

844AR Acrylic ESD Coating (Aerosol) MG Chemicals UK Limited

Version No: A-2.01

Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: 12/05/2021 Revision Date: 12/05/2021 L.REACH.GB.EN

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

1.1. Product Identifier

Product name	844AR
Synonyms	SDS Code: 844AR-Aerosol; 844AR-340G UFI:AKM0-900P-200W-7YA4
Other means of identification	Acrylic ESD Coating (Aerosol)

1.2. 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	
Uses advised against	Not Applicable	

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)	
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada	
Telephone	+(44) 1663 362888	+(1) 800-201-8822	
Fax	Not Available	+(1) 800-708-9888	
Website	Not Available	www.mgchemicals.com	
Email	sales@mgchemicals.com	Info@mgchemicals.com	

1.4. Emergency telephone number

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Association / Organisation	Verisk 3E (Access code: 335388)		
Emergency telephone numbers	+(44) 20 35147487		
Other emergency telephone numbers	+(0) 800 680 0425		

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments [1]	H336 - Specific target organ toxicity - single exposure Category 3 (narcotic effects), H319 - Eye Irritation Category 2, H229 - Aerosols Category 3, H317 - Skin Sensitizer Category 1	
Legend:	1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI	

2.2. Label elements

Hazard pictogram(s)





Signal word	Warnin
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Hazard statement(s)

H336	May cause drowsiness or dizziness.		
H319	Causes serious eye irritation.		
H223+H229	Flammable aerosol. Pressurised container: May burst if heated.		
H317	May cause an allergic skin reaction.		

Supplementary statement(s)

FUH066	Repeated exposure may cause skin dryness or cracking.

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.		
P251	Do not pierce or burn, even after use.		
P271	Use only outdoors or in a well-ventilated area.		
P280	Wear protective gloves/protective clothing/eye protection/face protection/hearing protection.		
P261	Avoid breathing mist/vapours/spray.		
P272	Contaminated work clothing should not be allowed out of the workplace.		

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/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.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

2.3. Other hazards

Cumulative effects may result following exposure*.

May produce discomfort of the respiratory system and $\mathsf{skin}^\star.$

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

3.2.IVIIXTURES				
1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	Nanoform Particle Characteristics
1.141-78-6 2.205-500-4 3.607-022-00-5 4.Not Available	50	ethyl acetate *	Flammable Liquid Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Eye Irritation Category 2; H225, H336, H319, EUH066 [2]	Not Available
1.74-98-6 2.200-827-9 3.601-003-00-5 4.Not Available	13	propane	Gas under Pressure, Flammable Gas Category 1; H280, H220 [2]	Not Available
1.18282-10-5 2.242-159-0 3.Not Available 4.Not Available	13	stannic oxide *	EUH210 ^[1]	Not Available
1.67-64-1 2.200-662-2 3.606-001-00-8 4.Not Available	10	acetone *	Flammable Liquid Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Eye Irritation Category 2; H225, H336, H319, EUH066 [2]	Not Available
1.75-28-5. 2.200-857-2 3.601-004-00-0 601-004-01-8 4.Not Available	7	iso-butane	Flammable Gas Category 1, Gas under Pressure (Liquefied gas); H220, H280, EUH044 [1]	Not Available
1.67-63-0 2.200-661-7 3.603-117-00-0 4.Not Available	3	isopropanol	Flammable Liquid Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Eye Irritation Category 2; H225, H336, H319 [2]	Not Available
1.108-65-6 2.203-603-9 3.607-195-00-7 4.Not Available	1	propylene glycol monomethyl ether acetate, alpha-isomer *	Flammable Liquid Category 3; H226 [2]	Not Available

1.CAS No 2.EC No 3.Index No 4.REACH No		%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	Nanoform Particle Characteristics
1.25619-56-1 2.247-132-7 3.Not Available 4.Not Available		0.3	barium dinonyl naphthalenesulfonate	Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Oral) Category 4, Acute Toxicity (Oral and Inhalation) Category 4, Skin Corrosion/Irritation Category 2; H318, H302, H302+H332, H315 [1]	Not Available
	Legend:	Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties			

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	If aerosols come in contact with the eyes: Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Transport to hospital or doctor without delay. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If solids or aerosol mists are deposited upon the skin: Flush skin and hair with running water (and soap if available). Remove any adhering solids with industrial skin cleansing cream. DO NOT use solvents. Seek medical attention in the event of irritation.
Inhalation	If aerosols, fumes or combustion products are inhaled: Remove to fresh air. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor.
Ingestion	 Not considered a normal route of entry. If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

Treat symptomatically for simple esters: BASIC TREATMENT

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

ADVANCED TREATMENT

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

EMERGENCY DEPARTMENT

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

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

SECTION 5 Firefighting measures

5.1. 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.

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility

Fire Fighting

▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

5.3. Advice for firefighters

- · 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.

Combustion products include:

carbon dioxide (CO2)

- Liquid and vapour are flammable.
- Moderate fire hazard when exposed to heat or flame.
- Vapour forms an explosive mixture with air
- Moderate explosion hazard when exposed to heat or flame.
- Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- Aerosol cans may explode on exposure to naked flame.
- 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).

carbon monoxide (CO)

metal oxides

other pyrolysis products typical of burning organic material.

Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.

Vented gas is more dense than air and may collect in pits, basements.

SECTION 6 Accidental release measures

Fire/Explosion Hazard

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

► 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.
- **Minor Spills**

 - ▶ 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.

Chemical Class: ester and ethers

For release onto land; recommended sorbents listed in order of priority.

SORBENT TYPE RANK	APPLICATION	COLLECTION	LIMITATIONS
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LAND SPILL - SMALL

Major Spills

cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R,I, P
wood fiber - particulate	3	shovel	shovel	R, W, P, DGC
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skiploader	R,W, SS

cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
wood fiber - particulate	4	blower	skiploader	R, W, P, DGC

Legend

DGC: Not effective where ground cover is dense

R: Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT:Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control:

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

- 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

6.4. Reference to other sections

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

SECTION 7 Handling and storage

7.1. Precautions for safe handling

- 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.
- DO NOT allow clothing wet with material to stay in contact with skin

Fire and explosion protection

See section 5

i See section s

- ▶ 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

Other information

Safe handling

- No smoking, naked lights, heat or ignition sources.
 Keep containers securely sealed. Contents under pressure.
- Store away from incompatible materials.
- 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.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	Aerosol dispenser. Check that containers are clearly labelled.
Storage incompatibility	Butane/ isobutane reacts violently with strong oxidisers reacts with acetylene, halogens and nitrous oxides is incompatible with chlorine dioxide, conc. nitric acid and some plastics may generate electrostatic charges, due to low conductivity, in flow or when agitated - these may ignite the vapour. Segregate from nickel carbonyl in the presence of oxygen, heat (20-40 C) Esters react with acids to liberate heat along with alcohols and acids. Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products. Heat is also generated by the interaction of esters with caustic solutions. Flammable hydrogen is generated by mixing esters with alkali metals and hydrides. Esters may be incompatible with aliphatic amines and nitrates. 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

