419E Premium Acrylic Conformal Coating
MG Chemicals UK Limited

Version No:**A-2.00**
Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: **07/07/2021**
Revision Date: **07/07/2021**
L.REACH.GB.EN

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

1.1. Product Identifier

Product name	419E
Synonyms	SDS Code: 419E-Liquid; 419E-P, 419E-55ML, 419E-1L, 419E-4L, 419E-20L | UFI:59A0-M0FT-000G-S3D5
Other means of identification	Premium Acrylic Conformal Coating

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

Relevant identified uses	Protective coating for printed circuit boards
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

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

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H336 - Specific target organ toxicity - single exposure Category 3 (narcotic effects), H225 - Flammable Liquid Category 2, H335 - Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), H319 - Eye Irritation Category 2, H317 - Skin Sensitizer Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567*

2.2. Label elements

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

Hazard statement(s)

H336	May cause drowsiness or dizziness.
H225	Highly flammable liquid and vapour.
H335	May cause respiratory irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.

Supplementary statement(s)

EUH066	Repeated exposure may cause skin dryness or cracking.

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Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P261	Avoid breathing mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
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.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.

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

Inhalation, skin contact and/or ingestion may produce health damage*.

Cumulative effects may result following exposure*.

HARMFUL: may cause lung damage if swallowed

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	**%[weight]**	**Name**	**Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567**	**Nanoform Particle Characteristics**
1.78-93-3 2.201-159-0 3.606-002-00-3 4.Not Available	46	methyl ethyl ketone * -	Flammable Liquid Category 2, Eye Irritation Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects); H225, H319, H336, EUH066 [2]	Not Available
1.97-85-8 2.202-612-5 3.Not Available 4.Not Available	24	iso-butyl iso-butyrate	Flammable Liquid Category 3, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation); H226, H315, H319, H335 [1]	Not Available
1.119-36-8 2.204-317-7 3.Not Available 4.Not Available	1	methyl salicylate	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Chronic Aquatic Hazard Category 2; H302, H315, H319, H317, H335, H411 [1]	Not Available
1.80-62-6 2.201-297-1 3.607-035-00-6 4.Not Available	0.1	methyl methacrylate * -	Flammable Liquid Category 2, Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation); H225, H315, H317, H335 [2]	Not Available
1.97-88-1 2.202-615-1 3.607-033-00-5 4.Not Available	0.1	n-butyl methacrylate	Flammable Liquid Category 3, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation); H226, H315, H319, H317, H335 [2]	Not Available
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn*			

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	*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 this product comes in contact with the eyes: ▸ Wash out immediately 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. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▸ 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

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
for salicylate intoxication:
· Pending gastric lavage, use emetics such as syrup of Ipecac or delay gastric emptying and absorption by swallowing a slurry of activated charcoal. Do not give ipecac after charcoal.
· Gastric lavage with water or perhaps sodium bicarbonate solution (3%-5%). Mild alkali delays salicylate absorption from the stomach and perhaps slightly from the duodenum.
· Saline catharsis with sodium or magnesium sulfate (15-30 gm in water).
· Take an immediate blood sample for an appraisal of the patient's acid-base status. A pH determination on an anaerobic sample of arterial blood is best. An analysis of the plasma salicylate concentration should be made at the same time. Laboratory controls are almost essential for the proper management of severe salicylism.
· In the presence of an established acidosis, alkali therapy is essential, but at least in an adult, alkali should be withheld until its need is demonstrated by chemical analysis. The intensity of treatment depends on the intensity of acidosis. In the presence of vomiting, intravenous sodium bicarbonate is the most satisfactory of all alkali therapy.
· Correct dehydration and hypoglycaemia (if present) by the intravenous administration of glucose in water or in isotonic saline. The administration of glucose may also serve to remedy ketosis which is often seen in poisoned children.
· Even in patients without hypoglycaemia, infusions of glucose adequate to produce distinct hyperglycaemia are recommended to prevent glucose depletion in the brain. This recommendation is based on impressive experimental data in animals.
· Renal function should be supported by correcting dehydration and incipient shock. Overhydration is not justified. An alkaline urine should be maintained by the administration of alkali if necessary with care to prevent a severe systemic alkalosis. As long as urine remains alkaline (pH above 7.5), administration of an osmotic diuretic such as mannitol or perhaps THAM is useful, but one must be careful to avoid hypokalaemia. Supplements of potassium chloride should be included in parenteral fluids.
· Small doses of barbiturates, diazepam, paraldehyde, or perhaps other sedatives (but probably not morphine) may be required to suppress extreme restlessness and convulsions.
· For hyperpyrexia, use sponge baths.
The presence of petechiae or other signs of haemorrhagic tendency calls for a large Vitamin K dose and perhaps ascorbic acid. Minor transfusions may be necessary since bleeding in salicylism is not always due to a prothrombin effect.
· Haemodialysis and haemoperfusion have proved useful in salicylate poisoning, as have peritoneal dialysis and exchange transfusions, but alkaline diuretic therapy is probably sufficient except in fulminating cases.
[*GOSSELIN, et.al.: Clinical Toxicology of Commercial Products*]
The mechanism of the toxic effect involves metabolic acidosis, respiratory alkalosis, hypoglycaemia, and potassium depletion. Salicylate poisoning is characterised by extreme acid-base disturbances, electrolyte disturbances and decreased levels of consciousness. There are differences between acute and chronic toxicity and a varying clinical picture which is dependent on the age of the patient and their kidney function. The major feature of poisoning is metabolic acidosis due to 'uncoupling of oxidative phosphorylation' which produces an increased metabolic rate, increased oxygen consumption, increased formation of carbon dioxide, increased heat production and increased utilisation of glucose. Direct stimulation of the respiratory centre leads to hyperventilation and respiratory alkalosis. This leads to compensatory increased renal excretion of bicarbonate which contributes to the metabolic acidosis which may coexist or develop subsequently. Hypoglycaemia may occur as a result of increased glucose demand, increased rates of tissue glycolysis, and impaired rate of glucose synthesis. **NOTE:** Tissue glucose levels may be lower than plasma levels. Hyperglycaemia may occur due to increased glycogenolysis. Potassium depletion occurs as a result of increased renal excretion as well as intracellular movement of potassium.
Salicylates competitively inhibit vitamin K dependent synthesis of factors II, VII, IX, X and in addition, may produce a mild dose dependent hepatitis. Salicylates are bound to albumin. The extent of protein binding is concentration dependent (and falls with higher blood levels). This, and the effects of acidosis, decreasing ionisation, means that the volume of distribution increases markedly in overdose as does CNS penetration. The extent of protein binding (50-80%) and the rate of metabolism are concentration dependent. Hepatic clearance has zero order kinetics and thus the therapeutic half-life of 2-4.5 hours but the half-life in overdose is 18-36 hours. Renal excretion is the most important route in overdose. Thus when the salicylate concentrations are in the toxic range there is increased tissue distribution and impaired clearance of the drug.
HyperTox 3.0 http://www.ozemail.com.au/-ouad/SALI0001.HTA
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.

