyl acetate
27138-31-4	1-5	dipropylene glycol dibenzoate
1119-40-0	1-5	dimethyl glutarate
108-94-1	1-5	cyclohexanone
64-17-5	1-5	ethanol
106-97-8.	10-20	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:

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	▸ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▸ 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

Treat symptomatically.
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.
- 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
Following acute or short term repeated exposures to toluene:
- Toluene is absorbed across the alveolar barrier, the blood/air mixture being 11.2/15.6 (at 37 degrees C.) The concentration of toluene, in expired breath, is of the order of 18 ppm following sustained exposure to 100 ppm. The tissue/blood proportion is 1/3 except in adipose where the proportion is 8/10.
- Metabolism by microsomal mono-oxygenation, results in the production of hippuric acid. This may be detected in the urine in amounts between 0.5 and 2.5 g/24 hr which represents, on average 0.8 gm/gm of creatinine. The biological half-life of hippuric acid is in the order of 1-2 hours.
- Primary threat to life from ingestion and/or inhalation is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (eg cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 <50 mm Hg or pCO2 > 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial damage 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 (adrenaline) 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.

BIOLOGICAL EXPOSURE INDEX - BEI

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

Determinant	Index	Sampling Time	Comments
o-Cresol in urine	0.5 mg/L	End of shift	B
Hippuric acid in urine	1.6 g/g creatinine	End of shift	B, NS

Continued...

Toluene in blood	0.05 mg/L	Prior to last shift of workweek

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

B: Background levels occur in specimens collected from subjects NOT exposed

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. ‣ 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) , 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.

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 personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by all means available, spillage from entering drains or water courses. ‣ Consider evacuation (or protect in place). ‣ No smoking, naked lights or ignition sources.

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- Increase ventilation.
- Stop leak if safe to do so.
- Water spray or fog may be used to disperse / absorb vapour.
- Contain or absorb spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- If contamination of drains or waterways occurs, advise emergency services.
- 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.
- 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 tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe

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

The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example.
Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.

- A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides

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	or disposed of before this date. ▸ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▸ Unopened containers received from the supplier should be safe to store for 18 months. ▸ Opened containers should not be stored for more than 12 months. ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights 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	Methyl ethyl ketone: ▸ reacts violently with strong oxidisers, aldehydes, nitric acid, perchloric acid, potassium tert-butoxide, oleum ▸ is incompatible with inorganic acids, aliphatic amines, ammonia, caustics, isocyanates, pyridines, chlorosulfonic aid ▸ forms unstable peroxides in storage, or on contact with propanol or hydrogen peroxide ▸ attacks some plastics ▸ may generate electrostatic charges, due to low conductivity, on flow or agitation Toluene: ▸ reacts violently with strong oxidisers, bromine, bromine trifluoride, chlorine, hydrochloric acid/ sulfuric acid mixture, 1,3-dichloro-5,5-dimethyl-2,4-imidazolidindione, dinitrogen tetraoxide, fluorine, concentrated nitric acid, nitrogen dioxide, silver chloride, sulfur dichloride, uranium fluoride, vinyl acetate ▸ forms explosive mixtures with strong acids, strong oxidisers, silver perchlorate, tetranitromethane ▸ is incompatible with bis-toluenediazo oxide ▸ attacks some plastics, rubber and coatings ▸ may generate electrostatic charges, due to low conductivity, on flow or agitation. ▸ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates. 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. ▸ 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 ▸ 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. 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) Butane/ isobutane ▸ reacts violently with strong oxidisers

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- 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.
- Glycol ethers may form peroxides under certain conditions; the potential for peroxide formation is enhanced when these substances are used in processes such as distillation where they are concentrated or even evaporated to near-dryness or dryness; storage under a nitrogen atmosphere is recommended to minimise the possible formation of highly reactive peroxides
- Nitrogen blanketing is recommended if transported in containers at temperatures within 15 deg C of the flash-point and at or above the flash-point - large containers may first need to be purged and inerted with nitrogen prior to loading
- In the presence of strong bases or the salts of strong bases, at elevated temperatures, the potential exists for runaway reactions.
- Contact with aluminium should be avoided; release of hydrogen gas may result- glycol ethers will corrode scratched aluminium surfaces.
- May discolour in mild steel/ copper; lined containers, glass or stainless steel is preferred
- Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water . Investigation of the hazards associated with use of 2-butoxyethanol for alloy electropolishing showed that mixtures with 50-95% of acid at 20 deg C, or 40-90% at 75 C, were explosive and initiable by sparks. Sparking caused mixtures with 40-50% of acid to become explosive, but 30% solutions appeared safe under static conditions of temperature and concentration.

