



408A Rubber Renue

MG Chemicals UK Limited

Version No: A-1.02

Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date: 27/02/2018

Revision Date: 14/09/2020

L.REACH.GBR.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

1.1. Product Identifier

Product name	408A Rubber Renue
Synonyms	SDS Code: 408A-Liquid; 408A-100ML, 408A-125ML, 408A-250ML, 408A-1L UFI: V890-J061-A00J-U0DD
Other means of identification	Not Available

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)	Not Available
Emergency telephone numbers	+(44) 20 35147487	Not Available
Other emergency telephone numbers	+(0) 800 680 0425	Not Available

SECTION 2 HAZARDS IDENTIFICATION

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	H226 - Flammable Liquid Category 3, H315 - Skin Corrosion/Irritation Category 2, H319 - Eye Irritation Category 2, H351 - Carcinogenicity Category 2, H335 - Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), H336 - Specific target organ toxicity - single exposure Category 3 (narcotic effects), H373 - Specific target organ toxicity - repeated exposure Category 2, H304 - Aspiration Hazard Category 1, H412 - Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

2.2. Label elements

Hazard pictogram(s)	
SIGNAL WORD	DANGER

Hazard statement(s)

H226	Flammable liquid and vapour.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H351	Suspected of causing cancer.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H373	May cause damage to organs through prolonged or repeated exposure.

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H304	May be fatal if swallowed and enters airways.
H412	Harmful to aquatic life with long lasting effects.

Supplementary statement(s)

EUH208	Contains methyl salicylate. May produce an allergic reaction.
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Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe fume/mist/vapours/spray.
P271	Use in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P331	Do NOT induce vomiting.
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 and soap.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/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 in accordance with local regulations.
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2.3. Other hazards

Ingestion may produce health damage*.

Cumulative effects may result following exposure*.

Vapours potentially cause drowsiness and dizziness*.

REACH - Art.57-59: The mixture does not contain Substances of Very High Concern (SVHC) at the SDS print date.

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]
1.1330-20-7 2.215-535-7 3.601-022-00-9 4.01-2119488216-32-XXXX	60-68	<u>xylene</u>	Flammable Liquid Category 3, Acute Toxicity (Inhalation) Category 4, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 2; H226, H332, H312, H315 ^[3]
1.100-41-4 2.202-849-4 3.601-023-00-4 4.01-2119489370-35-	17-26	<u>ethylbenzene</u>	Flammable Liquid Category 2, Acute Toxicity (Inhalation) Category 4, Specific target organ toxicity - repeated exposure Category 2 (hearing organs), Aspiration Hazard Category 1; H225, H332, H373, H304 ^[3]

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XXXX registration numbers missing			
1.119-36-8 2.204-317-7 3. Not Available 4.01-2119515671-44-XXXX 01-2120012459-59-XXXX	15	<u>methyl salicylate</u>	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Chronic Aquatic Hazard Category 2; H302, H315, H319, H317, H335, H411 ^[1]
Legend:		1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI 4. Classification drawn from C&L	

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

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

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

for salicylate intoxication:

- ▶ Pending gastric lavage, use emetics such as syrup of Ipecac or delay gastric emptying and absorption by swallowing a slurry of activated charcoal. **Do not give ipecac after charcoal.**
- ▶ Gastric lavage with water or perhaps sodium bicarbonate solution (3%-5%). Mild alkali delays salicylate absorption from the stomach and perhaps slightly from the duodenum.
- ▶ Saline catharsis with sodium or magnesium sulfate (15-30 gm in water).
- ▶ Take an immediate blood sample for an appraisal of the patient's acid-base status. A pH determination on an anaerobic sample of arterial blood is best. An analysis of the plasma salicylate concentration should be made at the same time. Laboratory controls are almost essential for the proper management of severe salicylism.
- ▶ In the presence of an established acidosis, alkali therapy is essential, but at least in an adult, alkali should be withheld until its need is demonstrated by chemical analysis. The intensity of treatment depends on the intensity of acidosis. In the presence of vomiting, intravenous sodium bicarbonate is the most satisfactory of all alkali therapy.
- ▶ Correct dehydration and hypoglycaemia (if present) by the intravenous administration of glucose in water or in isotonic saline. The administration of glucose may also serve to remedy ketosis which is often seen in poisoned children.
- ▶ Even in patients without hypoglycaemia, infusions of glucose adequate to produce distinct hyperglycaemia are recommended to prevent glucose depletion in the brain. This recommendation is based on impressive experimental data in animals.
- ▶ Renal function should be supported by correcting dehydration and incipient shock. Overhydration is not justified. An alkaline urine should be maintained by the administration of alkali if necessary with care to prevent a severe systemic alkalosis. As long as urine remains alkaline (pH above 7.5), administration of an osmotic diuretic such as mannitol or perhaps THAM is useful, but one must be careful to avoid hypokalaemia. Supplements of potassium chloride should be included in parenteral fluids.
- ▶ Small doses of barbiturates, diazepam, paraldehyde, or perhaps other sedatives (but probably not morphine) may be required to suppress extreme restlessness and convulsions.
- ▶ For hyperpyrexia, use sponge baths.

The presence of petechiae or other signs of haemorrhagic tendency calls for a large Vitamin K dose and perhaps ascorbic acid. Minor transfusions may be necessary since bleeding in salicylism is not always due to a prothrombin effect.

- ▶ Haemodialysis and haemoperfusion have proved useful in salicylate poisoning, as have peritoneal dialysis and exchange transfusions, but alkaline diuretic therapy is probably sufficient except in fulminating cases.

[GOSSELIN, et al.: Clinical Toxicology of Commercial Products]

The mechanism of the toxic effect involves metabolic acidosis, respiratory alkalosis, hypoglycaemia, and potassium depletion. Salicylate poisoning is characterised by extreme acid-base disturbances, electrolyte disturbances and decreased levels of consciousness. There are differences between acute and chronic toxicity and a varying clinical picture which is dependent on the age of the patient and their kidney function. The major feature of poisoning is metabolic acidosis due to 'uncoupling of oxidative phosphorylation' which produces an increased metabolic rate, increased oxygen consumption, increased formation of carbon dioxide, increased heat production and increased utilisation of glucose. Direct stimulation of the respiratory centre leads to hyperventilation and respiratory alkalosis. This leads to compensatory increased renal excretion of bicarbonate which contributes to the metabolic acidosis which may coexist or develop subsequently. Hypoglycaemia may occur as a result of increased glucose demand, increased rates of tissue glycolysis, and impaired rate of glucose synthesis. **NOTE:** Tissue glucose levels may be lower than plasma levels. Hyperglycaemia may occur due to increased glycogenolysis. Potassium depletion occurs as a result of increased renal excretion as well as intracellular movement of potassium.

Salicylates competitively inhibit vitamin K dependent synthesis of factors II, VII, IX, X and in addition, may produce a mild dose dependent hepatitis. Salicylates are bound to albumin. The extent of protein binding is concentration dependent (and falls with higher blood levels). This, and the effects of acidosis, decreasing ionisation, means that the volume of distribution increases markedly in overdose as does CNS penetration. The extent of protein binding (50-80%) and the rate of metabolism are concentration dependent. Hepatic clearance has zero order kinetics and thus the therapeutic half-life of 2-4.5 hours but the half-life in overdose is 18-36 hours. Renal excretion is the most important route in overdose. Thus when the salicylate concentrations are in the toxic range there is increased tissue distribution and impaired clearance of the drug.

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HyperTox 3.0 <https://www.ozemail.com.au/~ouad/SALI0001.HTA>

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.

For acute or short term repeated exposures to xylene:

- ▶ Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- ▶ Pulmonary absorption is rapid with about 60-65% retained at rest.
- ▶ Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ < 50 mm Hg or pCO₂ > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

BIOLOGICAL EXPOSURE INDEX - BEI

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

Determinant	Index	Sampling Time	Comments
Methylhippuric acids in urine	1.5 gm/gm creatinine	End of shift	
	2 mg/min	Last 4 hrs of shift	

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

- ▶ 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
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5.3. Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ 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	<ul style="list-style-type: none"> ▶ Liquid and vapour are flammable. ▶ Moderate fire hazard when exposed to heat or flame. ▶ Vapour forms an explosive mixture with air. ▶ Moderate explosion hazard when exposed to heat or flame. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include: carbon monoxide (CO) carbon dioxide (CO₂) other pyrolysis products typical of burning organic material.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

Continued...