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- **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.

5.2. Special hazards arising from the substrate or mixture

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

5.3. Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ May be violently or explosively reactive. ‣ Wear breathing apparatus plus protective gloves in the event of a fire. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Consider evacuation (or protect in place). ‣ Fight fire from a safe distance, with adequate cover. ‣ If safe, switch off electrical equipment until vapour fire hazard removed. ‣ Use water delivered as a fine spray to control the fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **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.
Fire/Explosion Hazard	‣ Liquid and vapour are highly flammable. ‣ Severe fire hazard when exposed to heat, flame and/or oxidisers. ‣ Vapour may travel a considerable distance to source of ignition. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions.

SECTION 6 Accidental release measures

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

Minor Spills	‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb small quantities with vermiculite or other absorbent material. ‣ Wipe up. ‣ Collect residues in a flammable waste container.

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Major Spills

Chemical Class: ester and ethers
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
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 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 course.
- Consider evacuation (or protect in place).
- 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.
- Contain spill with sand, earth or vermiculite.
- Use only spark-free shovels and explosion proof equipment.
- Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

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

Safe handling

- Containers, even those that have been emptied, may contain explosive vapours.
- Do NOT cut, drill, grind, weld or perform similar operations on or near containers.

Contains low boiling substance:
Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.

- Check for bulging containers.
- Vent periodically
- Always release caps or seals slowly to ensure slow dissipation of vapours
- 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, heat or ignition sources.
- When handling, **DO NOT** eat, drink or smoke.
- Vapour may ignite on pumping or pouring due to static electricity.
- **DO NOT** use plastic buckets.
- Earth and secure metal containers when dispensing or pouring product.
- Use spark-free tools when handling.
- Avoid contact with incompatible materials.
- Keep containers securely sealed.
- Avoid physical damage to containers.

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	‣ 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. ‣ **DO NOT** allow clothing wet with material to stay in contact with skin
Fire and explosion protection	See section 5
Other information	‣ Store in original containers in approved flame-proof area. ‣ No smoking, naked lights, heat or ignition sources. ‣ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped. ‣ Keep containers securely sealed. ‣ Store away from incompatible materials in a cool, dry well ventilated area. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	‣ Packing as supplied by manufacturer. ‣ Plastic containers may only be used if approved for flammable liquid. ‣ Check that containers are clearly labelled and free from leaks. ‣ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ‣ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ‣ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ‣ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ‣ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ‣ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	Methyl ethyl ketone: ‣ reacts violently with strong oxidisers, aldehydes, nitric acid, perchloric acid, potassium tert-butoxide, oleum ‣ is incompatible with inorganic acids, aliphatic amines, ammonia, caustics, isocyanates, pyridines, chlorosulfonic aid ‣ forms unstable peroxides in storage, or on contact with propanol or hydrogen peroxide ‣ attacks some plastics ‣ may generate electrostatic charges, due to low conductivity, on flow or agitation ‣ Esters react with acids to liberate heat along with alcohols and acids. ‣ Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products. ‣ Heat is also generated by the interaction of esters with caustic solutions. ‣ Flammable hydrogen is generated by mixing esters with alkali metals and hydrides. ‣ Esters may be incompatible with aliphatic amines and nitrates. Ketones in this group: ‣ are reactive with many acids and bases liberating heat and flammable gases (e.g., H2). ‣ react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H2) and heat. ‣ are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides. ‣ react violently with aldehydes, HNO3 (nitric acid), HNO3 + H2O2 (mixture of nitric acid and hydrogen peroxide), and HClO4 (perchloric acid). ‣ may react with hydrogen peroxide to form unstable peroxides; many are heat- and shock-sensitive explosives. A significant property of most ketones is that the hydrogen atoms on the carbons next to the carbonyl group are relatively acidic when compared to hydrogen atoms in typical hydrocarbons. Under strongly basic conditions these hydrogen atoms may be abstracted to form an enolate anion. This property allows ketones, especially methyl ketones, to participate in condensation reactions with other ketones and aldehydes. This type of condensation reaction is favoured by high substrate concentrations and high pH (greater than 1 wt% NaOH). ‣ Avoid strong acids, bases.

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
methyl ethyl ketone	Dermal 1 161 mg/kg bw/day (Systemic, Chronic) Inhalation 600 mg/m³ (Systemic, Chronic) *Dermal 412 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 106 mg/m³ (Systemic, Chronic) ** *Oral 31 mg/kg bw/day (Systemic, Chronic) **	55.8 mg/L (Water (Fresh)) 55.8 mg/L (Water - Intermittent release) 55.8 mg/L (Water (Marine)) 284.74 mg/kg sediment dw (Sediment (Fresh Water)) 284.7 mg/kg sediment dw (Sediment (Marine)) 22.5 mg/kg soil dw (Soil) 709 mg/L (STP) 1000 mg/kg food (Oral)
iso-butyl iso-butyrate	Inhalation 154.77 mg/m³ (Systemic, Chronic) *Inhalation 27.34 mg/m³ (Systemic, Chronic) ** *Oral 7.86 mg/kg bw/day (Systemic, Chronic) **	0.013 mg/L (Water (Fresh)) 0.001 mg/L (Water - Intermittent release) 0.13 mg/L (Water (Marine)) 0.08 mg/kg sediment dw (Sediment (Fresh Water)) 0.008 mg/kg sediment dw (Sediment (Marine)) 0.3 mg/L (STP)
methyl salicylate	Dermal 6 mg/kg bw/day (Systemic, Chronic) Inhalation 17.5 mg/m³ (Systemic, Chronic) Inhalation 285 mg/m³ (Systemic, Acute) *Dermal 3 mg/kg bw/day (Systemic, Chronic) **	20 µg/L (Water (Fresh)) 2 µg/L (Water - Intermittent release) 200 µg/L (Water (Marine)) 0.52 mg/kg sediment dw (Sediment (Fresh Water))