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).

Propylene glycol monomethyl ether (PGME):

· reacts violently with strong oxidisers, alkalis

· is incompatible with aliphatic amines, boranes, sulfuric acid, nitric acid, perchloric acid, caustics, isocyanates

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
- 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)	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)	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)	cyclohexanone	Cyclohexanone	25 ppm / 100 mg/m3	Not Available	Not Available	(skin)-Skin absorption
New Zealand Workplace Exposure Standards (WES)	ethanol	Ethyl alcohol (Ethanol)	1000 ppm / 1880 mg/m3	Not Available	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
methyl ethyl ketone	Not Available	Not Available	Not Available
toluene	Not Available	Not Available	Not Available
2-Methoxy-1-methylethyl acetate	100 ppm	160 ppm	660 ppm
2-Methoxy-1-methylethyl acetate	Not Available	Not Available	Not Available
cyclohexanone	60 ppm	830 ppm	5000* ppm
ethanol	Not Available	Not Available	15000* ppm

Continued...

Ingredient	TEEL-1	TEEL-2	TEEL-3
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
methyl ethyl ketone	3,000 ppm	Not Available
toluene	500 ppm	Not Available
2-Methoxy-1-methylethyl acetate	Not Available	Not Available
dipropylene glycol dibenzoate	Not Available	Not Available
dimethyl glutarate	Not Available	Not Available
cyclohexanone	700 ppm	Not Available
ethanol	3,300 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
dipropylene glycol dibenzoate	D	> 0.1 to ≤ 1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

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

For cyclohexanone
Odour Threshold Value: 0.12 ppm (detection and recognition)
Exposure at the TLV-TWA produces minimal irritation and this limit is significantly lower than the concentration reported to just induce demonstrable changes in the liver and kidneys of rabbits repeatedly exposed to the substance (190 ppm).
Odour Safety Factor (OSF)
OSF=28 (CYCLOHEXANONE)
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)

Continued...

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.
Odour Safety Factor(OSF)
OSF=17 (TOLUENE)

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

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. ▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▸ Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▸ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▸ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▸ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▸ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed. 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.
Personal protection	
Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or

Continued...

	national equivalent] ▸ 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 required due to the physical form of the product.
Skin protection	See Hand protection below
Hands/feet protection	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. For esters: ▸ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. ▸ 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	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ 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 ACRYLIC SATIN BLACK

Material	CPI
PE/EVAL/PE	A
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	AX-AUS / Class 1 P2	-	AX-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	AX-2 P2	AX-PAPR-2 P2
up to 50 x ES	-	AX-3 P2	-
50+ x ES	-	Air-line**	-

^ - 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 2-PLY	C
TEFLON	C
VITON	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	Black, aerosol

Physical state	article	**Relative density (Water = 1)**	0.74
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	Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression , headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation,

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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.
The most common signs of inhalation overexposure to ethanol, in animals, include ataxia, incoordination and drowsiness for those surviving narcosis. The narcotic dose for rats, after 2 hours of exposure, is 19260 ppm.
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.

Common, generalised symptoms associated with toxic gas inhalation include:
- central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures;
- respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest;
- cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest;
- gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain.

The odour of for propylene glycol monomethyl ether (PGME) becomes objectionable at 100 ppm and intolerable with anaesthetic effects at 1000 ppm. High vapour concentrations (above 1000 ppm) are intolerable due to severe eye, nose and throat irritation. Odour is transiently objectionable above 100 ppm. Obvious sedation, increased liver weights and reduced specific gravity of the urine were found in animals subject to concentrations of 3000 ppm PGME.
Inhalation may produce central nervous system depression. High concentrations of the beta-isomer produced slight growth depression and slight liver change and lung effects in rats and mice.

Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.
Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination
Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
The 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.
Acute exposure of humans to high concentrations of methyl ethyl ketone produces irritation to the eyes, nose, and throat. Other effects reported from acute inhalation exposure in humans include central nervous system depression, headache, and nausea.
Easy odour recognition and irritant properties of methyl ethyl ketone means that high vapour levels are readily detected and should be avoided by application of control measures; however odour fatigue may occur with loss of warning of exposure.
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

Ingestion of ethanol (ethyl alcohol, "alcohol") may produce nausea, vomiting, bleeding from the digestive tract, abdominal pain, and diarrhoea.
Effects on the body:

Blood concentration	Effects
<1.5 g/L	Mild: impaired vision, co-ordination and reaction time; emotional instability
1.5-3.0 g/L	Moderate: Slurred speech, confusion, inco-ordination, emotional instability, disturbances in perception and senses, possible blackouts, and impaired objective performance in standardized tests. Possible double vision, flushing, fast heart rate, sweating and incontinence. Slow breathing may occur rarely and fast breathing may develop in cases of metabolic acidosis, low blood sugar and low blood potassium. Central nervous system depression may progress to coma.
3-5 g/L	Severe: cold clammy skin, low body temperature and low blood pressure. Atrial fibrillation and heart block have been reported. Depression of breathing may occur, respiratory failure may follow serious poisoning, choking on vomit may result in lung inflammation and swelling. Convulsions due to severe low blood sugar may also occur. Acute liver inflammation

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	may develop. At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes) Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. 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	The material may accentuate any pre-existing dermatitis condition Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Dermatitis has been reported in humans following dermal exposure to methyl ethyl ketone. Tests involving acute exposure of rabbits has shown methyl ethyl ketone to have high acute toxicity from dermal exposure. Spray mist may produce discomfort Toxic amounts of for propylene glycol monomethyl ether (PGME) may be absorbed through the skin following extensive prolonged contact ; this may result in drowsiness. Constant contact with the beta-isomer, on the skin of rabbits, for several weeks caused very mild, simple irritation. Dose rates of 10 mg/kg produced incomplete anaesthesia, depression, and slight increase in kidney weights in test animals. 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. 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	Direct contact of the eye with ethanol may cause immediate stinging and burning with reflex closure of the lid and tearing, transient injury of the corneal epithelium and hyperaemia of the conjunctiva. Foreign-body type discomfort may persist for up to 2 days but healing is usually spontaneous and complete. 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.. The vapour when concentrated has pronounced eye irritation effects and this gives some warning of high vapour concentrations. If eye irritation occurs seek to reduce exposure with available control measures, or evacuate area. The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer. 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 Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats Which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer . beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents. Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size.