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
ethyl acetate	Dermal 63 mg/kg bw/day (Systemic, Chronic) Inhalation 734 mg/m³ (Systemic, Chronic) Inhalation 734 mg/m³ (Local, Chronic) Inhalation 1 468 mg/m³ (Systemic, Acute) Inhalation 1 468 mg/m³ (Local, Acute) Dermal 37 mg/kg bw/day (Systemic, Chronic) * Inhalation 367 mg/m³ (Systemic, Chronic) * Inhalation 367 mg/m³ (Systemic, Chronic) * Inhalation 367 mg/m³ (Local, Chronic) * Inhalation 734 mg/m³ (Systemic, Acute) * Inhalation 734 mg/m³ (Local, Acute) *	0.24 mg/L (Water (Fresh)) 0.024 mg/L (Water - Intermittent release) 1.65 mg/L (Water (Marine)) 1.15 mg/kg sediment dw (Sediment (Fresh Water)) 0.115 mg/kg sediment dw (Sediment (Marine)) 0.148 mg/kg soil dw (Soil) 650 mg/L (STP) 0.2 g/kg food (Oral)
stannic oxide	Dermal 5.7 mg/kg bw/day (Systemic, Chronic) Inhalation 2 mg/m³ (Systemic, Chronic) Dermal 5.7 mg/kg bw/day (Systemic, Acute) Inhalation 2 mg/m³ (Systemic, Acute) Dermal 2 mg/kg bw/day (Systemic, Chronic) * Inhalation 6 mg/m³ (Systemic, Chronic) * Oral 2 mg/kg bw/day (Systemic, Chronic) * Dermal 2 mg/kg bw/day (Systemic, Acute) * Inhalation 6 mg/m³ (Systemic, Acute) * Oral 2 mg/kg bw/day (Systemic, Acute) *	0.1 mg/L (Water (Fresh)) 0.01 mg/L (Water - Intermittent release) 1 mg/L (Water (Marine)) 100 mg/L (STP)
acetone	Dermal 186 mg/kg bw/day (Systemic, Chronic) Inhalation 1 210 mg/m³ (Systemic, Chronic) Inhalation 2 420 mg/m³ (Local, Acute) Dermal 62 mg/kg bw/day (Systemic, Chronic) * Inhalation 200 mg/m³ (Systemic, Chronic) * Oral 62 mg/kg bw/day (Systemic, Chronic) *	10.6 mg/L (Water (Fresh)) 1.06 mg/L (Water - Intermittent release) 21 mg/L (Water (Marine)) 30.4 mg/kg sediment dw (Sediment (Fresh Water)) 3.04 mg/kg sediment dw (Sediment (Marine)) 29.5 mg/kg soil dw (Soil) 100 mg/L (STP)
isopropanol	Dermal 888 mg/kg bw/day (Systemic, Chronic) Inhalation 500 mg/m³ (Systemic, Chronic) Dermal 319 mg/kg bw/day (Systemic, Chronic) * Inhalation 89 mg/m³ (Systemic, Chronic) * Oral 26 mg/kg bw/day (Systemic, Chronic) *	140.9 mg/L (Water (Fresh)) 140.9 mg/L (Water - Intermittent release) 140.9 mg/L (Water (Marine)) 552 mg/kg sediment dw (Sediment (Fresh Water)) 552 mg/kg sediment dw (Sediment (Marine)) 28 mg/kg soil dw (Soil) 2251 mg/L (STP) 160 mg/kg food (Oral)
propylene glycol monomethyl ether acetate, alpha-isomer	Dermal 796 mg/kg bw/day (Systemic, Chronic) Inhalation 275 mg/m³ (Systemic, Chronic) Inhalation 550 mg/m³ (Local, Acute) Dermal 320 mg/kg bw/day (Systemic, Chronic) * Inhalation 33 mg/m³ (Systemic, Chronic) * Oral 36 mg/kg bw/day (Systemic, Chronic) * Inhalation 33 mg/m³ (Local, Chronic) *	0.635 mg/L (Water (Fresh)) 0.064 mg/L (Water - Intermittent release) 6.35 mg/L (Water (Marine)) 3.29 mg/kg sediment dw (Sediment (Fresh Water)) 0.329 mg/kg sediment dw (Sediment (Marine)) 0.29 mg/kg soil dw (Soil) 100 mg/L (STP)

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	ethyl acetate	Ethyl acetate	200 ppm / 734 mg/m3	1468 mg/m3 / 400 ppm	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	ethyl acetate	Ethyl acetate	200 ppm / 734 mg/m3	1 468 mg/m3 / 400 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	stannic oxide	Tin compounds, inorganic except SnH4, (as Sn)	2 mg/m3	4 mg/m3	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	stannic oxide	Tin and inorganic tin compounds	2 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	acetone	Acetone	500 ppm / 1210 mg/m3	3620 mg/m3 / 1500 ppm	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	acetone	Acetone	500 ppm / 1210 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	isopropanol	Propan-2-ol	400 ppm / 999 mg/m3	1250 mg/m3 / 500 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Sk
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl-2-acetate	50 ppm / 275 mg/m3	550 mg/m3 / 100 ppm	Not Available	Skin

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
ethyl acetate	1,200 ppm	1,700 ppm	10000** ppm
propane	Not Available	Not Available	Not Available
stannic oxide	7.6 mg/m3	85 mg/m3	510 mg/m3
stannic oxide	7.6 mg/m3	85 mg/m3	510 mg/m3
acetone	Not Available	Not Available	Not Available
iso-butane	5500* ppm	17000** ppm	53000*** ppm
isopropanol	400 ppm	2000* ppm	12000** ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
ethyl acetate	2,000 ppm	Not Available
propane	2,100 ppm	Not Available
stannic oxide	100 mg/m3	Not Available
acetone	2,500 ppm	Not Available
iso-butane	Not Available	Not Available
isopropanol	2,000 ppm	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
barium dinonyl naphthalenesulfonate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit	
barium dinonyl naphthalenesulfonate	E	≤ 0.01 mg/m³	
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.		