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See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none">➤ 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	Chemical Class: aromatic hydrocarbons For release onto land: recommended sorbents listed in order of priority.																																		
	<table><tr><th>SORBENT TYPE</th><th>RANK</th><th>APPLICATION</th><th>COLLECTION</th><th>LIMITATIONS</th></tr></table>					SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS																									
	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS																														
	LAND SPILL - SMALL																																		
	<table><tr><td>Feathers - pillow</td><td>1</td><td>throw</td><td>pitchfork</td><td>DGC, RT</td></tr><tr><td>cross-linked polymer - particulate</td><td>2</td><td>shovel</td><td>shovel</td><td>R,W,SS</td></tr><tr><td>cross-linked polymer- pillow</td><td>2</td><td>throw</td><td>pitchfork</td><td>R, DGC, RT</td></tr><tr><td>sorbent clay - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, I, P,</td></tr><tr><td>treated clay/ treated natural organic - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, I</td></tr><tr><td>wood fibre - pillow</td><td>4</td><td>throw</td><td>pitchfork</td><td>R, P, DGC, RT</td></tr></table>					Feathers - pillow	1	throw	pitchfork	DGC, RT	cross-linked polymer - particulate	2	shovel	shovel	R,W,SS	cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	3	shovel	shovel	R, I, P,	treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I	wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT
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LAND SPILL - MEDIUM																																			
<table><tr><td>cross-linked polymer -particulate</td><td>1</td><td>blower</td><td>skiploader</td><td>R, W, SS</td></tr><tr><td>treated clay/ treated natural organic - particulate</td><td>2</td><td>blower</td><td>skiploader</td><td>R, I</td></tr><tr><td>sorbent clay - particulate</td><td>3</td><td>blower</td><td>skiploader</td><td>R, I, P</td></tr><tr><td>polypropylene - particulate</td><td>3</td><td>blower</td><td>skiploader</td><td>W, SS, DGC</td></tr><tr><td>feathers - pillow</td><td>3</td><td>throw</td><td>skiploader</td><td>DGC, RT</td></tr><tr><td>expanded mineral - particulate</td><td>4</td><td>blower</td><td>skiploader</td><td>R, I, W, P, DGC</td></tr></table>					cross-linked polymer -particulate	1	blower	skiploader	R, W, SS	treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I	sorbent clay - particulate	3	blower	skiploader	R, I, P	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	feathers - pillow	3	throw	skiploader	DGC, RT	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC	
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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																																			
<ul style="list-style-type: none">➤ 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	<ul style="list-style-type: none"> 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. Electrostatic discharge may be generated during pumping - this may result in fire. Ensure electrical continuity by bonding and grounding (earthing) all equipment. Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (≤ 1 m/sec until fill pipe submerged to twice its diameter, then ≤ 7 m/sec).
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	<ul style="list-style-type: none"> ▶ Avoid splash filling. ▶ Do NOT use compressed air for filling discharging or handling operations. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of overexposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid generation of static electricity. ▶ DO NOT use plastic buckets. ▶ Earth all lines and equipment. ▶ Use spark-free tools when handling. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ 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. ▶ DO NOT allow clothing wet with material to stay in contact with skin
Fire and explosion protection	See section 5
Other information	<ul style="list-style-type: none"> ▶ Store in original containers in approved flammable liquid storage area. ▶ Store away from incompatible materials in a cool, dry, well-ventilated area. ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped. ▶ No smoking, naked lights, heat or ignition sources. ▶ Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access. ▶ Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances. ▶ Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems. ▶ Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors. ▶ Keep adsorbents for leaks and spills readily available. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. <p>In addition, for tank storages (where appropriate):</p> <ul style="list-style-type: none"> ▶ Store in grounded, properly designed and approved vessels and away from incompatible materials. ▶ For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up. ▶ Storage tanks should be above ground and diked to hold entire contents.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ 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	<p>Xylenes:</p> <ul style="list-style-type: none"> ▶ may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride ▶ attack some plastics, rubber and coatings ▶ may generate electrostatic charges on flow or agitation due to low conductivity. ▶ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ▶ Aromatics can react exothermically with bases and with diazo compounds. <p>For alkyl aromatics:</p> <p>The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.</p> <ul style="list-style-type: none"> ▶ Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen ▶ Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids. ▶ Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides. ▶ Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily. ▶ Alkali metals accelerate the oxidation while CO₂ as co-oxidant enhances the selectivity. ▶ Microwave conditions give improved yields of the oxidation products. ▶ Photo-oxidation products may occur following reaction with hydroxyl radicals and NO_x - these may be components of photochemical smogs. <p>Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007</p>

