408C Rubber Renue
MG Chemicals UK Limited

Version No: **A-1.01**
Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: **21/04/2021**
Revision Date: **01/09/2021**
L.REACH.GB.EN

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

1.1. Product Identifier

Product name	408C
Synonyms	SDS Code: 408C-Liquid; 408C-125ML, 408C-225ML, 408C-1L | UFI:A3R0-109X-N006-CY6S
Other means of identification	Rubber Renue

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

Relevant identified uses	Liquid for rejuvenating and reconditioning rubber.
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

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments [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), H315 - Skin Corrosion/Irritation Category 2, H319 - Eye Irritation Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

2.2. Label elements

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

Hazard statement(s)

H336	May cause drowsiness or dizziness.
H225	Highly flammable liquid and vapour.
H335	May cause respiratory irritation.
H315	Causes skin irritation.
H319	Causes serious eye irritation.

Supplementary statement(s)

Not Applicable

Page 1 continued...

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.
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.
P280	Wear protective gloves/protective clothing/eye protection/face protection/hearing protection.

Precautionary statement(s) Response

P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
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.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
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.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Cumulative effects may result following exposure*.

isopropanol	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)

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**	**Classification according to regulation (EC) No 1272/2008 [CLP] and amendments**	**Nanoform Particle Characteristics**
1.67-63-0 2.200-661-7 3.603-117-00-0 4.Not Available	73	isopropanol	Flammable Liquid Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Eye Irritation Category 2; H225, H336, H319 [2]	Not Available
1.119-36-8 2.204-317-7 3.Not Available 4.Not Available	27	methyl salicylate	Acute Toxicity (Oral) Category 4, Eye Irritation Category 2, Chronic Aquatic Hazard Category 2, Skin Corrosion/Irritation Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Skin Sensitizer Category 1; H302, H319, H411, H315, H335, H317 [1]	Not Available
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties*			

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	If 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.

Continued...

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

Continued...

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 acute or short term repeated exposures to isopropanol:
- Rapid onset respiratory depression and hypotension indicates serious ingestions that require careful cardiac and respiratory monitoring together with immediate intravenous access.
- Rapid absorption precludes the usefulness of emesis or lavage 2 hours post-ingestion. Activated charcoal and cathartics are not clinically useful. Ipecac is most useful when given 30 mins. post-ingestion.
- There are no antidotes.
- Management is supportive. Treat hypotension with fluids followed by vasopressors.
- Watch closely, within the first few hours for respiratory depression; follow arterial blood gases and tidal volumes.
- Ice water lavage and serial haemoglobin levels are indicated for those patients with evidence of gastrointestinal bleeding.

For acute and short term repeated exposures to methanol:
- Toxicity results from accumulation of formaldehyde/formic acid.
- Clinical signs are usually limited to CNS, eyes and GI tract Severe metabolic acidosis may produce dyspnea and profound systemic effects which may become intractable. All symptomatic patients should have arterial pH measured. Evaluate airway, breathing and circulation.
- Stabilise obtunded patients by giving naloxone, glucose and thiamine.
- Decontaminate with Ipecac or lavage for patients presenting 2 hours post-ingestion. Charcoal does not absorb well; the usefulness of cathartic is not established.
- Forced diuresis is not effective; haemodialysis is recommended where peak methanol levels exceed 50 mg/dL (this correlates with serum bicarbonate levels below 18 meq/L).
- Ethanol, maintained at levels between 100 and 150 mg/dL, inhibits formation of toxic metabolites and may be indicated when peak methanol levels exceed 20 mg/dL. An intravenous solution of ethanol in D5W is optimal.
- Folate, as leucovorin, may increase the oxidative removal of formic acid. 4-methylpyrazole may be an effective adjunct in the treatment. 8.Phenytoin may be preferable to diazepam for controlling seizure.

[Ellenhorn Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI

Determinant	Index	Sampling Time	Comment
1. Methanol in urine	15 mg/l	End of shift	B, NS
2. Formic acid in urine	80 mg/gm creatinine	Before the shift at end of workweek	B, NS

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

NS: Non-specific determinant - observed following exposure to other materials.

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

Continued...

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. **WARNING**: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.

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	Environmental hazard - contain spillage. ▸ 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.
Major Spills	Environmental hazard - contain spillage. Chemical Class: alcohols and glycols 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 - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
foamed glass - pillow	4	throw	pichfork	R, P, DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
polypropylene - particulate	2	blower	skiploader	W, SS, DGC
sorbent clay - particulate	2	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	3	throw	skiploader	DGC, RT
expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC
polyurethane - mat	4	throw	skiploader	DGC, RT

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

Continued...