MATERIAL DATA

For ethyl acetate:

Odour Threshold Value: 6.4-50 ppm (detection), 13.3-75 ppm (recognition)

The TLV-TWA provides a significant margin of safety from the standpoint of adverse health effects. Unacclimated subjects found the odour objectionably strong at 200 ppm. Mild nose, eye and throat irritation was experienced at 400 ppm. Workers exposed regularly at concentrations ranging from 375 ppm to 1500 ppm for several months showed no unusual signs or symptoms.

Odour Safety Factor(OSF) OSF=51 (ETHYL ACETATE)

^{*} Values for General Population

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)

Saturation vapour concentration: 237000 ppm @ 20 C

NOTE: Detector tubes measuring in excess of 40 ppm, are available.

Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.

Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.

A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.

Odour Safety Factor(OSF)

OSF=38 (ACETONE)

For 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 acetate (PGMEA)

Saturated vapour concentration: 4868 ppm at 20 C.

A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternabral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

May act as a simple asphyxiants; these are gases which, when present in high concentrations, reduce the oxygen content in air below that required to support breathing, consciousness and life; loss of consciousness, with death by suffocation may rapidly occur in an oxygen deficient atmosphere.

CARE: Most simple asphyxiants are odourless or possess low odour and there is no warning on entry into an oxygen deficient atmosphere. If there is any doubt, oxygen content can be checked simply and quickly. It may not be appropriate to only recommend an exposure standard for simple asphyxiants rather it is essential that sufficient oxygen be maintained. Air normally has 21 percent oxygen by volume, with 18 percent regarded as minimum under normal atmospheric pressure to maintain consciousness / life. At pressures significantly higher or lower than normal atmospheric pressure, expert guidance should be sought.

For propane

Odour Safety Factor(OSF)

OSF=0.16 (PROPANE)

Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)

Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

8.2. Exposure controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.

Provide adequate ventilation in warehouse or closed storage areas.

Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

8.2.1. Appropriate engineering controls

Type of Contaminant:	Speed:
aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s
direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
Contaminants of low toxicity or of nuisance value only. 2: Contaminants of high	
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

8.2.2. Personal 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 national equivalent]
- Eye and face protection
 - Close fitting gas tight goggles

DO NOT wear contact lense

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 equivalent1

Skin protection

See Hand protection below

- ▶ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.
- No special equipment needed when handling small quantities

Hands/feet protection

▶ OTHERWISE:

For esters:

- For potentially moderate exposures:
- ▶ Wear general protective gloves, eg. light weight rubber gloves.
- For potentially heavy exposures:
- ▶ Wear chemical protective gloves, eg. PVC. and safety footwear.

Body protection

See Other protection below

- 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.

Other protection OTHERWISE:

- Overalls.
- Skin cleansing cream.
- Evewash unit.
- Do not spray on hot surfaces.

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 computergenerated selection:

844AR Acrylic ESD Coating (Aerosol)

Material	СРІ
PE/EVAL/PE	A
BUTYL	С
BUTYL/NEOPRENE	С
CPE	С
HYPALON	С
NAT+NEOPR+NITRILE	С
NATURAL RUBBER	С
NATURAL+NEOPRENE	С
NEOPRENE	С
NEOPRENE/NATURAL	С
NITRILE	С
NITRILE+PVC	С
PVA	С
PVC	С
PVDC/PE/PVDC	С
SARANEX-23	С
SARANEX-23 2-PLY	С
TEFLON	С

Respiratory protection

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

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

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

^ - Full-face

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

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used
- ▶ Generally not applicable.

VITON/CHLOROBUTYL	С
VITON/NEOPRENE	С

* 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.

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.

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	colorless		
Physical state	Liquid	Relative density (Water = 1)	1.06
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	427
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	<20.5
Initial boiling point and boiling range (°C)	>56	Molecular weight (g/mol)	Not Available
Flash point (°C)	-17	Taste	Not Available
Evaporation rate	Not Available BuAC = 1	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	11	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	2	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	350	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	>1.5	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2	
10.2. Chemical stability	 Elevated temperatures. Presence of open flame. Product is considered stable. Hazardous polymerisation will not occur. 	
10.3. Possibility of hazardous reactions	See section 7.2	
10.4. Conditions to avoid	See section 7.2	
10.5. Incompatible materials	See section 7.2	
10.6. Hazardous decomposition products	See section 5.3	

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled

The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other

route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.

Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation.

The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression , headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures.

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.

Mild eye, nose and throat irritation occurs at 400 ppm ethyl acetate with unacclimated persons. Production workers with regular exposure may intermittently tolerate 1500 ppm for periods up to 3 months without adverse symptoms.

Inhalation hazard is increased at higher temperatures.

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.

Symptoms of asphyxia (suffocation) may include headache, dizziness, shortness of breath, muscular weakness, drowsiness and ringing in the ears. If the asphyxia is allowed to progress, there may be nausea and vomiting, further physical weakness and unconsciousness and, finally, convulsions, coma and death. Significant concentrations of the non-toxic gas reduce the oxygen level in the air. As the amount of oxygen is reduced from 21 to 14 volume %, the pulse rate accelerates and the rate and volume of breathing increase. The ability to maintain attention and think clearly is diminished and muscular coordination is somewhat disturbed. As oxygen decreases from 14-10% judgement becomes faulty; severe injuries may cause no pain. Muscular exertion leads to rapid fatigue. Further reduction to 6% may produce nausea and vomiting and the ability to move may be lost. Permanent brain damage may result even after resuscitation at exposures to this lower oxygen level. Below 6% breathing is in gasps and convulsions may occur. Inhalation of a mixture containing no oxygen may result in unconsciousness from the first breath and death will follow in a few minutes.

The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation.

WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.

The odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal.

Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.

Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.

Not normally a hazard due to physical form of product.

Considered an unlikely route of entry in commercial/industrial environments

Acute intoxication by ethyl acetate causes impaired coordination, exhilaration, slurred speech, vertigo, flushed face, nausea, vomiting, and may progress to stupor, coma and death may result from respiratory or circulation failure.

Many aliphatic hydrocarbons create a burning sensation because they are irritating to the GI mucosa. Vomiting has been reported in up to one third of all hydrocarbon exposures. While most aliphatic hydrocarbons have little GI absorption, aspiration frequently occurs, either initially or in a semi-delayed fashion as the patient coughs or vomits, thereby resulting in pulmonary effects. Once aspirated, the hydrocarbons can create a severe pneumonitis.

Rats given isoparaffinic hydrocarbons - isoalkanes- (after 18-24 hours fasting) showed lethargy and/or general weakness, ataxia and diarrhoea. Symptoms disappeared within 24-28 hours.

Following ingestion, a single exposure to isopropyl alcohol produced lethargy and non-specific effects such as weight loss and irritation. Ingestion of near-lethal doses of isopropanol produces histopathological changes of the stomach, lungs and kidneys, incoordination, lethargy, gastrointestinal tract irritation, and inactivity or anaesthesia.