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Continued...

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8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	xylene	Xylene, mixed isomers, pure	221 mg/m ³ / 50 ppm	442 mg/m ³ / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	xylene	Xylene, o-,m-,p- or mixed isomers	220 mg/m ³ / 50 ppm	441 mg/m ³ / 100 ppm	Not Available	Sk, BMGV
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	xylene	Xylene (mixed isomers, pure)	221 mg/m ³ / 50 ppm	442 mg/m ³ / 100 ppm	Not Available	Skin
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	ethylbenzene	Ethylbenzene	442 mg/m ³ / 100 ppm	884 mg/m ³ / 200 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	ethylbenzene	Ethylbenzene	441 mg/m ³ / 100 ppm	552 mg/m ³ / 125 ppm	Not Available	Sk
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	ethylbenzene	Ethyl benzene	442 mg/m ³ / 100 ppm	884 mg/m ³ / 200 ppm	Not Available	Skin

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
xylene	Xylenes	Not Available	Not Available	Not Available
ethylbenzene	Ethyl benzene	Not Available	Not Available	Not Available
methyl salicylate	Methyl salicylate	2.3 ppm	25 ppm	150 ppm

Ingredient	Original IDLH	Revised IDLH
xylene	900 ppm	Not Available
ethylbenzene	800 [LEL] ppm	Not Available
methyl salicylate	Not Available	Not Available

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

for xylenes:

IDLH Level: 900 ppm

Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)

NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).

Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.

Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially contribute to the dose received by inhalation.

Odour Safety Factor(OSF)

OSF=4 (XYLENE)

for ethyl benzene:

Odour Threshold Value: 0.46-0.60 ppm

NOTE: Detector tubes for ethylbenzene, measuring in excess of 30 ppm, are commercially available.

Ethyl benzene produces irritation of the skin and mucous membranes and appears to produce acute and chronic effects on the central nervous system. Animal experiments also suggest the effects of chronic exposure include damage to the liver, kidneys and testes. In spite of structural similarities to benzene, the material does not appear to cause damage to the haemopoietic system. The TLV-TWA is thought to be protective against skin and eye irritation. Exposure at this concentration probably will not result in systemic effects.

Subjects exposed at 200 ppm experienced transient irritation of the eyes; at 1000 ppm there was eye irritation with profuse lachrymation; at 2000 ppm eye irritation and lachrymation were immediate and severe accompanied by moderate nasal irritation, constriction in the chest and vertigo; at 5000 ppm exposure produced intolerable irritation of the eyes and throat.

Odour Safety Factor(OSF)

OSF=43 (ETHYL BENZENE)

8.2. Exposure controls

8.2.1. Appropriate engineering controls	<p>CARE: Use of a quantity of this material in confined space or poorly ventilated area, where rapid build up of concentrated atmosphere may occur, could require increased ventilation and/or protective gear</p> <p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p>
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Continued...

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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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Body protection

See Other protection below

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Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower. <ul style="list-style-type: none"> • 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 and 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.
Thermal hazards	Not Available

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:
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Material	CPI
TEFLON	A
VITON	A
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	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.