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Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1).
(1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996
Principal route of occupational exposure to the gas is by inhalation.
Animal testing shows that methyl ethyl ketone may have slight effects on the nervous system, liver, kidney and respiratory system; there may also be developmental effects and an increase in birth defects. However, there is limited information available on the long-term effects of methyl ethyl ketone in humans, and no information is available on whether it causes developmental or reproductive toxicity or cancer. It is generally considered to have low toxicity, but it is often used in combination with other solvents, and the toxic effects of the mixture may be greater than with either solvent alone. Combinations of n-hexane or methyl n-butyl ketone with methyl ethyl ketone may increase the rate of peripheral neuropathy, a progressive disorder of the nerves of the extremities. Combinations with chloroform also show increase in toxicity.
Chronic toluene habituation occurs following intentional abuse (glue sniffing) or from occupational exposure. Ataxia, incoordination and tremors of the hands and feet (as a consequence of diffuse cerebral atrophy), headache, abnormal speech, transient memory loss, convulsions, coma, drowsiness, reduced colour perception, frank blindness, nystagmus (rapid, involuntary eye-movements), hearing loss leading to deafness and mild dementia have all been associated with chronic abuse. Peripheral nerve damage, encephalopathy, giant axonopathy electrolyte disturbances in the cerebrospinal fluid and abnormal computer tomographic (CT scans) are common amongst toluene addicts. Although toluene abuse has been linked with kidney disease, this does not commonly appear in cases of occupational toluene exposures. Cardiac and haematological toxicity are however associated with chronic toluene exposures. Cardiac arrhythmia, multifocal and premature ventricular contractions and supraventricular tachycardia are present in 20% of patients who abused toluene-containing paints. Previous suggestions that chronic toluene inhalation produced human peripheral neuropathy have been discounted. However central nervous system (CNS) depression is well documented where blood toluene exceeds 2.2 mg%. Toluene abusers can achieve transient circulating concentrations of 6.5 %. Amongst workers exposed for a median time of 29 years, to toluene, no subacute effects on neurasthenic complaints and psychometric test results could be established.
The prenatal toxicity of very high toluene concentrations has been documented for several animal species and man. Malformations indicative of specific teratogenicity have not generally been found. Neonatal toxicity, described in the literature, takes the form of embryo death or delayed foetal growth and delayed skeletal system development. Permanent damage of children has been seen only when mothers have suffered from chronic intoxication as a result of "sniffing".
Repeated oral doses of 3 g/kg for propylene glycol monomethyl ether (PGME) produced minor changes in the liver and kidneys in rats. Repeated doses on the skin over a 90-day period resulted in absorption and anaesthetic death at 7-10 ml/kg/day. Mild narcosis was observed after topical application of 2-4 ml/kg/day.
Administration of 2% PGME in drinking water ad libitum to males for 25 days did not elicit significant changes in testes or seminal vesicle and coagulating gland weights or in peripheral leukocyte counts. No significant testicular toxicity was found in rats or rabbits that were exposed at up to 3000 PGME, 6 hours/day, 5 days/week for 13 weeks. Oral and parenteral administration to pregnant rabbits, mice and rats did not induce congenital malformations at concentrations up to 1800 mg/kg/day.
In a study on the teratogenic potential of the acetate of the beta-isomer (2-methoxy-1-propyl acetate), a significant increase in the number of litters with abnormal rats and rabbits was found after inhalation exposure by the mothers to 2700 ppm or 550 ppm, respectively, on days 6 to 15, or 6 to 18 of gestation. The rabbit inhalation no-observed-adverse effect concentration was 145 ppm. A similar embryotoxicity profile was seen after inhalation of 2-methoxy-1-propanol (beta-PGMA). In contrast to the alpha-isomer, beta-PGMA is oxidised in rats to 2-methoxypropionic acid. Male dogs exposed to the beta-isomer, developed numerous spermiophages in epididymi. Administration of high doses of the beta-isomer to rats, by gavage, caused delayed ossification of the skull of rat foetus.
Whilst alpha-PGMA undergoes hepatic O-demethylation as the principal pathway, the beta-isomer is detoxified by alcohol/ aldehyde dehydrogenase. Commercial PGME contains low concentrations of the beta-isomer.
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.
Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

COLORPAK PRO SERIES AEROSOL ACRYLIC SATIN BLACK	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

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

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]

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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]
dipropylene glycol dibenzoate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >200 mg/l4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; 3295 mg/kg[1]	
dimethyl glutarate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): Irritant
	Inhalation(Rat) LC50; >11 mg/l4h[1]	Skin (human): Irritant
	Oral (Rat) LD50; >2000 mg/kg[1]	
cyclohexanone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 948 mg/kg[2]	Eye (human): 75 ppm
	Inhalation(Rat) LC50; 8000 ppm4h[2]	Eye (rabbit): 0.25 mg/24h SEVERE
	Oral (Rat) LD50; 1535 mg/kg[2]	Eye (rabbit): 4.74 mg SEVERE
		Skin (rabbit): 500 mg(open) mild
ethanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 17100 mg/kg[1]	Eye (rabbit): 500 mg SEVERE
	Inhalation(Rat) LC50; 64000 ppm4h[2]	Eye (rabbit):100mg/24hr-moderate
	Oral (Rat) LD50; 7060 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):20 mg/24hr-moderate
		Skin (rabbit):400 mg (open)-mild
		Skin: no adverse effect observed (not irritating)[1]
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 ACRYLIC SATIN BLACK	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. 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. 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