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Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
	*Inhalation 4 mg/m³ (Systemic, Chronic) ** *Oral 1 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 213 mg/m³ (Systemic, Acute) ** *Oral 5 mg/kg bw/day (Systemic, Acute) **	0.052 mg/kg sediment dw (Sediment (Marine)) 0.35 mg/kg soil dw (Soil) 140 mg/L (STP)
methyl methacrylate	Dermal 13.67 mg/kg bw/day (Systemic, Chronic) Inhalation 208 mg/m³ (Systemic, Chronic) Dermal 1.5 mg/cm² (Local, Chronic) Inhalation 208 mg/m³ (Local, Chronic) Dermal 1.5 mg/cm² (Local, Acute) *Dermal 8.2 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 74.3 mg/m³ (Systemic, Chronic) ** *Dermal 1.5 mg/cm² (Local, Chronic) ** *Inhalation 104 mg/m³ (Local, Chronic) ** *Dermal 1.5 mg/cm² (Local, Acute) **	0.94 mg/L (Water (Fresh)) 0.94 mg/L (Water - Intermittent release) 0.94 mg/L (Water (Marine)) 5.74 mg/kg sediment dw (Sediment (Fresh Water)) 1.47 mg/kg soil dw (Soil) 10 mg/L (STP)
n-butyl methacrylate	Dermal 5 mg/kg bw/day (Systemic, Chronic) Inhalation 415.9 mg/m³ (Systemic, Chronic) Dermal 1 % in mixture (weight basis) (Local, Chronic) Inhalation 409 mg/m³ (Local, Chronic) Dermal 1 % in mixture (weight basis) (Local, Acute) *Dermal 3 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 66.5 mg/m³ (Systemic, Chronic) ** *Dermal 1 % in mixture (weight basis) (Local, Chronic) ** *Inhalation 366.4 mg/m³ (Local, Chronic) ** *Dermal 1 % in mixture (weight basis) (Local, Acute) **	0.017 mg/L (Water (Fresh)) 0.002 mg/L (Water - Intermittent release) 0.056 mg/L (Water (Marine)) 4.73 mg/kg sediment dw (Sediment (Fresh Water)) 0.473 mg/kg sediment dw (Sediment (Marine)) 0.935 mg/kg soil dw (Soil) 31.7 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	methyl ethyl ketone	Butanone	200 ppm / 600 mg/m3	900 mg/m3 / 300 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	methyl ethyl ketone	Butan-2-one (methyl ethyl ketone)	200 ppm / 600 mg/m3	899 mg/m3 / 300 ppm	Not Available	Sk, BMGV
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	methyl methacrylate	Methyl methacrylate	50 ppm	100 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	methyl methacrylate	Methyl methacrylate	50 ppm / 208 mg/m3	416 mg/m3 / 100 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
methyl ethyl ketone	Not Available	Not Available	Not Available
iso-butyl iso-butyrate	23 mg/m3	250 mg/m3	1,500 mg/m3
methyl salicylate	2.3 ppm	25 ppm	150 ppm
methyl methacrylate	Not Available	Not Available	Not Available
n-butyl methacrylate	19 mg/m3	210 mg/m3	1,300 mg/m3

Ingredient	Original IDLH	Revised IDLH
methyl ethyl ketone	3,000 ppm	Not Available
iso-butyl iso-butyrate	Not Available	Not Available
methyl salicylate	Not Available	Not Available
methyl methacrylate	1,000 ppm	Not Available
n-butyl methacrylate	Not Available	Not Available

Occupational Exposure Banding

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

MATERIAL DATA

Fragrance substance with is an established contact allergen in humans.
Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012
IFRA Prohibited Fragrance Substance
The International Fragrance Association (IFRA) Standards form the basis for the globally accepted and recognized risk management system for the safe use of fragrance ingredients and are part of the IFRA Code of Practice. This is the self-regulating system of the industry, based on risk assessments carried out by an independent Expert Panel

Continued...

For methyl ethyl ketone:
Odour Threshold Value: Variously reported as 2 ppm and 4.8 ppm
Odour threshold: 2 ppm (detection); 5 ppm (recognition) 25 ppm (easy recognition); 300 ppm IRRITATING
Exposures at or below the recommended TLV-TWA are thought to prevent injurious systemic effects and to minimise objections to odour and irritation. Where synergism or potentiation may occur stringent control of the primary toxin (e.g. n-hexane or methyl butyl ketone) is desirable and additional consideration should be given to lowering MEK exposures.
Odour Safety Factor(OSF)
OSF=28 (METHYL ETHYL KETONE)

Odour Threshold Value (methyl methacrylate): 0.049 ppm (detection), 0.34 ppm (recognition)
NOTE: Detector tubes measuring in excess of 50 ppm, are available.
Concentrations as low as 125 ppm methyl methacrylate have produced irritation of the mucous membranes of exposed workers. The recommended TLV-TWA is thought to be sufficiently low to protect against discomfort from irritation and acute systemic intoxication.

NOTE D: Certain substances which are susceptible to spontaneous polymerisation or decomposition are generally placed on the market in a stabilised form. It is in this form that they are listed on Annex I
When they are placed on the market in a non-stabilised form, the label must state the name of the substance followed by the words 'non-stabilised'
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

8.2. Exposure controls

8.2.1. Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.
Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

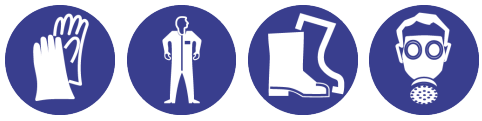
Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

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

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

8.2.2. Personal protection



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

Continued...

	NOTE: ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. For esters: ▸ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower. ▸ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▸ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▸ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
'Forsberg Clothing Performance Index'.
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
419E Premium Acrylic Conformal Coating

Material	CPI
PE/EVAL/PE	A
TEFLON	A
PVA	B
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C

Respiratory protection

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

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

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

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

▸ Cartridge respirators should never be used for emergency ingress or in areas of

Continued...

NITRILE+PVC	C
PVC	C
SARANEX-23	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as 'feel' or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

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

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	Clear

Physical state	Liquid	**Relative density (Water = 1)**	0.88
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	400
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)	80	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	-9	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	11.6	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1.8	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	6	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (%)**	Not Available
Vapour density (Air = 1)	>2.14	**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	- Unstable in the presence of incompatible materials. - 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

Continued...