Respiratory protection

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class - 1	
up to 50	1000	-	A-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	A-2
up to 100	10000	-	A-3
100+		-	Airline**

* - Continuous Flow

** - Continuous-flow or positive pressure demand.

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

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, orange tint		
Physical state	Liquid	Relative density (Water = 1)	0.91
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
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)	137	Molecular weight (g/mol)	Not Available
Flash point (°C)	27	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available

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Upper Explosive Limit (%)	7	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	0.95	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	>2	VOC g/L	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1. Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 TOXICOLOGICAL INFORMATION

11.1. Information on toxicological effects

Inhaled	<p>The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.</p> <p>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.</p> <p>Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.</p> <p>Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p> <p>When humans were exposed to the 100 and 200 ppm for 8 hours about 45-65% is retained in the body. Only traces of unchanged ethyl benzene are excreted in expired air following termination of inhalation exposure.</p> <p>Humans exposed to concentrations of 23-85 ppm excreted most of the retained dose in the urine (mainly as metabolites). Guinea pigs that died from exposure had intense congestion of the lungs and generalised visceral hyperaemia. Rats exposed for three days at 8700 mg/m³ (2000 ppm) showed changes in the levels of dopamine and noradrenaline in various parts of the brain.</p> <p>Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue.</p> <p>Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p>
Ingestion	<p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>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 A dose of 6 ml of methyl salicylate has proved fatal in an adult.</p>

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	<p>Considered an unlikely route of entry in commercial/industrial environments The liquid may produce considerable gastrointestinal discomfort and may be harmful or toxic if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis</p>
Skin Contact	<p>The material may accentuate any pre-existing dermatitis condition</p> <p>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.</p> <p>Toxic effects may result from skin absorption</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p> <p>Repeated application of the undiluted product to the abdominal area of rabbits (10-20 applications over 2-4 weeks) resulted in erythema, oedema and superficial necrosis. The material did not appear to be absorbed through the skin in sufficient quantity to produce outward signs of toxicity.</p> <p>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.</p> <p>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</p> <p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>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.</p>
Eye	<p>Two drops of the ethylbenzene in to the conjunctival sac produced only slight irritation of the conjunctival membrane but no corneal injury.</p> <p>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.</p> <p>The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.</p>
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>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.</p> <p>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).</p> <p>Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms. Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.</p> <p>Xylene has been classed as a developmental toxin in some jurisdictions.</p> <p>Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.</p> <p>Industrial workers exposed to a maximum level of ethylbenzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.</p> <p>Prolonged and repeated exposure may be harmful to the central nervous system (CNS), upper respiratory tract, and/ or may cause liver disorders. It may also cause drying, scaling and blistering of the skin.</p> <p>Rats and mice exposed to ethylbenzene for 6 hours daily, 5 days a week for 104 and 103 weeks respectively showed a statistically significant increase in kidney tumours in male and female rats, lung tumours in male mice, and liver tumours in female mice exposed to 750 ppm ethylbenzene.</p> <p>Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]</p>

408A Rubber Renue	TOXICITY	IRRITATION
	Not Available	Not Available
xylene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 4994.295 mg/l/4h ^[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kg ^[2]	Eye (rabbit): 87 mg mild

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		Skin (rabbit):500 mg/24h moderate
ethylbenzene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye (rabbit): 500 mg - SEVERE
	Inhalation (mouse) LC50: 17.75 mg/l/2H ^[2]	Skin (rabbit): 15 mg/24h mild
	Oral (rat) LD50: 3500 mg/kg ^[2]	
methyl salicylate	TOXICITY	IRRITATION
	dermal (rat) LD50: >=2500 mg/kg ^[1]	Eye (rabbit): 500 mg/24 h - mild
	Oral (rat) LD50: 887 mg/kg ^[2]	Skin (rabbit): 500 mg/24 h - moderate
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	

ETHYLBENZENE	<p>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.</p> <p>Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylglyoxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances.</p> <p>Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys.</p> <p>Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (400 ppm and greater) of ethylbenzene</p> <p>In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncytial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland.</p> <p>In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity</p> <p>Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination.</p> <p>NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p> <p>Liver changes, uterine tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded.</p>
METHYL SALICYLATE	<p>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.</p> <p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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.</p> <p>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 conjugal contact dermatitis occur.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens.</p>

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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 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 prohaptens 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 prohaptens 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 prohaptens or as a prohaptens, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.