Swallowing 10 ml. of isopropanol may cause serious injury; 100 ml. may be fatal if not promptly treated. The adult single lethal doses is approximately 250 ml. The toxicity of isopropanol is twice that of ethanol and the symptoms of intoxication appear to be similar except for the absence of an initial euphoric effect; gastritis and vomiting are more prominent. Ingestion may cause nausea, vomiting, and diarrhoea.

There is evidence that a slight tolerance to isopropanol may be acquired.

Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.

Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).

Accidental ingestion of the material may be damaging to the health of the individual.

Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.

Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.

 $Skin\ contact\ with\ the\ material\ may\ damage\ the\ health\ of\ the\ individual;\ systemic\ effects\ may\ result\ following\ absorption.$

Skin Contact

Ingestion

Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Spray mist may produce discomfort

Open cuts, abraded or irritated skin should not be exposed to this material

Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects.

Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

511ipa

Eye

Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..

Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage. Eye contact may cause tearing or blurring of vision.

The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.

Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Chronic exposure to tin dusts and fume results in 'stannosis' a mild form of pneumoconiosis. Chest symptoms develop several years after breathing difficulties (dyspnae) occur. No case of massive fibrosis from over-exposure to tin has been reported.

Principal route of occupational exposure to the gas is by inhalation.

Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain.

Chronic

Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals.

There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol.

Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects. NOTE: Commercial isopropanol does not contain 'isopropyl oil'. An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct 'isopropyl oil'. Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.

11.2.1. Endocrine Disruption Properties

Not Available

	844AR Acrylic ESD Coating	TOXICITY		IRRITATION
(Aerosol)		Not Available		Not Available
		TOXICITY	IRRITATION	
		Dermal (rabbit) LD50: >18000 mg/kg ^[2]	Eye (h	uman): 400 ppm
ethyl acetate	Inhalation(Mouse) LC50; >18 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]		
	Oral(Mouse) LD50; 709 mg/kg ^[2]	Skin: n	o adverse effect observed (not irritating) ^[1]	

propane

TOXICITY	IRRITATION
Inhalation(Rat) LC50; >13023 ppm4h ^[1]	Not Available

stannic oxide

TOXICITY	IRRITATION
Inhalation(Rat) LC50; >2.04 mg/l4h ^[1]	Not Available
Oral(Rat) LD50; >6600 mg/kg ^[2]	

acetone

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 20 mg/kg ^[2] Eye (human): 500 ppm - irritant	
Inhalation(Mouse) LC50; 44 mg/L4h ^[2]	Eye (rabbit): 20mg/24hr -moderate
Oral(Rat) LD50; 1738 mg/kg ^[1]	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]

iso-butane

TOXICITY	IRRITATION
Inhalation(Rat) LC50; >13023 ppm4h ^[1]	Not Available

isopropanol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 12792 mg/kg ^[1]	Eye (rabbit): 10 mg - moderate
Inhalation(Mouse) LC50; 27.2 mg/l4h ^[2]	Eye (rabbit): 100 mg - SEVERE

	Oral(Rabbit) LD50; 667 mg/kg ^[2]		(rabbit): 100mg/24hr-moderate	
	Skin (Skin (rabbit): 500 mg - mild	
	TOXICITY	IRRITATION		
propylene glycol monomethyl ether acetate, alpha-isomer	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse	effect observed (not irritating) ^[1]	
	Oral(Rat) LD50; 5155 mg/kg ^[1] Skin: no adverse effo		fect observed (not irritating) ^[1]	
	TOXICITY		IRRITATION	
barium dinonyl	Dermal (rabbit) LD50: >2000 mg/kg ^[2]		Eye (rabbit): 250 mg/5d mild	
naphthalenesulfonate	Inhalation(Rat) LC50; >5.25 mg/L4h ^[2]			
	Oral(Rat) LD50; 3000 mg/kg ^[2]			

844AR Acrylic ESD Coating (Aerosol)

Generally, linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic.

The JEFCA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods

InternationI 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

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.

For isopropanol (IPA):

recovery usually occurred.

Acute toxicity: Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat. Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock,

Repeat dose studies: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney.

ISOPROPANOL

Reproductive toxicity: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.

Developmental toxicity: The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity

Genotoxicity: All genotoxicity assays reported for isopropanol have been negative

Carcinogenicity: rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment

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.

A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I] *Shin-Etsu SDS

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. 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. A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.

The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I]

For dinonylnaphthalenes

The chemicals exhibit a very low order of toxicity to rats or rabbits by the oral, inhalation, or dermal routes.

Human sensitisation study results are available for two members of the category (dinonylnaphthalene sulfonic acid, calcium salt; dinonylnaphthalene sulfonic acid, barium salt). Neither is a sensitiser.

Based on the available toxicity results, dinonylnaphthalene sulfonic acid, barium salt appears to be the most biologically active member of the category.

for alkaryl sulfonate petroleum additives:

Mammalian Toxicology - Acute. Existing data on acute mammalian toxicity indicates a low concern for acute toxicity.

Acute oral toxicity: In all but one studies, there were no deaths that could be attributed to treatment with the test material when administered at the limit dose of 2000 or 5000 mg/kg. In some studies, the primary clinical observations were diarrhea and reduced food consumption (without a change in body weight). These effects are consistent with the gastrointestinal irritant properties of detergents in an oil-based vehicle. In other studies, decreased body weight gain or ruffled fur was observed. In one study where deaths occurred, animals were administered dose levels well above the 2000 mg/kg limit dose. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.

Acute dermal toxicity: No mortality was observed for any tested substance when administered at the limit dose of 2000 or 5000 mg/kg. The principal clinical observation was erythema and/or edema at the site of dermal application. In some cases, the cutaneous findings included dry, flaky skin, desquamation and hyperkeratosis. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER

BARIUM DINONYL NAPHTHALENESULFONATE

Acute inhalation toxicity: One member of the petroleum additive alkaryl sulfonate category (CAS RN: 6878396-0) was tested for acute inhalation toxicity (OECD Guideline 403, Acute Inhalation Toxicity). Rats were exposed whole-body to an aerosol of the substance at a nominal atmospheric concentration of 1.9 mg/L for four hours. This was the maximum attainable concentration due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. Clinical signs of toxicity during exposure included reduced activity, matted coat, and closed eyes. Clinical signs of toxicity observed post exposure included lacrimation, nasal discharge, salivation rates, matted coat, hunched appearance, soft stools and closed eyes. No treatment-related macroscopic findings were noted. The lack of mortality at a concentration just below the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for this substance.