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	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**
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 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.
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.
DIPROPYLENE GLYCOL DIBENZOATE	The U.S. EPA High Production Volume Information System (HPVIS 2009) lists both diethylene glycol dibenzoate (DEGDB) and dipropylene glycol dibenzoate (DPGDB) as non-mutagenic and non-carcinogenic.
DIMETHYL GLUTARATE	The family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CAS No. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixture DBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produce DMS, DMG, and DMA and three other fractions that are mixtures of these esters generally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbon straight-chain dicarboxylic acid dimethyl esters differing incrementally by one carbon atom. The four members of the category produce similar levels of acute and repeated-dose toxicity in experimental animals DBEs have very low acute oral toxicities with LD50 s in rats generally > 5,000 mg/kg (with two exceptions reported as >500 and <5,000 mg/kg b.wt. for DBE (the mixture) and DMS By skin absorption, DBEs have a low order of acute toxicity to rabbits with dermal LD50s of 3,000 mg/kg . Based upon the most recent GLP studies DBEs are not considered to produce primary dermal irritation as defined in EPA Guidelines . Earlier studies did show moderate irritation in one of six rabbits, but these results were not repeated in later studies. All four DBE materials are considered to produce eye irritation as defined by EPA Guidelines. Mild to moderate irritation involving the cornea was observed in rabbits with recovery by 7 days. DBEs are not skin sensitisers, and are not harmful via skin or inhalation exposures. DBE is slightly toxic by inhalation with 1-and 4-hour LC50s in rats of > 10.7 and > 11 mg/L, respectively. In subchronic inhalation studies with all four DBEs, degeneration of the olfactory epithelium of the nose was observed. This change in the nasal tissues is related to enzymatic hydrolysis of DBE within the nasal cavity. However, risk to human nasal tissue due to DBE toxicity is likely to be reduced when compared to rats since DBEs are hydrolysed more slowly in humans. No information is available on the carcinogenic potential of DBEs. A range of studies with DMS, DMG, DMA and DBE did not produce genetic damage in animals or bacterial cell cultures. DBE was positive in one study with cultured mammalian cells, but the positive findings were not apparent when the assay was repeated. Testing in rats indicates DBEs are not developmental or reproductive toxicants.

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CYCLOHEXANONE	Cyclohexanone: **Acute toxicity:** Cyclohexanone exhibits low to slight acute toxicity by the oral and inhalation routes and is moderately toxic by the dermal route. It is an eye and skin irritant; however, it did not induce skin sensitisation. There has been no consistent indication that cyclohexanone causes neurotoxicity, although signs of CNS depression were noted at doses near the LD50. Therefore, this material could not be classified regarding its potential neurotoxicity to humans. **Repeat dose toxicity:** Upon repeated administration to rats in drinking water, the NOAEL was 4700 ppm after 25 weeks and the LOAEL was 3300 ppm after 2 years. Effects at higher concentrations were primarily body weight decreases. The NOAEL in published repeated dose inhalation studies was 100-190 ppm. Those values were based on either gray mottling of the lungs or ocular irritation and degenerative changes in the liver and kidney at higher concentrations. However, the NOAEL in those studies was not confirmed in more conclusive and GLP inhalation studies for reproductive and developmental effects (NOAEL = 650-1000 ppm). **Genotoxicity:** The majority of the experimental evidence indicates that cyclohexanone is not genotoxic, and this material was not considered to be carcinogenic in mice or rats following two years of exposure via the drinking water. Reproductive toxicity: In a two-generation reproduction study, decreased fertility was observed in rats exposed via inhalation at 1400 ppm but not at 500 ppm; however, the effect was found to be reversible following a post-exposure recovery period. The NOAEL of 500 ppm for this reproductive effect is 1000 times greater than the highest occupational personal monitoring value (0.5 ppm) reported. **Developmental toxicity:** Developmental studies indicate that foetal toxicity was present only at concentrations which were maternally toxic, and no malformations were detected. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.
COLORPAK PRO SERIES AEROSOL ACRYLIC SATIN BLACK & METHYL ETHYL KETONE & 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 ACRYLIC SATIN BLACK & DIPROPYLENE GLYCOL DIBENZOATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
COLORPAK PRO SERIES AEROSOL ACRYLIC SATIN BLACK & 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 >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.