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as 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. The material has **NOT** been classified by EC Directives or other classification systems as 'harmful by inhalation'. This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols. Acute exposure of humans to high concentrations of methyl ethyl ketone produces irritation to the eyes, nose, and throat. Other effects reported from acute inhalation exposure in humans include central nervous system depression, headache, and nausea. Easy odour recognition and irritant properties of methyl ethyl ketone means that high vapour levels are readily detected and should be avoided by application of control measures; however odour fatigue may occur with loss of warning of exposure. Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Large oral doses of salicylates may cause mild burning pain in the throat, stomach and usually prompt vomiting. Several hours may elapse before the development of deep and rapid breathing, lassitude, anorexia, nausea, vomiting, thirst and occasional diarrhoea. Common derivatives of salicylic acid produce substantially the same toxic syndrome, ('salicylism'). Major signs and symptoms arise from stimulation and terminal depression of the central nervous system. Stimulation produces vomiting, hyperpnea (abnormal increase in rate and depth of respiration), headache, tinnitus (ringing in the ears) confusion, bizarre behaviour or mania, generalised convulsions. Death is due to respiratory failure or cardiovascular collapse. Severe sensory disturbances such as deafness and dimness of vision are common. Less common features include sweating, skin eruptions, gastrointestinal and other hemorrhages, renal failure and pancreatitis. A tendency to bleed may be manifest by blood in the vomitus (haematemesis), bloody stools (melena) or purplish-red spots (petechiae) on the skin. Many of the toxic effects detailed here are due to or aggravated by severe disturbance of acid-base balance with the chief cause being prolonged hyperventilation from central stimulation. An assessment of acute salicylate intoxication based on dose suggests; 500 mg/kg: Potentially lethal The material has **NOT** been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. 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).
Skin Contact	Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Dermatitis has been reported in humans following dermal exposure to methyl ethyl ketone. Tests involving acute exposure of rabbits has shown methyl ethyl ketone to have high acute toxicity from dermal exposure. 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. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either: ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or ▸ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
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. The vapour when concentrated has pronounced eye irritation effects and this gives some warning of high vapour concentrations. If eye irritation occurs seek to reduce exposure with available control measures, or evacuate area.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not

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possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.
Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.
Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.
Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.
Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.
Mild chronic salicylate intoxication, or 'salicylism', may occur after repeated exposures to large doses. Symptoms include dizziness, tinnitus, deafness, sweating, nausea and vomiting, headache and mental confusion. Symptoms of more severe intoxication include hyperventilation, fever, restlessness, ketosis, and respiratory alkalosis and metabolic acidosis. Depression of the central nervous system may lead to coma, cardiovascular collapse and respiratory failure.
Chronic exposure to the salicylates (o-hydroxybenzoates) may produce metabolic and central system disturbances or damage to the kidneys.
Persons with pre-existing skin disorders, eye problems or impaired kidney function may be more susceptible to the effects of these substances.
Certain individuals (atopics), notably asthmatics, exhibit significant hyper- sensitivity to salicylic acid derivatives. Reactions include urticaria and other skin eruptions, rhinitis and severe (even fatal) bronchospasm and dyspnea. Chronic exposure to the p-hydroxybenzoates (parabens) is associated with hypersensitivity reactions following application of these to the skin. Hypersensitivity reactions have also been reported following parenteral or oral administration. Cross-sensitivity occurs between the p-hydroxybenzoates Hypersensitivity reactions may include by acute bronchospasm, hives (urticaria), deep dermal wheals (angioneurotic oedema), running nose (rhinitis) and blurred vision. Anaphylactic shock and skin rash (non- thrombocytopenic purpura) may also occur. Any individual may be predisposed to such anti-body mediated reaction if other chemical agents have caused prior sensitisation (cross-sensitivity).
Limited information is available on the chronic (long-term) effects of methyl ethyl ketone in humans. Chronic inhalation studies in animals have reported slight neurological, liver, kidney, and respiratory effects. No information is available on the developmental, reproductive, or carcinogenic effects of methyl ethyl ketone in humans. Developmental effects, including decreased foetal weight and foetal malformations, have been reported in mice and rats exposed to methyl ethyl ketone via inhalation and ingestion.
Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities.
Combinations with chloroform also show increase in toxicity

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TOXICITY	IRRITATION
Not Available	Not Available

methyl ethyl ketone

TOXICITY	IRRITATION
Dermal (rabbit) LD50: ~6400-8000 mg/kg[2]	Eye (human): 350 ppm -irritant
Inhalation(Mouse) LC50; 32 mg/L4h[2]	Eye (rabbit): 80 mg - irritant
Oral(Rat) LD50; 2054 mg/kg[1]	Skin (rabbit): 402 mg/24 hr - mild
	Skin (rabbit):13.78mg/24 hr open

iso-butyl iso-butyrate

TOXICITY	IRRITATION
dermal (guinea pig) LD50: >8550 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Oral(Rat) LD50; >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

methyl salicylate

TOXICITY	IRRITATION
dermal (guinea pig) LD50: ~700 mg/kg[2]	Eye (rabbit): 500 mg/24 h - mild
Inhalation(Rat) LC50; >0.225 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
Oral(Mouse) LD50; 580 mg/kg[1]	Skin (rabbit): 500 mg/24 h - moderate
	Skin: no adverse effect observed (not irritating)[1]

methyl methacrylate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg[2]	Eye (rabbit): 150 mg
Inhalation(Rat) LC50; 29.8 mg/l4h[1]	Skin (rabbit): 10000 mg/kg (open)
Oral(Mouse) LD50; 3625 mg/kg[2]	

n-butyl methacrylate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; 4910 ppm4h[2]	Skin (rabbit): 10000 mg/kg (open)
Oral(Rabbit) LD50; >6300 mg/kg[1]	Skin: adverse effect observed (irritating)[1]