Mammalian Toxicology - Subchronic Toxicity. Existing data from repeated-dose toxicity studies indicates minimal signs of toxicity following repeated oral exposure. Adverse effects at the site of contact were observed following repeated dermal exposure (injury to the skin) and repeated inhalation (injury to the lungs).

NOAELs rage from 49.5 mg/m3 to 1000 mg/kg/day

Mammalian Toxicology - Reproductive and Developmental Toxicity. A one-generation reproductive toxicity test was conducted on one member of the category (CAS # 115733-09-0). Exposure to the alkaryl sulfonate did not significantly impact reproduction or development and these results were bridged to the remainder of the category.

Mammalian Toxicology - Mutagenicity. Existing data from bacterial reverse mutation assays and in vitro and in vivo chromosome aberration studies indicate a low concern for mutagenicity.

Animal Irritation

An acute eye irritation study indicates that calcium dodecylbenzenesulfonate caused irritation.

Result: irritating at 0.1 ml

An acute skin study indicate that calcium dodecylbenzenesulfonate is irritant to skin 0.5 ml according to OECD GHS guidelines.

Respiratory irritation was not observed. There were no treatment-related changes in the haematological or urinalysis values in any of the animals. No signs of irritation of respiratory tract and nasal effects were observed.

For dinonylnaphthalenesulfonic acid (DNNSA) and its salts:

In general, a compound needs to be dissolved before it can be taken up from the gastro-intestinal tract after oral administration. Calcium bis(di C8-C10, branched, C9 rich, alkylnaphthalene sulphonate) (CaDNNSA) has a measured water solubility of 0.266 mg/L and therefore it is expected to dissolve into the gastro-intestinal fluids to a very limited extent. Uptake by passive diffusion is possible, but limited due to the high molecular weight of the salt (average MW 959) and its dissociation product DNNSA (MW 461). CaDNNSA has a high log Pow 6.6), which makes the compound relatively hydrophobic. This characteristic will enable micellular solubilisation by bile salts in the gastro-intestinal tract which allows some crossing of lipid biomembranes. The structure contains an ionizable group (SO3H), which might hamper diffusion across biological membranes. In addition, the molecular size of the molecule of 19 Å does not favor uptake across the biological membranes. In the 90-day study on CaDNNSA in the highest dose group 6/10 females died showing alterations in the gastro-intestinal tract, a small thymus and bone marrow atrophy. The surviving females at 1000 mg/kg bw showed similar effects and a reduced body weight (gain). The effects on the gastro-intestinal tract also became apparent in males at 300 and 1000 mg/kg bw. These animals also had a reduced body weight (gain). Other effects included changes in numbers of white blood cells, lymphocytes, platelets as well as effects on several biochemical parameters.

Macroscopy and histopathology indicated that next to the GI-tract mainly the thymus and bone marrow could be considered as potentially

affected in males at 300 mg/kg bw and above and in females at 1000 mg/kg bw. The effects on blood and blood forming organs as well as on the immune system are indicative for some absorption of the substance. This absorption may be enhanced due to the effects on the gastro intestinal tract lining.

The metabolism of DNNSA salts is mainly contingent on both the nature of the alkyl groups and the nature and extent of naphthalene ring

substitutions. There are currently no metabolism studies of CaDNNSA, however, the US EPA has evaluated the metabolism of analogs in in the sodium alkyl naphthalenesulfonate cluster (SANS), a group of sodium salts of naphthalenesulfonic acids. In a US EPA final rule for SANS, it was stated that "the 1- or 2-sulfonic acid sodium salt moieties on the naphthalene ring may provide a handle by which these compounds can be readily conjugated and eliminated." Though the available information is not definitive for CaDNNSA, where the alkyl chains are much larger than for the naphthalenesulfonic acids evaluated by EPA, it is expected that the metabolism of the substance will be a factor, enhancing elimination. If absorbed, wide distribution of the CaDNNSA throughout the body is not expected based on its molecular size (18 Å). In general, molecules of this size do not pass readily through cell membranes, thus limiting wide distribution. Excretion of CaDNNSA and its potential metabolites will occur via the bile (high molecular weight) or the urine (low molecular weight).

Calcium bis(di-C8-10, branched, C9 rich, alkylnaphthalenesulphonate) is irritating to skin and eyes. It is not corrosive.

Sensitisation

In the Buehler assay the substance was shown to be a weak skin sensitiser, while a human patch test showed no sensitization in human volunteers.

Genetic toxicity:

The Barium analog was found to be non-mutagenic in the Ames bacterial reverse mutation assay and the mouse lymphoma test (MLA). The substance was did not cause chromosomal aberrations in human peripheral lymphocytes.

Reproductive toxicity:

DNNSA (di C8-C10, branched, C9 rich, alkylnaphthalene sulphonic acid) is the major structural component of Calcium bis(di c8-c10, branched, c9 rich, alkylnaphthalene sulphonate). The OECD 422 repeat dose and reproduction/development study with DNNSA provides reliable read-across for developmental endpoints for Calcium bis(di c8-c10, branched, c9 rich, alkylnaphthalene sulphonate).

A second OECD 422 study conducted with another analog, Barium bis(di c8-c10, branched, c9 rich, alkylnaphthalene sulphonate), showed no effects on development at the highest dose in the study of 150 mg/kg/day. Together these studies show that Calcium bis(di c8-c10, branched, c9 rich, alkylnaphthalene sulphonate) is not a developmental toxin.

*REACh Dossie

Linear alkylbenzene sulfonates (LAS) are classified as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) according to CESIO (CESIO 2000). LAS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC. Linear alkylbenzene sulfonic acids (LABS) are strong acids (pKa<2) are classified as corrosive (R34)

Branched materials exhibit comparable toxicity to linear species.

Acute toxicity: The available data indicate minimal to moderate toxicity, with LD50 values ranging from 500 to 2000 mg/kg body weight (bw). Acute inhalation data also indicate a lack of significant toxicity. Available dermal exposure data also shows a lack of significant toxicity. LAS are readily absorbed by the gastrointestinal tract after oral administration in animals. LAS are not readily absorbed through the skin. The bulk is metabolised in the liver to sulfophenylic acids. The metabolites are excreted primarily via the urine and faeces. The main urinary metabolites in rats are sulfophenyl butanoic acid and sulfophenyl pentanoic acid. Accumulation of LAS or its main metabolites has not been established in any organ after repeated oral ingestion.

No serious injuries or fatalities in man have been reported following accidental ingestion of LAS-containing detergent. The main clinical signs observed after oral administration to rats of doses near or greater than the LD50 values consisted of reduced voluntary activity, diarrhoea, weakness etc. Death usually occurred within 24 hours of administration. Rats appear to be more sensitive to LAS than mice.