	▸ 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. ▸ 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. ▸ 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.
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	▸ **DO NOT** use aluminium or galvanised containers ▸ 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	Isopropanol (syn: isopropyl alcohol, IPA): ▸ forms ketones and unstable peroxides on contact with air or oxygen; the presence of ketones especially methyl ethyl ketone (MEK, 2-butanone) will accelerate the rate of peroxidation ▸ reacts violently with strong oxidisers, powdered aluminium (exothermic), crotonaldehyde, diethyl aluminium bromide (ignition), dioxygenyl tetrafluoroborate (ignition/ ambient temperature), chromium trioxide (ignition), potassium-tert-butoxide (ignition), nitroform (possible explosion), oleum (pressure increased in closed container), cobalt chloride, aluminium triisopropoxide, hydrogen plus palladium dust (ignition), oxygen gas, phosgene, phosgene plus iron salts (possible explosion), sodium dichromate plus sulfuric acid (exothermic/ incandescence), triisobutyl aluminium ▸ reacts with phosphorus trichloride forming hydrogen chloride gas ▸ reacts, possibly violently, with alkaline earth and alkali metals, strong acids, strong caustics, acid anhydrides, halogens,aliphatic amines,

Continued...

	aluminium isopropoxide, isocyanates, acetaldehyde, barium perchlorate (forms highly explosive perchloric ester compound), benzoyl peroxide, chromic acid, dialkylzincs, dichlorine oxide, ethylene oxide (possible explosion), hexamethylene diisocyanate (possible explosion), hydrogen peroxide (forms explosive compound), hypochlorous acid, isopropyl chlorocarbonate, lithium aluminium hydride, lithium tetrahydroaluminate, nitric acid, nitrogen dioxide, nitrogen tetraoxide (possible explosion), pentafluoroguanidine, perchloric acid (especially hot), permonosulfuric acid, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium, trinitromethane ▸ attacks some plastics, rubber and coatings ▸ reacts with metallic aluminium at high temperature ▸ may generate electrostatic charges Alcohols ▸ are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents. ▸ reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen ▸ react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium ▸ should not be heated above 49 deg. C. when in contact with aluminium equipment Secondary alcohols and some branched primary alcohols may produce potentially explosive peroxides after exposure to light and/ or heat.

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
isopropanol	Dermal 888 mg/kg bw/day (Systemic, Chronic) Inhalation 500 mg/m³ (Systemic, Chronic) *Dermal 319 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 89 mg/m³ (Systemic, Chronic) ** *Oral 26 mg/kg bw/day (Systemic, Chronic) **	140.9 mg/L (Water (Fresh)) 140.9 mg/L (Water - Intermittent release) 140.9 mg/L (Water (Marine)) 552 mg/kg sediment dw (Sediment (Fresh Water)) 552 mg/kg sediment dw (Sediment (Marine)) 28 mg/kg soil dw (Soil) 2251 mg/L (STP) 160 mg/kg food (Oral)
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) ** *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) **	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)) 0.052 mg/kg sediment dw (Sediment (Marine)) 0.35 mg/kg soil dw (Soil) 140 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	isopropanol	Propan-2-ol	400 ppm / 999 mg/m3	1250 mg/m3 / 500 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
isopropanol	400 ppm	2000* ppm	12000** ppm
methyl salicylate	2.3 ppm	25 ppm	150 ppm

Ingredient	Original IDLH	Revised IDLH
isopropanol	2,000 ppm	Not Available
methyl salicylate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
methyl salicylate	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
Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)
Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

8.2. Exposure controls

8.2.1. Appropriate engineering	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can

Continued...

controls	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
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:
408C Rubber Renue

Continued...

Material	CPI
NEOPRENE	A
NITRILE	A
NITRILE+PVC	A
PE/EVAL/PE	A
PVC	B
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+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.

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)**	0.86
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	450
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)	83	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	11.7	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	12	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	2	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	0.38	**Gas group**	Not Available
Solubility in water	Partly miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	>2	**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

Continued...

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well. 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. The odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal. Minor but regular methanol exposures may effect the central nervous system, optic nerves and retinae. Symptoms may be delayed, with headache, fatigue, nausea, blurring of vision and double vision. Continued or severe exposures may cause damage to optic nerves, which may become severe with permanent visual impairment even blindness resulting. **WARNING**: Methanol is only slowly eliminated from the body and should be regarded as a cumulative poison which cannot be made non-harmful [*CCINFO*] 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	Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased. Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema. Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in he case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent. 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. Following ingestion, a single exposure to isopropyl alcohol produced lethargy and non-specific effects such as weight loss and irritation. Ingestion of near-lethal doses of isopropanol produces histopathological changes of the stomach, lungs and kidneys, incoordination, lethargy, gastrointestinal tract irritation, and inactivity or anaesthesia. Swallowing 10 ml. of isopropanol may cause serious injury; 100 ml. may be fatal if not promptly treated. The adult single lethal doses is approximately 250 ml. The toxicity of isopropanol is twice that of ethanol and the symptoms of intoxication appear to be similar except for the absence of an initial euphoric effect; gastritis and vomiting are more prominent. Ingestion may cause nausea, vomiting, and diarrhoea. There is evidence that a slight tolerance to isopropanol may be acquired. A dose of 6 ml of methyl salicylate has proved fatal in an adult. Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	The material may accentuate any pre-existing dermatitis condition 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. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.