Continued...

	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 ACRYLIC SATIN BLACK & 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.
COLORPAK PRO SERIES AEROSOL ACRYLIC SATIN BLACK & 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
ACETONE & 2-METHOXY-1-METHYLETHYL ACETATE & CYCLOHEXANONE	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.
METHYL ETHYL KETONE & TOLUENE & DIMETHYL GLUTARATE & ETHANOL	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.
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

	Endpoint	Test Duration (hr)	Species	Value	Source
COLORPAK PRO SERIES AEROSOL ACRYLIC SATIN BLACK	Not Available	Not Available	Not Available	Not Available	Not Available
acetone	Not Available	Not Available	Not Available	Not Available	Not Available
methyl ethyl ketone	Not Available	Not Available	Not Available	Not Available	Not Available
toluene	Not Available	Not Available	Not Available	Not Available	Not Available
2-Methoxy-1-methylethyl acetate	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...

dipropylene glycol dibenzoate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
dimethyl glutarate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
cyclohexanone	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
ethanol	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
butane	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
propane	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
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*				

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
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 Propylene Glycol Ethers: log Kow's range from 0.309 for TPM to 1.523 for DPnB. Calculated BCFs range from 1.47 for DPnB to 3.16 for DPMA and TPM, indicating low bioaccumulation. Henry's Law Constants are low for all category members, ranging from 5.7 x 10-9 atm-m3/mole for TPM to 2.7 x10-9 atm-m3/mole for PnB.
Environmental Fate: Most are liquids at room temperature and all are water-soluble.
Atmospheric Fate: In air, the half-life due to direct reactions with photochemically generated hydroxyl radicals, range from 2.0 hours for TPM to 4.6 hours for PnB.
Aquatic/Terrestrial Fate: Most propylene glycol ethers are likely to partition roughly equally into the soil and water compartments in the environment with small to negligible amounts remaining in other environmental compartments (air, sediment, and aquatic biota). In water, most members of this family are "readily biodegradable" under aerobic conditions. In soil, biodegradation is rapid for PM and PMA.
Ecotoxicity: Propylene glycol ethers are unlikely to persist in the environment. Acute aquatic toxicity testing indicates low toxicity for both ethers and acetates.
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 Ethanol:
log Kow: -0.31 to -0.32;
Koc 1: Estimated BCF= 3;
Half-life (hr) air: 144;
Half-life (hr) H2O surface water: 144;
Henry's atm m3 /mol: 6.29E-06;
BOD 5 if unstated: 0.93-1.67,63%
COD: 1.99-2.11,97%;
ThOD : 2.1.
Environmental Fate: Terrestrial - Ethanol quickly biodegrades in soil but may leach into ground water; most is lost by evaporation. Ethanol is expected to have very high mobility in soil. Volatilization of ethanol from moist soil surfaces is expected to be an important fate process. The potential for volatilization of ethanol from dry soil surfaces may exist. Biodegradation is expected to be an important fate process for ethanol based on half-lives on the order of a few days for ethanol in sandy soil/groundwater microcosms.
Atmospheric Fate: Ethanol is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase ethanol is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 5 days. Ethanol readily degraded by reaction with photochemically produced hydroxy radicals; release into air will result in photodegradation and wet deposition.
Aquatic Fate: When released into water ethanol readily evaporates and is biodegradable. Ethanol is not expected to adsorb to suspended solids and sediment. Volatilization from water surfaces is expected and volatilization half-lives for a model river and model lake are 3 and 39 days, respectively. Bioconcentration in aquatic organisms is considered to be low. Hydrolysis and photolysis in sunlit surface waters is not expected to be an important environmental fate process for ethanol and is unlikely to be persistent in aquatic environments.
For Methyl Ethyl Ketone:
log Kow: 0.26-0.69;
log Koc: 0.69;
Koc: 34;
Half-life (hr) air: 2.3;
Half-life (hr) H2O surface water: 72-288;
Henry's atm m3 /mol: 1.05E-05;

Continued...