LAS and branched alkylbenzene sulfonates may cause irritation of the eyes, skin and mucous membranes. LAS are relatively more irritating to the skin than the corresponding branched alkylbenzene sulfonates. The potential of LAS to irritate the skin depends on the concentration applied. LAS have been classified as irritating to skin at concentrations above 20% according to EU-criteria. Human skin can tolerate contact with solution of up to 1% LAS for 24 hours resulting in only mild irritation. Application of > 5% LAS to the eyes of rabbits produced irritation. Concentration of < 0.1% LAS produced mild to no irritation.

Skin sensitization was not seen in 2,294 volunteers exposed to LAS or in 17,887 exposed to formulations of LAS.

Repeat dose toxicity: A feeding study indicated that LAS, when administered for 2 years at extremely high levels (0.5%) in the diets to rats, produced no adverse effects on growth, health or feed efficiency.

Genotoxicity: The mutagenic potential of LAS was tested using *Salmonella typhimurium* strains, using Ames test. In these studies, LAS was not mutagenic. The available long-term studies are inadequate for evaluating the carcinogenic potential of LAS in laboratory animals. The studies available (oral administration to rats and mice) do not show any evidence of carcinogenicity.

Reproductive toxicity: In general no specific effect of LAS on reproductive processes has been seen, although dosages causing maternal

toxicity may also induce some effects on reproduction. No teratogenic effects attributed to LAS exposure have been observed.

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljoministeriet (Danish Environmental Protection Agency)

For aromatic sulfonic acids

Aromatic sulfonic acids are very corrosive as was demonstrated in skin and eye irritation studies, in the acute oral studies, and in the single repeated dose oral study.

Health records from industrial manufacturing exposure, including manufacturing plant book of injuries and a physician report, show toluene-4-sulphonic acid (as handled in manufacturing plants; i.e., a 65% aqueous solution with < 5% free sulphuric acid) is an irritant to the eye and skin. **Sensitisation:**

There is a single, key study for sensitization of the aromatic sulphonic acids. None of the tested animals showed positive responses in a, well documented, GLP guinea pig sensitization study with toluene-4-sulphonic acid (CAS No. 104-15-4). The test substance can be considered a non-sensitizer in guinea pigs as none of the test animals showed a positive response to combined intradermal and topical induction followed by topical challenge.

Repeat dose toxicity:

A GLP guideline study with p-toluenesulphonic acid (CAS No. 104-15-4) reported no adverse effects to male and female rats exposed orally for 28 days. The highest dose was 500 mg/kg bw/day (>490 mg/kg bw/day based on >98% active ingredient). Therefore the NOAEL was set at 500 mg/kg bw/day.

Toxicity to reproduction:

No fertility studies are reported for the aromatic sulphonic acids. There are however studies for the chemically related hydrotrope substances that looked at reproductive organs and development of offspring. Hydrotropes are the salt form of the sulphonic acids and therefore are used as read-across for this endpoint. The 90-day oral rat and oral mouse studies and the 2-year chronic dermal rat and mouse studies with the closely related compound sodium xylene sulfonate (CAS No. 1300-72-7) included examination of sex organs of both sexes. No treatment related effects on reproductive organs were reported at doses roughly equivalent to those in the developmental toxicity study. he NOAEL for both maternal and foetal toxicity was the highest dose tested - 3000 mg/kg bw /day which is equivalent to 936 mg active ingredient per kilogram body weight per day. The conclusion of the study was no indications of developmental toxicity including teratogenesis.

Genetic toxicity:

There is a fully documented, GLP Guideline (OECD 471) Ames Test and a fully documented, GLP Guideline (OECD 473) Chromosome Aberration Test for one of the aromatic sulphonic acids, p-toluenesulphonic acid (CAS No. 104-15-4). Both tests were conducted with and without metabolic activation. The Ames test exposed up to 5000 micrograms/plate and the chromosome aberration test exposed up to 1902 micrograms per liter of the test substance. These studies conclude the substance is neither mutagenic norcytotoxic.

There is an additional, published report of an Ames Test for another of the aromatic sulphonic acids, benzenesulfonic acid (CAS No. 98-11-3). Exposures up to 10,000 micrograms/plate were done with and without metabolic activation. The conclusion is the same as for the p-toluenesulphonic acid: that is, not mutagenic and not cytotoxic.

There are no in vivo mutagenicity studies for the aromatic sulphonic acids, but there are two in vivo mouse micronucleus studies for the related hydrotropes – sodium cumene sulfonate (CAS 28348-53-0) and calcium xylene sulfonate (CAS 28088-63-3). Both are GLP-compliant Guideline mouse micronucleus studies with full documentation. Both studies conclude the test substances were not mutagenic in these assays. Disulfonic acids have not been the subject of concern.

Carcinogenicity:

There are no carcinogenicity studies for the aromatic sulphonic acids Two hydrotrope studies involve 2-year rat and mouse dermal exposures conducted under GLP. Up to 240 mg (rats) and 727 mg (mice) sodium xylenesulfonate/kg body weight in 50% ethanol were dosed 5 days per week for 104 weeks. There were no treatment related incidences of mononuclear cell leukenia, neoplasms, or nonneoplatic lesions of the skin and other organs. The increased incidence of epidermal hyperplasia may have been related to exposure to the test substance. The NOAEL was reported as 240 mg/kg bw/day for rats and 727 mg/kg bw/day for mice.

Elimination:

The US EPA has evaluated the metabolism of analogs in in the sodium alkyl naphthalenesulfonate cluster (SANS), a group of sodium salts of naphthalenesulfonic acids. In a US EPA final rule for SANS, it was stated that "the 1- or 2-sulfonic acid sodium salt moieties on the naphthalene ring may provide a handle by which these compounds can be readily conjugated and eliminated."

Toxicity information for barium sulfonates (barium salts of various alkyl and aryl sulfonic acids in oil solution):

ETHYL ACETATE & ISOPROPANOL

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

PROPANE & BARIUM DINONYL NAPHTHALENESULFONATE

No significant acute toxicological data identified in literature search.

ACETONE & ISOPROPANOL

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.