Continued...

	Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. 511ipa Methyl salicylate sometimes produces systemic poisoning by penetrating intact skin. There are numerous reports of fatal as well as non-fatal salicylate poisonings following topical use for the treatment of various skin disorders. The majority of fatal cases have occurred in children. Systemic effects may include skin and muscle necrosis, interstitial nephritis, hepatic dysfunction and metabolic acidosis. Methyl salicylate can cause a localised as well as generalised urticaria. Contact dermatitis of either an allergic or irritant nature may occur. Severe urticaria and angioedema, following the use of methyl salicylate containing mints, toothpaste or liniments were seen in one individual with a past history of nasal allergy or aspirin (acetyl salicylate) hypersensitivity The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either: ▸ produces severe inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant and severe 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. **NOTE:** Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
Eye	Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage. Eye contact may cause tearing or blurring of vision. Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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. 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). Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain. Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals. There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol. Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects. NOTE: Commercial isopropanol does not contain 'isopropyl oil'. An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct 'isopropyl oil'. Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.

11.2.1. Endocrine Disruption Properties

Not Available

408C Rubber Renue	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
isopropanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 21.026 mg/kg[1]	Eye (rabbit): 10 mg - moderate
	Inhalation(Mouse) LC50; 27.2 mg/l4h[2]	Eye (rabbit): 100 mg - SEVERE
	Oral(Rabbit) LD50; 667 mg/kg[2]	Eye (rabbit): 100mg/24hr-moderate
		Skin (rabbit): 500 mg - mild
methyl salicylate	**TOXICITY**	**IRRITATION**

Continued...

	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]
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*	

ISOPROPANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. 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.
METHYL SALICYLATE	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. 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.

Continued...

	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. 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
408C Rubber Renue & ISOPROPANOL & METHYL SALICYLATE	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.
408C Rubber Renue & 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 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

Continued...

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 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.

408C Rubber Renue & ISOPROPANOL

For isopropanol (IPA):
Acute toxicity: Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers

Continued...

reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat.
Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred.
Repeat dose studies: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified
from these studies were to the kidney.
Reproductive toxicity: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.
Developmental toxicity: The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity
Genotoxicity: All genotoxicity assays reported for isopropanol have been negative
Carcinogenicity: rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans.
Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment

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

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

SECTION 12 Ecological information

12.1. Toxicity

408C Rubber Renue

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

isopropanol

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

methyl salicylate

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

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*

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
For methyl salicylate:
Half-life (hr) air : 138
Half-life (hr) H2O surface water : 3.2-528
BOD 5 55%
Environmental fate:
When released into the soil, methyl salicylate is expected to evaporate quickly or to leach into ground water; remaining material is readily biodegrade.
Methyl salicylate is readily biodegradable in water (half-life between 10 and 30 days)
With a log Kow of less than 3, methyl salicylate has an estimated bioconcentration factor (BCF) of less than 100. and is not expected to significantly bioaccumulate.
When released into the air, methyl salicylate is expected to be readily degraded by reaction with photochemically produced hydroxyl radicals. It is expected to be removed from the atmosphere to a moderate extent by wet deposition. When released into the air, this material is expected to have a half-life between 1 and 10 days.

Continued...