Legend: *1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

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419E Premium Acrylic Conformal Coating	Generally,linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic. The JEFCA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods **Internationl Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)** **Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998**
METHYL ETHYL KETONE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
METHYL SALICYLATE	Not irritating to human skin at concentrations of 8% in mineral oil* Not sensitising to human skin at concentrations of 8% in mineral oil* Not sensitising to guinea pig (Magnusson and Kligman method) * Not irritating to rabbits on ocular application * Ames test: negative* * Rhodia MSDS For certain benzyl derivatives: All members of this group (benzyl, benzoate and 2-hydroxybenzoate (salicylate) esters) contain a benzene ring bonded directly to an oxygenated functional group (aldehyde or ester) that is hydrolysed and/or oxidised to a benzoic acid derivative. As a stable animal metabolite, benzoic acid derivatives are efficiently excreted primarily in the urine. These reaction pathways have been reported in both aquatic and terrestrial species. The similarity of their toxicologic properties is a reflection their participation in these common metabolic pathways. In general, members of this group are rapidly absorbed through the gastrointestinal tract, metabolised primarily in the liver, and excreted in the urine either unchanged or as conjugates of benzoic acid derivatives At high doses, conjugation pathways (*e.g.,* glycine) may be saturated; in which case, free benzoic acid is excreted unchanged. Absorption, distribution and excretion studies have been conducted several members of this group and structural relatives. These substances exhibit remarkably similar patterns of pharmacokinetics and metabolism. The benzyl, benzoate, and 2-hydroxybenzoate (salicylate) esters which comprise this category are hydrolysed to the corresponding alcohols and carboxylic acids. The benzyl alcohol and benzaldehyde derivatives are oxidised to the corresponding benzoic acid derivatives that are subsequently excreted unchanged or as glycine or glucuronic acid conjugates. If methoxy or phenolic functional groups are present on the benzene ring, additional minor metabolic options become available. *O*-demethylation yields the corresponding phenol that is subsequently excreted as the glucuronic acid or sulfate conjugates. At high dose levels, gut microflora may act to produce minor amounts of reduction metabolites. **Acute toxicity**: Oral LD50 values ranged from 887 to greater than 5,000 mg/kg bw demonstrating the low to moderate toxicity of these compounds. **Repeat dose toxicity:** Overall, numerous repeat-dose studies using various routes of exposure have been conducted in different animal species with members of this chemical category or their close structural relatives. It is important to note that all the benzyl derivatives in this category are eventually metabolised to a common metabolite, benzoic acid, and are rapidly excreted in the urine as benzoic acid or as its glycine, sulfate, or glucuronic acid conjugate. For this reason, the repeat-dose studies currently available provide adequate support for the safety of the benzyl derivatives. Moreover, the levels at which no adverse effects were reported were sufficiently high to accommodate any potential differences among the members of the category. **Reproductive toxicity:** Several reproductive toxicity studies have been conducted with representatives of this group and produced no evidence of reproductive toxicity As with the repeat-dose studies, the benzyl derivatives generally follow the similar metabolic pathways and the studies conducted provide an adequate database for this endpoint. In addition, the dose levels tested provide margins of safety large enough to accommodate any differences among the group. **Developmental toxicity:** Representative substances from this group were tested for developmental toxicity with uniform results, and indicated no teratogenic potential in the absence of maternal toxicity. Again, the representative substances undergo similar metabolism to the entire benzyl derivative group and therefore, provide an adequate representation for this endpoint. **Genetic toxicity:** Overall, *in vitro* and *in vivo* genotoxicity studies have been conducted with substances representing the structural characteristics of the benzyl category. The results of these studies were predominantly negative demonstrating a low order of genotoxic potential.. Limited positive and/or equivocal findings have been reported for 3 aldehydes and benzyl acetate, but, in most cases, other studies of the same endpoint with same test substance show no activity. Most importantly, *in vivo* studies on benzaldehyde derivatives and closely related benzyl esters have all yielded negative results. These negative *in vivo* genotoxicity assays are supported by the lack of tumorigenicity in chronic animal studies with representatives of this group. Data available for more than 100 *in vitro* genotoxicity assays for 9 members of the category and five metabolic precursors or metabolites of benzyl derivatives indicate a low genotoxic potential for members of this chemical category Equivocal results have been reported mainly for aromatic aldehydes in the MLA and ABS assays. A member or analogue of a group of hydroxy and alkoxy-substituted benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption. metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances. All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals. The structural features common to all members of the group is a primary oxygenated functional group bonded directly to a benzene ring. The ring also contains hydroxy or alkoxy substituents. The hydroxy- and alkoxy- substituted benzyl derivatives are raidly absorbed by the gastrointestinal tract, metabolised in the liver to yield benzoic acid derivatives and excreted primarily in the urine either unchanged or conjugated. It is expected than aromatic esters and acetals will be hydrolysed in vivo through the catalytic activity of carboxylesterases, (A-esterases), Acetals hydrolyse uncatalysed in gastric juices and intestinal fluids to yield acetaldehydes. Substituted benzyl esters and benzaldehyde acetals are hydrolysed to the corresponding alcoholic alcohols and carboxylic acid. In general hydroxy- and alkoxy- derivatives of benzaldehyde and benzyl alcohol are oxidised to the corresponding benzoic aid derivatives and, to a lesser extent reduced to corresponding benzyl alcohol derivatives. Following conjugation these are excreted in the urine. Benzyl alcohol derivatives may also be reduced in gut microflora to toluene derivatives. Flavor and Extract Manufacturers Association (FEMA) The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
METHYL METHACRYLATE	**For methyl methacrylate:** Acute toxicity: MMA is rapidly absorbed after oral or inhalatory administration. *In vitro* skin absorption studies in human skin indicate that MMA can be absorbed through human skin. After inhalation to rats 10 to 20% of the substance is deposited in the upper respiratory tract where it is