BOD 5: 1.5-2.24, 46%;
COD: 2.2-2.31, 100%;
ThOD: 2.44;
BCF: 1.
Environmental Fate: Terrestrial Fate - Measured Koc values of 29 and 34 were obtained for methyl ethyl ketone in silt loams. Methyl ethyl ketone is expected to have very high mobility in soil. Volatilization of methyl ethyl ketone from moist and dry soil surfaces is expected. The volatilization half-life of methyl ethyl ketone from silt and sandy loams was measured as 4.9 days. Methyl ethyl ketone is expected to biodegrade under both aerobic and anaerobic conditions.
Aquatic Fate: Methyl ethyl ketone is not expected to adsorb to suspended solids and sediment in water and is expected to volatilize from water surfaces. Estimated half-lives for a model river and model lake are 19 and 197, hours respectively. Bioconcentration is expected to be low in aquatic systems.
Atmospheric Fate: Methyl ethyl ketone will exist solely as a vapour in the ambient atmosphere. Vapour-phase methyl ethyl ketone 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 14 days. Methyl ethyl ketone is also expected to undergo photodecomposition in the atmosphere by natural sunlight.
Ecotoxicity: Methyl ethyl ketone is not acutely toxic to fish, specifically, bluegill sunfish, guppy, goldfish, fathead minnow, mosquito fish, Daphnia magna water fleas and brine shrimp.
For Glycol Ethers:
Environmental Fate: Several glycol ethers have been shown to biodegrade however; biodegradation slows as molecular weight increases. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes.
Atmospheric Fate: Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photo-degradation (atmospheric half lives = 2.4-2.5 hr). Aquatic Fate: In water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).
Ecotoxicity: Tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers. Glycols exert a high oxygen demand for decomposition and once released to the environment death of aquatic organisms occurs if dissolved oxygen is depleted.
For Ketones: Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds.
Aquatic Fate: Hydrolysis of ketones in water is thermodynamically favourable only for low molecular weight ketones. Reactions with water are reversible with no permanent change in the structure of the ketone substrate. Ketones are stable to water under ambient environmental conditions. When pH levels are greater than 10, condensation reactions can occur which produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavourable. Based on its reactions in air, it seems likely that ketones undergo photolysis in water.
Terrestrial Fate: It is probable that ketones will be biodegraded by micro-organisms in soil and water.
Ecotoxicity: Ketones are unlikely to bioconcentrate or biomagnify.
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.
For Toluene:
log Kow : 2.1-3;
log Koc : 1.12-2.85;
Koc : 37-260;
log Kom : 1.39-2.89;
Half-life (hr) air : 2.4-104;
Half-life (hr) H2O surface water : 5.55-528;
Half-life (hr) H2O ground : 168-2628;
Half-life (hr) soil : <48-240;
Henry's Pa m3 /mol : 518-694;
Henry's atm m3 /mol : 5.94;
E-03BOD 5 0.86-2.12, 5%COD - 0.7-2.52,21-27%;
ThOD - 3.13 ; BCF - 1.67-380;
log BCF - 0.22-3.28.
Atmospheric Fate: The majority of toluene evaporates to the atmosphere from the water and soil. The main degradation pathway for toluene in the atmosphere is reaction with photochemically produced hydroxyl radicals. The estimated atmospheric half life for toluene is about 13 hours. Toluene is also oxidized by reactions with atmospheric nitrogen dioxide, oxygen, and ozone, but these are minor degradation pathways. Photolysis is not considered a significant degradative pathway for toluene.
Terrestrial Fate: Toluene is moderately retarded by adsorption to soils rich in organic material, therefore, transport to ground water is dependent on soil composition. In unsaturated topsoil containing organic material, it has been estimated that 97% of the toluene is adsorbed to the soil and only about 2% is in the soil-water phase and transported with flowing groundwater. There is little retardation in sandy soils and 2-13% of the toluene was estimated to migrate with flowing water; the remainder was volatilized, biodegraded, or unaccounted for. In saturated deep soils with no soil-air phase, about 48% may be transported with flowing groundwater. In surface soil, volatilization to air is an important fate process for toluene. In the environment, biodegradation of toluene to carbon dioxide occurs with a typical half life of 1-7 days.
Aquatic Fate: An important fate process for toluene is volatilization, the rate of which depends on the amount of turbulence in the surface water. The volatilization of toluene from static water has a half life of 1-16 days, whereas from turbulent water the half life is 5-6 hours. Degradation of toluene in surface water occurs primarily by biodegradation with a half life of less than one day under favorable conditions (presence of microorganisms, microbial adaptation, and optimum temperature). Biodegradation also occurs in shallow groundwater and in salt water (at a reduced rate). No data are available on anaerobic degradation of toluene in deep ground water conditions where aerobic degradation would be minimal.
Ecotoxicity: Bioaccumulation in the food chain is predicted to be low. Toluene has moderate acute toxicity to aquatic organisms. Toluene is, on the average, slightly toxic to fathead minnow, guppies and goldfish and not acutely toxic to bluegill or channel catfish and crab. Toluene, on the average, is slightly toxic to crustaceans specifically, shrimp species including grass shrimp and daggerblade grass shrimp. Toluene has a negative effect on green algae during their growth phase.
DO NOT discharge into sewer or waterways.