Prohaptens

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

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

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species.

Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

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

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

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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 rapidly 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 that 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 acid 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 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 salicylic 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 salicylic 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.

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	<p>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.</p> <p>The Research Institute for Fragrance Materials (RIFM) Expert Panel</p> <p>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.</p> <p>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.</p> <p>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</p>
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ETHYLBENZENE & METHYL SALICYLATE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
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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 available but does not fill the criteria for classification
 ✓ – Data available to make classification
 ☐ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

408A Rubber Renue	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
xylene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	2.6mg/L	2
	EC50	48	Crustacea	>3.4mg/L	2
	EC50	72	Algae or other aquatic plants	4.6mg/L	2
	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
ethylbenzene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.0043mg/L	4
	EC50	48	Crustacea	1.184mg/L	4
	EC50	96	Algae or other aquatic plants	3.6mg/L	4
	NOEC	168	Crustacea	0.96mg/L	5
methyl salicylate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	19.8mg/L	2
	EC50	48	Crustacea	28mg/L	2
	EC50	72	Algae or other aquatic plants	1.1mg/L	2
	NOEC	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			

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the oxygen transfer between the air and the water

Oils of any kind can cause:

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

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

Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (*Palaemonetes pugio*) and brown shrimp (*Penaeus aztecus*) was dimethylnaphthalenes > methylnaphthalenes > naphthalenes.

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are

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more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

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 K_{ow} 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.

For xylenes :

log K_{oc} : 2.05-3.08

K_{oc} : 25.4-204

Half-life (hr) air : 0.24-42

Half-life (hr) H₂O surface water : 24-672

Half-life (hr) H₂O ground : 336-8640

Half-life (hr) soil : 52-672

Henry's Pa m³/mol: 637-879

Henry's atm m³/mol: 7.68E-03

BOD 5 if unstated: 1.4,1%

COD : 2.56,13%

ThOD : 3.125

BCF : 23

log BCF : 1.17-2.41

Environmental Fate

Terrestrial fate: Measured K_{oc} values of 166 and 182, indicate that 3-xylene is expected to have moderate mobility in soil. Volatilisation of p-xylene is expected to be important from moist soil surfaces given a measured Henry's Law constant of 7.18x10⁻³ atm-cu m/mole. The potential for volatilisation of 3-xylene from dry soil surfaces may exist based on a measured vapor pressure of 8.29 mm Hg. p-Xylene may be degraded during its passage through soil). The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. p-Xylene, present in soil samples contaminated with jet fuel, was completely degraded aerobically within 5 days. In aquifer studies under anaerobic conditions, p-xylene was degraded, usually within several weeks, with the production of 3-methylbenzylfumaric acid, 3-methylbenzylsuccinic acid, 3-methylbenzoate, and 3-methylbenzaldehyde as metabolites.

Aquatic fate: K_{oc} values indicate that p-xylene may adsorb to suspended solids and sediment in water. p-Xylene is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. BCF values of 14.8, 23.4, and 6, measured in goldfish, eels, and clams, respectively, indicate that bioconcentration in aquatic organisms is low. p-Xylene in water with added humic substances was 50% degraded following 3 hours irradiation suggesting that indirect photooxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. Although p-xylene is biodegradable and has been observed to degrade in pond water, there are insufficient data to assess the rate of this process in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater in several studies; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high.

Atmospheric fate:

Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere primarily by reaction with photochemically-produced hydroxyl radicals, with an estimated atmospheric lifetime of about 0.5 to 2 days.

Xylenes' susceptibility to photochemical oxidation in the troposphere is to the extent that they may contribute to photochemical smog formation.