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	metabolised by local tissue esterases. Acute toxicity of MMA by the oral, dermal, and inhalative routes is low as judged by tests with different species: The oral LD50 for rats, mice, and rabbits is found to exceed 5000 mg/kg bw. Acute inhalation toxicity for rats and mice is described by LC50 values of > 25 mg/l/4 hours. Acute dermal toxicity is reported for rabbits to exceed 5000 mg/kg bw. Skin and respiratory irritation are reported for subjects exposed to monomeric MMA. The substance has been shown to produce severe skin irritation when tested undiluted on rabbit skin. There are indications from studies in animals that MMA can be irritating to the respiratory system. In contact with eyes MMA has shown only weak irritation of the conjunctivae. MMA has a moderate to strong sensitising potential in experimental animals**.** Cases of contact dermatitis have been reported for workers exposed to the monomeric chemical. There is no convincing evidence that MMA is a respiratory sensitiser in humans. The lead effect caused by MMA is a degeneration of the olfactory region of the nose being the most sensitive target tissue. For this effect a NOAEC of 25 ppm (104 mg/m3) in a two-year inhalation study in rats was identified but only slight effects on the olfactory tissues have been observed at 100 ppm. Concerning systemic effects, two different valid studies have been considered for identifying a N(L)OAEL. Due to different dose selections, different values for N(L)OEALs are available. The LOEALs and the NOEALs for female rats ranges between 400 and 500 ppm and from 100 to 250 ppm respectively. In subchronic inhalation studies systemic toxic effects were seen in rats >1000 ppm, respectively in mice >500 ppm, including degenerative and necrotic lesions in liver, kidney, brain, and atrophic changes in spleen and bone marrow. These effects were not seen in chronic studies up to 1000 ppm. Oral administration to rats resulted in a NOAEL of 200 mg/kg bw/d. MMA has *in vitro* the potential for induction of mutagenic effects, especially clastogenicity. However, this potential is limited to high doses with strong toxic effects. Furthermore, the negative *in vivo* micronucleus test and the negative dominant lethal assay indicate that this potential is not expressed *in vivo*. There is no relevant concern on carcinogenicity of MMA in humans and animals. Epidemiology data on increased tumour rates in exposed cohorts are of limited reliability and cannot be related to MMA as the solely causal agent. MMA did not reveal an effect on male fertility when animals had been exposed to up to 9000 ppm. From the available developmental toxicity investigations, including an inhalation study according to OECD Guideline 414, no teratogenicity, embryotoxicity or foetotoxicity has been observed at exposure levels up to and including 2028 ppm (8425 mg/m3). The available human data on sexual disorders in male and female workers cannot be considered to conclude on reproductive toxicity effects of MMA due to the uncertain validity of the studies 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. Inhalation (human) TCLo: 60 mg/m3(15 ppm) [* Manuf. Rohm & Haas]
N-BUTYL METHACRYLATE	For iso-butyl methacrylate (i-BMA) and n-butyl methacrylate (n-BMA): **Acute toxicity:** It is anticipated that BMA is absorbed after oral or inhalation exposure. In vitro studies using isolated rat liver microsomes or porcine liver esterase showed rapid hydrolysis of n-BMA yielding methacrylic acid and n-butanol. No in vivo metabolism data is available on n-BMA/ i-BMA, but from the in vitro data rapid hydrolysis to methacrylic acid and the corresponding alcohol can be anticipated. n-BMA did not bind to glutathione (GSH) in vitro. It is expected that after hydrolysis the respective cleavage products, methacrylic acid and n-butanol or or isobutanol are further metabolised to CO2. In mammals n-BMA/ i-BMA is of low oral toxicity by the oral, dermal or inhalation route. The have local irritating properties to rabbit skin and eyes. Respiratory tract irritation was observed after inhalation exposure to rats of n-BMA. Whilst n-BMA is a weak skin sensitiser in guinea pigs there is no such evidence for i-BMA. From available human clinical data it can be concluded that the sensitisation potential to humans of n-BMA is low. **Repeat dose toxicity:** A repeat dose oral study of limited reliability, indicates that n-BMA is of low oral toxicity. A reliable 28-day exposure inhalation study in rats, for n-BMA demonstrated the formation of nasal lesions indicative of a local irritant effect of the nose without indication of systemic toxicity. **Genotoxicity:** Neither n-BMA nor i-BMA was mutagenic in a number of gene mutation assays with Salmonella typhimurium. i-BMA was not clastogenic in a mouse micronucleus assay. There appears to be little concern for genotoxicity despite limited data. **Carcinogenicity:** Given the lack of carcinogenicity observed with methyl methacrylic (the metabolite) and the lack of genotoxic potential there appears to be little concern for possible carcinogenicity of BMA. Neither isobutanol or n-butanol exhibit carcinogenic potential. **Developmental toxicity:** Available data for methyl methacrylate and n-butanol an isobutanol suggests that there is little concern for possible developmental effects arising out of inhalation exposure to non-maternally toxic concentrations of n-BMA/ i-BMA. **Repeat dose toxicity:** Limited data from repeated dose studies with n-BMA, methyl methacrylate, methacrylic acid and a fertility study with n-butanol did not reveal an y indications for possible toxicity on the reproductive organ
419E Premium Acrylic Conformal Coating & METHYL ETHYL KETONE & ISO-BUTYL ISO-BUTYRATE & METHYL SALICYLATE & METHYL METHACRYLATE & N-BUTYL METHACRYLATE	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.
419E Premium Acrylic Conformal Coating & METHYL SALICYLATE & METHYL METHACRYLATE & N-BUTYL METHACRYLATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
419E Premium Acrylic Conformal Coating & METHYL SALICYLATE	Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to 'perfume mix'. The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits. Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water. Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis. Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact

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allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic.

Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

Pigmentary anomalies: The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or as a prohapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.

Prohaptens

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.

In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation

The Research Institute for Fragrance Materials (RIFM) Expert Panel study of fragrance salicylates concluded.

The salicylates are well absorbed by the oral route, and oral bioavailability is assumed to be 100%. Absorption by the dermal route in humans is more limited with bioavailability in the range of 11.8-30.7%.

The salicylates are expected to undergo extensive hydrolysis, primarily in the liver, to salicylic acid which is conjugated with either glycine or glucuronide and is excreted in the urine as salicyluric acid and acyl and phenolic glucuronides. The hydrolyzed side chains are metabolized by common and well-characterized metabolic pathways leading to the formation of innocuous end products. The expected metabolism of the salicylates does not present toxicological concerns.