For isopropanol (IPA):
log Kow : -0.16- 0.28
Half-life (hr) air : 33-84
Half-life (hr) H2O surface water : 130
Henry's atm m3 /mol: 8.07E-06
BOD 5: 1.19,60%
COD : 1.61-2.30,97%
ThOD : 2.4
BOD 20: >70% * [Akzo Nobel]
Environmental Fate
Based on calculated results from a lever 1 fugacity model,IPA is expected to partition primarily to the aquatic compartment (77.7%) with the remainder to the air (22.3%). IPA has been shown to biodegrade rapidly in aerobic, aqueous biodegradation tests and therefore, would not be expected to persist in aquatic habitats. IPA is also not expected to persist in surface soils due to rapid evaporation to the air. In the air, physical degradation will occur rapidly due to hydroxy
radical (OH) attack. Overall, IPA presents a low potential hazard to aquatic or terrestrial biota.
IPA is expected to volatilise slowly from water based on a calculated Henry's Law constant of 7.52 x 10 -6 atm.m 3 /mole. The calculated half-life for the volatilisation from surface water (1 meter depth) is predicted to range from 4 days (from a river) to 31 days (from a lake). Hydrolysis is not considered a significant degradation process for IPA. However, aerobic biodegradation of IPA has been shown to occur rapidly under non-acclimated conditions, based on a result of 49% biodegradation from a 5 day BOD test. Additional biodegradation data developed using standardized test methods show that IPA is readily biodegradable in both freshwater and saltwater media (72 to 78% biodegradation in 20 days).
IPA will evaporate quickly from soil due to its high vapor pressure (43 hPa at 20°C), and is not expected to partition to the soil based on a calculated soil adsorption coefficient (log Koc) of 0.03.
IPA has the potential to leach through the soil due to its low soil adsorption
In the air, isopropanol is subject to oxidation predominantly by hydroxy radical attack. The room temperature rate constants determined by several investigators are in good agreement for the reaction of IPA with hydroxy radicals. The atmospheric half-life is expected to be 10 to 25 hours, based on measured degradation rates ranging from 5.1 to 7.1 x 10 -12 cm3 /molecule-sec, and an OH concentration of 1.5 x 106 molecule/cm3 , which is a commonly used default value for calculating atmospheric half-lives. Using OH concentrations representative of polluted (3 x 106) and pristine (3 x 105) air, the atmospheric half-life of IPA would range from 9 to 126 hours, respectively. Direct photolysis is not expected to be an important transformation process for the degradation of IPA.
Ecotoxicity:
IPA has been shown to have a low order of acute aquatic toxicity. Results from 24- to 96-hour LC50 studies range from 1,400 to more than 10,000 mg/L for freshwater and saltwater fish and invertebrates. In addition, 16-hour to 8-day toxicity threshold levels (equivalent to 3% inhibition in cell growth) ranging from 104 to 4,930 mg/L have been demonstrated for various microorganisms.
Chronic aquatic toxicity has also been shown to be of low concern, based on 16- to 21-day NOEC values of 141 to 30 mg/L, respectively, for a freshwater invertebrate.
Bioconcentration of IPA in aquatic organisms is not expected to occur based on a measured log octanol/water partition coefficient (log Kow) of 0.05, a calculated bioconcentration factor of 1 for a freshwater fish, and the unlikelihood of constant, long-term exposures.
Toxicity to Plants
Toxicity of IPA to plants is expected to be low, based on a 7-day toxicity threshold value of 1,800 mg/L for a freshwater algae, and an EC50 value of 2,100 mg/L from a lettuce seed germination test.
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
methyl salicylate	LOW	LOW

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
isopropanol	LOW (LogKOW = 0.05)
methyl salicylate	LOW (LogKOW = 2.55)

12.4. Mobility in soil

Ingredient	Mobility
isopropanol	HIGH (KOC = 1.06)
methyl salicylate	LOW (KOC = 128.2)

12.5.Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Applicable	Not Applicable	Not Applicable
PBT Criteria fulfilled?	Not Applicable	Not Applicable	Not Applicable

12.6. Endocrine Disruption Properties

Not Available

12.7. Other adverse effects

Not Available

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	**DO NOT** allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. 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

Continued...

	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: 408C-125ML, 408C-225ML, 408C-1L

Land transport (ADR-RID)

14.1. **UN number**	1993
14.2. **UN proper shipping name**	FLAMMABLE LIQUID, N.O.S. (vapour pressure at 50 °C more than 110 kPa) (contains isopropanol)
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**	Special provisions: 274 Limited quantity: 1 L

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	1993
14.2. **UN proper shipping name**	Flammable liquid, n.o.s. * (contains isopropanol)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3H
14.4. **Packing group**	II
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: A3 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**	1993
14.2. **UN proper shipping name**	FLAMMABLE LIQUID, N.O.S. (contains isopropanol)
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: 274 Limited Quantities: 1 L

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

Continued...

Product name	Group
isopropanol	Not Available
methyl salicylate	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
isopropanol	Not Available
methyl salicylate	Not Available

SECTION 15 Regulatory information

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

isopropanol is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
Europe EC Inventory
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
UK Workplace Exposure Limits (WELs)

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)

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 (isopropanol; methyl salicylate)
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	01/09/2021
Initial Date	30/03/2021

Full text Risk and Hazard codes

H302	Harmful if swallowed.
H317	May cause an allergic skin reaction.
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.

Continued...

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:
EN 166 Personal eye-protection
EN 340 Protective clothing
EN 374 Protective gloves against chemicals and micro-organisms
EN 13832 Footwear protecting against chemicals
EN 133 Respiratory protective devices

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

Reason For Change

A-1.01 - Added new part number

end of SDS