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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.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
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)
2-Methoxy-1-methylethyl acetate	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
dipropylene glycol dibenzoate	HIGH	HIGH
dimethyl glutarate	LOW	LOW
cyclohexanone	LOW	LOW
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
butane	LOW	LOW
propane	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
acetone	LOW (BCF = 0.69)
methyl ethyl ketone	LOW (LogKOW = 0.29)
toluene	LOW (BCF = 90)
2-Methoxy-1-methylethyl acetate	LOW (BCF = 2)
dipropylene glycol dibenzoate	MEDIUM (LogKOW = 4.0228)
dimethyl glutarate	LOW (LogKOW = 0.62)
cyclohexanone	LOW (BCF = 2.45)
ethanol	LOW (LogKOW = -0.31)
butane	LOW (LogKOW = 2.89)
propane	LOW (LogKOW = 2.36)

Mobility in soil

Ingredient	Mobility
acetone	HIGH (KOC = 1.981)
methyl ethyl ketone	MEDIUM (KOC = 3.827)
toluene	LOW (KOC = 268)
2-Methoxy-1-methylethyl acetate	HIGH (KOC = 1)

Continued...

Ingredient	Mobility
dipropylene glycol dibenzoate	LOW (KOC = 1845)
dimethyl glutarate	LOW (KOC = 10)
cyclohexanone	LOW (KOC = 15.15)
ethanol	HIGH (KOC = 1)
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.

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	NO
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	Not Applicable
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	Not Applicable

Continued...

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	Not Applicable	
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
methyl ethyl ketone	Not Available
toluene	Not Available
2-Methoxy-1-methylethyl acetate	Not Available
dipropylene glycol dibenzoate	Not Available
dimethyl glutarate	Not Available
cyclohexanone	Not Available
ethanol	Not Available
butane	Not Available
propane	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
acetone	Not Available
methyl ethyl ketone	Not Available
toluene	Not Available
2-Methoxy-1-methylethyl acetate	Not Available
dipropylene glycol dibenzoate	Not Available
dimethyl glutarate	Not Available
cyclohexanone	Not Available
ethanol	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
HSR002515	Aerosols Flammable 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

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)

methyl ethyl ketone 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)

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)

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)

dipropylene glycol dibenzoate is found on the following regulatory lists

New Zealand Inventory of Chemicals (NZIoC)

dimethyl glutarate 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)

cyclohexanone 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)

ethanol 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)

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)

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

Continued...

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
6.5A or 6.5B	120	1	3	
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; methyl ethyl ketone; toluene; dipropylene glycol dibenzoate; dimethyl glutarate; cyclohexanone; ethanol; 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	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	22/07/2022
Initial Date	21/07/2022

Other information

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

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

Definitions and abbreviations

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