The acute dermal toxicity of the salicylates is very low, with LD50 values in rabbits reported to be greater than 5000 mg/kg body weight. The

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	acute oral toxicity of the salicylates is moderate, with toxicity generally decreasing with increasing size of the ester R-group and with LD50's between 1000 and >5000 g/kg. In dermal subchronic toxicity studies, extreme doses of methyl salicylate (5 g/kg body weight/day) possibly were nephrotoxic but the data were minimal. The subchronic oral NOAEL is concluded to be 50 mg/kg body weight/day. Genetic toxicity data, for methyl salicylate, a few other salicylates and for structurally related alkyl- and alkoxy-benzyl derivatives are negative for genotoxicity. Given the metabolism of salicylate and the evidence that they are non-genotoxic, it can be concluded that the salicylates are without carcinogenic potential. The reproductive and developmental toxicity data on methyl salicylate demonstrate that high, maternally toxic doses result in a pattern of embryotoxicity and teratogenesis similar to that characterized for salicylic acid. At concentrations likely to be encountered by humans through the use of the salicylates as fragrance ingredients, these chemicals are considered to be non-irritating to the skin. The salicylates (with the exception of benzyl salicylate) in general have no or very limited skin sensitization potential. The salicylates are non-phototoxic and have no photoirritant or photoallergenic activity The use of the salicylates in fragrances produces low levels of exposure relative to doses that elicit adverse systemic effects in laboratory animals exposed by the dermal or oral route. Based on NOAEL values of 50 mg/kg body weight/day identified in the subchronic and the chronic toxicity studies , a margin of safety for systemic exposure of humans to the individual salicylates in cosmetic products, may be calculated to range from 125 to 2,500,000 (depending upon the assumption of either 12–30% or 100% bioavailability following dermal application) times the maximum daily exposure. The acute dermal toxicity of the salicylates is very low. Rabbit dermal LD50 values have been reported to be >5000 mg/kg body weight for 15 of the 16 salicylates tested, findings likely related to the limited degree of dermal absorption, the retention of salicylate in the skin, and the relatively moderate toxicity of salicylic acid itself upon systemic exposure (i.e., oral LD50 value of 891 mg/kg body weight in rats) . Overall, the acute oral toxicity of the salicylates is moderate, with toxicity generally decreasing with increasing size of the ester R-group. For the longer carbon chain salicylates, acute oral LD50's range from 1320 to >5000 mg/kg body weight. The acute oral toxicity of the unsaturated salicylates is likewise low to moderate with rat oral LD50's in the 3200 to >5000 mg/kg body weight range as are the acute oral toxicities of the aromatic salicylates (1300 to >5000 mg/kg body weight) The 17 compounds assessed in this report include the core salicylate moiety that upon hydrolysis yield salicylic acid and the alcohol of the corresponding alkyl, alkenyl, benzyl, phenyl, phenethyl, etc. side chain. This is consistent with information on other alkyl- and alkoxy- benzyl derivatives whereby aromatic esters are hydrolyzed in vivo by carboxylesterases, or esterases, especially the A-esterases. Potential differences in the metabolism of the individual salicylates would be related to the manner in which the hydrolyzed side chain undergoes further oxidation/reduction and/or conjugation reactions. Salicylic acid undergoes metabolism primarily in the liver. At low, non-toxic doses, approximately 80% of salicylic acid is further metabolized in the liver via conjugation with glycine and subsequent formation of salicyluric acid. For each of the salicylates, following hydrolysis to salicylic acid, the resulting side chains, hydroxylated alkyl, alkenyl, and phenyl moieties, could be expected to be further metabolized. In the case of the alcohols formed following hydrolysis. Further metabolism would result in the formation of the corresponding aldehydes and acids, with eventual degradation to CO2 by the fatty acid pathway and the tricarboxylic acid cycle. The secondary alcohols formed by hydrolysis of isobutyl and isoamyl salicylate, would primarily be conjugated with glucuronic acid and excreted. They could also interconvert to the corresponding ketones. Salicylates bearing alkenyl side chains, may undergo epoxidation and subsequent hydroxylation at points of unsaturation. However, since both the alkyl and alkenyl side chains would be hydroxylated at one terminus following hydrolysis of the corresponding salicylate, a significant proportion of these hydrolysis products would be excreted in the urine precluding further metabolism and epoxidation. In the case of hydrolysis of the salicylates containing aromatic side chains, phenyl salicylate and benzyl salicylate, phenol and benzyl alcohol, respectively, would be formed. Salicylates were potent and selective inhibitors for AKR1C1 enzymes , a family of aldo-keto reductases implicated in biosynthesis, intermediary metabolism and detoxification.
419E Premium Acrylic Conformal Coating & METHYL ETHYL KETONE	Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities. Combinations with chloroform also show increase in toxicity
METHYL METHACRYLATE & N-BUTYL METHACRYLATE	Where no 'official' classification for acrylates and methacrylates exists, there has been cautious attempts to create classifications in the absence of contrary evidence. For example Monalkyl or monoarylesters of acrylic acids should be classified as R36/37/38 and R51/53 Monoalkyl or monoaryl esters of methacrylic acid should be classified as R36/37/38 Based on the available oncogenicity data and without a better understanding of the carcinogenic mechanism the Health and Environmental Review Division (HERD), Office of Toxic Substances (OTS), of the US EPA previously concluded that all chemicals that contain the acrylate or methacrylate moiety (CH2=CHCOO or CH2=C(CH3)COO) should be considered to be a carcinogenic hazard unless shown otherwise by adequate testing. This position has now been revised and acrylates and methacrylates are no longer *de facto* carcinogens.

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

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

11.2.1. Endocrine Disruption Properties

Not Available

SECTION 12 Ecological information

12.1. Toxicity

419E Premium Acrylic Conformal Coating

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

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methyl ethyl ketone

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

iso-butyl iso-butyrate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	4.7mg/l	2
EC50	72h	Algae or other aquatic plants	12mg/l	2
LC50	96h	Fish	12.5mg/l	2
EC50	48h	Crustacea	55.8mg/l	2

methyl salicylate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	1.1mg/l	2
EC50	48h	Crustacea	28mg/l	2
LC50	96h	Fish	19.8mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.79mg/l	2

methyl methacrylate

Endpoint	Test Duration (hr)	Species	Value	Source
EC0(ECx)	48h	Crustacea	48mg/l	1
EC50	72h	Algae or other aquatic plants	>110mg/l	2
LC50	96h	Fish	>79mg/l	2
EC50	48h	Crustacea	69mg/l	1
EC50	96h	Algae or other aquatic plants	170mg/l	1

n-butyl methacrylate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	48h	Crustacea	23mg/l	1
EC50	72h	Algae or other aquatic plants	31.2mg/l	2
LC50	96h	Fish	5.57mg/l	2
EC50	96h	Algae or other aquatic plants	57mg/l	1
EC50	48h	Crustacea	32mg/l	1