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:

X − Data either not available or does not fill the criteria for classification
✓ − Data available to make classification

SECTION 12 Ecological information

12.1. Toxicity

844AR Acrylic ESD Coating
(Aerosol)

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

	Endpoint	Test Duration (hr)	Species		Value	Source
ethyl acetate	NOEC(ECx)	72h	Algae or other aqu	atic plants	>100mg/	1 1
city acctate	EC50	48h	Crustacea		164mg/l	1
	LC50	96h	Fish		>75.6mg	/I 2
	Endpoint	Test Duration (hr)	Species		Value	Source
	EC50(ECx)	96h	•	Algae or other aquatic plants		2
propane	EC50	96h	Algae or other aqua		7.71mg/l 7.71mg/l	2
	LC50	96h	Fish			/1 2
	Endpoint	Test Duration (hr)	Species		Value	Source
	NOEC(ECx)	72h	Algae or other aqu	atic plants	9.77mg/l	2
stannic oxide	EC50	72h	Algae or other aqu	atic plants	>100mg/	
	LC50	96h	Fish		>100mg/	
	EC50	48h	Crustacea		>100mg/	L 2
	Endpoint	Test Duration (hr)	Species		Value	Source
	NOEC(ECx)	48h	Fish		0.001mg/L	4
acetone	EC50	96h	Algae or other aquatic	plants	9.873-27.684mg/	4
	LC50	96h Fish		13.303mg/L	4	
	EC50	48h	Crustacea	Crustacea 6098.4r		5
	Endpoint	Test Duration (hr)	Species		Value	Source
iso-butane	EC50(ECx)	96h	Algae or other aqua	Algae or other aquatic plants		2
iso-butane	EC50	96h	Algae or other aqua	Algae or other aquatic plants		2
	LC50	96h	Fish		24.11mg	/1 2
	Endpoint	Test Duration (hr)	Species		Value	Source
	EC50(ECx)	24h		Algae or other aquatic plants 0.0		_ 4
	EC50	96h	Algae or other aqua	·	>1000mg/	
isopropanol	EC50	72h	Algae or other aqua	-	>1000mg/	
	LC50	96h	Fish			4
	EC50	48h	Crustacea		7550mg/l	4
	Endpoint	Test Duration (hr)	Species		Value	Source
	NOEC(ECx)	336h	Fish		47.5mg/l	2
onvione alveel menemathy	EC50	72h	Algae or other aqua	atic plants	>1000mg	
opylene glycol monomethyl ether acetate, alpha-isomer	EC50	48h	Crustacea	- France	373mg/l	2
	LC50	96h	Fish		>100mg/l	2
	EC50	96h	Algae or other aqua	atic plants	>100mg/i	
barium dinonyl	Endpoint	Test Duration (hr)	Species	Value		Source
naphthalenesulfonate						

V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Tin may exist in either divalent (Sn2+) or tetravalent (Sn4+) cationic (positively charged) ions under environmental conditions. Tin(II) dominates in reduced (oxygen-poor) water, and

Tin may exist in either divalent (Sn2+) or tetravalent (Sn4+) cationic (positively charged) ions under environmental conditions. Tin(II) dominates in reduced (oxygen-poor) water, and will readily precipitate as a sulfide (SnS) or as a hydroxide (Sn(OH)2) in alkaline water. Tin(IV) readily hydrolyses, and can precipitate as a hydroxide. The solubility product of Sn(OH)4 has been measured at approximately 10 exp(-56) g/L at 25 °C. In general, tin(IV) would be expected to be the only stable ionic species in the weathering cycle.

The inwester may partition to sails and sediments. Cations such as Sn2+ and Sn4+ will generally be adsorted by soils to some extent, which reduces their mability. The generally

Tin in water may partition to soils and sediments. Cations such as Sn2+ and Sn4+ will generally be adsorbed by soils to some extent, which reduces their mobility. Tin is generally regarded as being relatively immobile in the environment. However, tin may be transported in water if it partitions to suspended sediments, but the significance of this mechanism has not been studied in detail. Transfer coefficients for tin in a soil-plant system were reported to be 0.01-0.1.

A bioconcentration factor (BCF) relates the concentration of a chemical in plants and animals to the concentration of the chemical in the medium in which they live. It was estimated that the BCFs of inorganic tin were 100, 1,000, and 3,000 for marine and freshwater plants, invertebrates, and fish, respectively. Marine algae can bioconcentrate tin(IV) ion by a factor of 1,900.

Inorganic tin cannot be degraded in the environment, but may undergo oxidation-reduction, ligand exchange, and precipitation reactions. It has been established that inorganic tin can be transformed into organometallic forms by microbial methylation. Inorganic tin may also be converted to stannane (H4Sn) in extremely anaerobic (oxygen-poor) conditions by macroalgae.

For ketones

Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds

Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrateThe higher molecular weight ketones do no form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (e.g., ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable.

Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.

Refrigerant Gas: Saturated Hydrocarbons have zero ozone depletion potential (ODP) and will photodegrade under atmospheric conditions. [Calor Gas]

Terrestrial fate: An estimated Koc value of 35 suggests that isobutane will have very high mobility in soil. Its very high Henry's Law constant, 4.08 atm-cu m/mole, (calculated from its vapor pressure and water solubility, high vapor pressure, 2611 mm Hg at 25 deg C, and low adsorptivity to soil indicate that volatilisation will be an important fate process from both moist and dry soil surfaces. Isobutane is biodegradable, especially under acclimated conditions, and may biodegrade in soil.

Aquatic fate: The estimated Koc value suggests that isobutane would not adsorb to sediment and particulate matter in the water column. Additional evidence that isobutane is not removed to sediment has been obtained from microcosm experiments. Isobutane will readily volatilise from water based on its estimated Henry's Law constant of 4.08 atm-cu m/mole. Estimated half-lives for a model river and model lake are 2.2 hr and 3.0 days, respectively. An estimated BCF value of 74 based on the log Kow suggests that isobutane will not bioconcentrate in aquatic organisms

Results indicate that gas exchange is the dominant removal mechanism for isobutane gases from the water column following a hypothetical input. The volatilisation half-lives for isobutane from the water columns in natural estuaries are estimated to be 4.4 and 6.8 days at 20 and 10 deg C, respectively.

Isobutane also biodegrades in the microcosm at a rate that is slower than for n-butane and falls between propane and ethane in susceptibility. Biodegradation of isobutane initially occurs with a half-lives of 16-26 days at 20 deg C and 33-139 days at 10 deg C, significantly slower than the loss predicted by gas exchange from typical natural estuaries. However, after a lag of 2-4 weeks, the biodegradation rate increases markedly so that in the case of chronic inputs, biodegradation can become the dominant removal mechanism

Atmospheric fate:: Isobutane is a gas at ordinary temperatures. It is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is 6.9 days, assuming a hydroxyl radical concn of 5x105 radicals per cubic cm. When isobutane was exposed to sunlight for 6 hr in a tedlar bag filled with Los Angeles air, 6% of the isobutane degraded The air contained 4529 ppb-C hydrocarbons and 870 ppb of NOX. The tropospheric loss of volatile hydrocarbons such as isobutane by wet and dry deposition are believed to be of minor importance. Indeed, isobutane assimilated into precipitation may evaporate during transport as well as being reemitted into the atmosphere after deposition. Isobutane is a contributor to the production of PAN (peroxyacyl nitrates) under photochemical smog conditions For propane:

Environmental Fate

Terrestrial fate:: An estimated Koc value of 460 determined from a log Kow of 2.36 indicates that propane is expected to have moderate mobility in soil. Volatilisation of propane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 7.07x10-1 atm-cu m/mole, derived from its vapor pressure, 7150 mm Hg, and water solubility, 62.4 mg/L. Propane is expected to volatilise from dry soil surfaces based upon its vapor pressure. Using cell suspensions of microorganisms isolated from soil and water, propane was oxidised to acetone within 24 hours, suggesting that biodegradation may be an important fate process in soil and sediment.

Aquatic fate: The estimated Koc value indicates that propane is expected to 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 41 minutes and 2.6 days, respectively. An estimated BCF of 13.1 using log Kow suggests the potential for bioconcentration in aquatic organisms is low. After 192 hr, the trace concentration of propane contained in gasoline remained unchanged for both a sterile control and a mixed culture sample collected from ground water contaminated with gasoline. This indicates that biodegradation may not be an important fate process in water

Atmospheric fate:: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and vapour pressure, 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, calculated from its rate constant of 1.15x10-12 cu cm/molecule-sec at 25 deg C. Propane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.

DO NOT discharge into sewer or waterways

for acetone: log Kow: -0.24 Half-life (hr) air: 312-1896 Half-life (hr) H2O surface water: 20 Henry's atm m3 /mol: 3.67E-05 BOD 5: 0.31-1.76.46-55% COD: 1 12-2 07 ThOD: 2.2

BCF: 0.69 **Environmental fate:**

Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water

In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source

Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.

Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades

Acetone does not concentrate in the food chain.

Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period

Drinking Water Standard: none available.

Soil Guidelines: none available

Air Quality Standards: none available.

Ecotoxicity:

Testing shows that acetone exhibits a low order of toxicity Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l

Aquatic invertebrate 2100 - 16700 mg/l Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (Tribolium confusum) and the flour moth (Ephestia kuehniella) were exposed to an airborne acetone concentration of 61.5 mg/m3. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (Entosiphon sulcatum) which yielded a 3-day NOEC of 28 mg/L.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethyl acetate	LOW (Half-life = 14 days)	LOW (Half-life = 14.71 days)
propane	LOW	LOW
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
iso-butane	HIGH	HIGH
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW

12.3. Bioaccumulative potential

•	
Ingredient	Bioaccumulation
ethyl acetate	HIGH (BCF = 3300)
propane	LOW (LogKOW = 2.36)
acetone	LOW (BCF = 0.69)
iso-butane	LOW (BCF = 1.97)
isopropanol	LOW (LogKOW = 0.05)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)

12.4. Mobility in soil

Ingredient	Mobility
ethyl acetate	LOW (KOC = 6.131)
propane	LOW (KOC = 23.74)
acetone	HIGH (KOC = 1.981)
iso-butane	LOW (KOC = 35.04)
isopropanol	HIGH (KOC = 1.06)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)

12.5.Results of PBT and vPvB assessment

	P	В	Т
Relevant available data	Not Applicable	Not Applicable	Not Applicable
PBT Criteria fulfilled?	Not Applicable	Not Applicable	Not Applicable

12.6. Endocrine Disruption Properties

Not Available

12.7. Other adverse effects

Not Available

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- ► Reduction
- ► Reuse
- Recycling
- Disposal (if all else fails)

Product / Packaging disposal

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- DO NOT allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- ▶ Consult State Land Waste Management Authority for disposal.
- Discharge contents of damaged aerosol cans at an approved site.
- ► Allow small quantities to evaporate.
- DO NOT incinerate or puncture aerosol cans.
- Bury residues and emptied aerosol cans at an approved site.

	1	 ''		
Waste treatment options	Not Available			
Sewage disposal options	Not Available			

SECTION 14 Transport information

Labels Required



Land transport (ADR-RID)

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

Air transport (ICAO-IATA / DGR)

14.1. UN number	1950			
14.2. UN proper shipping name	Aerosols, flammable			
14.3. Transport hazard class(es)	ICAO/IATA Class 2.1			
	ICAO / IATA Subrisk	isk Not Applicable		
	ERG Code 10L			
14.4. Packing group	Not Applicable			
14.5. Environmental hazard	Not Applicable			
14.6. Special precautions for user	Special provisions		A145 A167 A802	
	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)

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

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

Not Applicable

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

Product name	Group
ethyl acetate	Not Available
propane	Not Available
stannic oxide	Not Available
acetone	Not Available

Product name	Group
iso-butane	Not Available
isopropanol	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
barium dinonyl naphthalenesulfonate	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
ethyl acetate	Not Available
propane	Not Available
stannic oxide	Not Available
acetone	Not Available
iso-butane	Not Available
isopropanol	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
barium dinonyl naphthalenesulfonate	Not Available

SECTION 15 Regulatory information

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

ethyl acetate is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs) EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

propane is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

stannic oxide is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs) Europe EC Inventory

acetone is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs) EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

iso-butane is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 1) Carcinogens: category 1A (Table 3.1)/category 1 (Table 3.2)

EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 4) Mutagens: category 1B (Table 3.1)/category 2 (Table 3.2)

isopropanol is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union - European Inventory of Existing Commercial Chemical Substances

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

UK Workplace Exposure Limits (WELs)

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

UK Workplace Exposure Limits (WELs)

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

UK Workplace Exposure Limits (WELs)

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

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

UK Workplace Exposure Limits (WELs)

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

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs) EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

UK Workplace Exposure Limits (WELs)

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

National Inventory Status

National Inventory	Status	
Australia - AIIC / Australia Non-Industrial Use	Yes	
Canada - DSL	Yes	
Canada - NDSL	No (ethyl acetate; propane; stannic oxide; acetone; iso-butane; isopropanol; propylene glycol monomethyl ether acetate, alpha-isomer; barium dinonyl naphthalenesulfonate)	
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 and are not exempt from listing(see specific ingredients in brackets)	

SECTION 16 Other information

Revision Date	12/05/2021
Initial Date	09/10/2017

Full text Risk and Hazard codes

H220	Extremely flammable gas.
H225	Highly flammable liquid and vapour.
H226	Flammable liquid and vapour.
H280	Contains gas under pressure; may explode if heated.
H302	Harmful if swallowed.
H302+H332	Harmful if swallowed or if inhaled.
H315	Causes skin irritation.
H318	Causes serious eye damage.

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. For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

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

Reason For Change

A-2.01 - update to sds format and section 3