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

For methyl ethyl ketone:
log Kow : 0.26-0.69
log Koc : 0.69
Koc : 34
Half-life (hr) air : 2.3
Half-life (hr) H2O surface water : 72-288
Henry's atm m3 /mol: 1.05E-05
BOD 5 : 1.5-2.24, 46%
COD : 2.2-2.31, 100%
ThOD : 2.44
BCF : 1

Environmental fate:

TERRESTRIAL FATE: Measured Koc values of 29 and 34 were obtained for methyl ethyl ketone in silt loams. Methyl ethyl ketone is expected to have very high mobility in soil. Volatilisation of methyl ethyl ketone from dry soil surfaces is expected based upon an experimental vapor pressure of 91 mm Hg at 25 deg C. Volatilization from moist soil surfaces is also expected given the measured Henry's Law constant of 4.7x10-5 atm-cu m/mole. The volatilisation half-life of methyl ethyl ketone from silt and sandy loams was measured as 4.9 days. Methyl ethyl ketone is expected to biodegrade under both aerobic and anaerobic conditions as indicated by numerous screening tests.
AQUATIC FATE: Based on Koc values, methyl ethyl ketone is not expected to adsorb to suspended solids and sediment in water. Methyl ethyl ketone is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated half-lives for a model river and model lake are 19 and 197, hours respectively. Biodegradation of this compound is expected based upon numerous screening tests. An estimated BCF value of 1 based on an experimental log Kow of 0.29, suggests that bioconcentration in aquatic organisms is low.
ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, methyl ethyl ketone, which has an experimental vapor pressure of 91 mm Hg at 25 deg C, will exist solely as a vapor in the ambient atmosphere. Vapour-phase methyl ethyl ketone is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 14 days. Methyl ethyl ketone is also expected to undergo photodecomposition in the atmosphere by natural sunlight. Photochemical degradation of methyl ethyl ketone by natural sunlight is expected to occur at approximately 1/5 the rate of degradation by photochemically produced hydroxyl radicals.

Ecotoxicity:

Fish LC50 (24 h): bluegill sunfish (Lepomis macrochirus) 1690-5640 mg/l; guppy (Lebistes reticulatus) 5700 mg/l; goldfish (Carassius auratus) >5000 mg/l
Fish LC50 (96 h): fathead minnow (Pimephales promelas) 3200 mg/l; bluegill sunfish (Lepomis macrochirus) 4467 mg/l; mosquito fish (Gambusia affinis) 5600 mg/l
Daphnia magna LC50 (48 h):<520-1382 mg/l
Daphnia magna LC50 (24 h): 8890 mg/l
Brine shrimp (Artemia salina) LC50 (24 h): 1950 mg/l

Continued...

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.
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
iso-butyl iso-butyrate	LOW	LOW
methyl salicylate	LOW	LOW
methyl methacrylate	LOW	LOW
n-butyl methacrylate	LOW	LOW

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
methyl ethyl ketone	LOW (LogKOW = 0.29)
iso-butyl iso-butyrate	LOW (LogKOW = 2.6816)
methyl salicylate	LOW (LogKOW = 2.55)
methyl methacrylate	LOW (BCF = 6.6)
n-butyl methacrylate	LOW (BCF = 114)

12.4. Mobility in soil

Ingredient	Mobility
methyl ethyl ketone	MEDIUM (KOC = 3.827)
iso-butyl iso-butyrate	LOW (KOC = 53.31)
methyl salicylate	LOW (KOC = 128.2)
methyl methacrylate	LOW (KOC = 10.14)
n-butyl methacrylate	LOW (KOC = 63.6)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗

PBT Criteria fulfilled?	No
vPvB	No

12.6. Endocrine Disruption Properties

Not Available

12.7. Other adverse effects

Not Available

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been

Continued...

	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. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 Transport information

Labels Required

	 Limited Quantity: 419E-55ML, 419E-1L, 419E-4L

Land transport (ADR-RID)

14.1. **UN number**	1263
14.2. **UN proper shipping name**	PAINT or PAINT RELATED MATERIAL
14.3. **Transport hazard class(es)**	Class: 3 Subrisk: Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Hazard identification (Kemler): 33 Classification code: F1 Hazard Label: 3 Special provisions: 163 367 640C 650 640D Limited quantity: 5 L Tunnel Restriction Code: 2 (D/E)

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	1263
14.2. **UN proper shipping name**	Paint related material (including paint thinning or reducing compounds); Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
14.4. **Packing group**	II
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: A3 A72 A192 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L Passenger and Cargo Packing Instructions: 353 Passenger and Cargo Maximum Qty / Pack: 5 L Passenger and Cargo Limited Quantity Packing Instructions: Y341 Passenger and Cargo Limited Maximum Qty / Pack: 1 L

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	1263
14.2. **UN proper shipping name**	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)

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14.3. **Transport hazard class(es)**	IMDG Class: 3 IMDG Subrisk: Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	EMS Number: F-E , S-E Special provisions: 163 367 Limited Quantities: 5 L

Inland waterways transport (ADN)

14.1. **UN number**	1263
14.2. **UN proper shipping name**	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning and reducing compound)
14.3. **Transport hazard class(es)**	3 | Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Classification code: F1 Special provisions: 163; 367; 640C; 640D; 650 Limited quantity: 5 L Equipment required: PP, EX, A Fire cones number: 1

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
methyl ethyl ketone	Not Available
iso-butyl iso-butyrate	Not Available
methyl salicylate	Not Available
methyl methacrylate	Not Available
n-butyl methacrylate	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
methyl ethyl ketone	Not Available
iso-butyl iso-butyrate	Not Available
methyl salicylate	Not Available
methyl methacrylate	Not Available
n-butyl methacrylate	Not Available

SECTION 15 Regulatory information

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

methyl ethyl ketone is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
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

iso-butyl iso-butyrate is found on the following regulatory lists

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

methyl salicylate is found on the following regulatory lists

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

methyl methacrylate is found on the following regulatory lists

Continued...

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
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
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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

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 (methyl ethyl ketone; iso-butyl iso-butyrate; methyl salicylate; methyl methacrylate; n-butyl methacrylate)
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	07/07/2021
Initial Date	28/11/2018

Full text Risk and Hazard codes

H226	Flammable liquid and vapour.
H302	Harmful if swallowed.
H315	Causes skin irritation.
H411	Toxic to aquatic life with long lasting effects.

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

Continued...

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-2.00 - Update to chemical ingredients based on new supplier information.