According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and from its vapour pressure, p-xylene, is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase p-xylene is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 16 hours. A half-life of 1.0 hr in summer and 10 hr in winter was measured for the reaction of p-xylene with photochemically-produced hydroxyl radicals. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers, with loss rates varying from 9-42% per hr. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzyl nitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

Ecotoxicity:

for xylenes

Fish LC50 (96 h) *Pimephales promelas* 13.4 mg/l; *Oncorhynchus mykiss* 8.05 mg/l; *Lepomis macrochirus* 16.1 mg/l (all flow through values); *Pimephales promelas* 26.7 (static)

Daphnia EC50 948 h): 3.83 mg/l

Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/l

Gammarus lacustris LC50 (48 h): 0.6 mg/l

For ethylbenzene:

log K_{ow}, 3.15

log K_{oc} : 1.98-3.04

K_{oc} : 164

log K_{om} : 1.73-3.23

Vapour Pressure, 1270 Pa (1.27 kPa)

Half-life (hr) air : 0.24-85.6

Half-life (hr) H₂O surface water : 5-240

Half-life (hr) H₂O ground : 144-5472

Half-life (hr) soil : 72-240

Henry's Pa m³/mol: 748-887

Henry's atm m³/mol: 8.44E-03

ThOD : 3.17

BCF : 3.15-146

log BCF : 1.19-2.67

Water solubility, 169 mg/l at 25 C

Environmental fate:

Ethylbenzene partitions to air from water and soil, and is degraded in air. Ethylbenzene is volatile and when released will quickly vaporize. Photodegradation is the primary route of removal in the environment. Photodegradation is estimated with a half-life of 1 day. Ethylbenzene is considered inherently biodegradable and removal from water occurs primarily by evaporation but in the summer biodegradation plays a key role in the removal process. Level I and Level III fugacity modeling indicate that partitioning is primarily to the air compartment, 98 and 96%, respectively. Ethylbenzene is inherently biodegradable in water and in soil under aerobic conditions, and not rapidly biodegradable in anaerobic conditions. Ethylbenzene is expected to be moderately adsorbed to soil.

Based on measured data, ethylbenzene is not expected to bioaccumulate (BCF 1.1-15).

Ecotoxicity:

In acute aquatic toxicity testing LC50 values range approximately between 1 and 10 mg/l. In acute aquatic fish tests (fresh water species), the 96-hr LC50 for *Pimephales promelas* and *Oncorhynchus mykiss* are 12.1 and 4.2 mg/L, respectively. Data are available in the saltwater species *Menidia menidia* and give results within the same range as for the fresh water species with a 96-hr LC50 = 5.1 mg/L. In fresh water invertebrate species *Daphnia magna* and *Ceriodaphnia dubia*, 48-hr LC50 values were 1.81 and 3.2 mg/L, respectively. Additional data is available in the saltwater species *Crangon franciscorum* (96-hr LC50 = 0.49 mg/L) and *Mysidopsis bahia* (96-hr LC50 = 2.6 mg/L). In 96-hr algal toxicity testing, results indicate that ethylbenzene inhibits algae growth in *Selenastrum capricornatum* at 3.6 mg/L and in *Skeletonema costatum* at 7.7 mg/L.

DO NOT discharge into sewer or waterways.

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12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
methyl salicylate	LOW	LOW

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
xylene	MEDIUM (BCF = 740)
ethylbenzene	LOW (BCF = 79.43)
methyl salicylate	LOW (LogKOW = 2.55)

12.4. Mobility in soil

Ingredient	Mobility
ethylbenzene	LOW (KOC = 517.8)
methyl salicylate	LOW (KOC = 128.2)

12.5. Results of PBT and vPvB assessment

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

12.6. Other adverse effects

No data available


SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none">▶ Containers may still present a chemical hazard/ danger when empty.▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none">▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none">▶ Reduction▶ Reuse▶ Recycling▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none">▶ DO NOT allow wash water from cleaning or process equipment to enter drains.▶ It may be necessary to collect all wash water for treatment before disposal.▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.▶ Where in doubt contact the responsible authority.▶ Recycle wherever possible.▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material).▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
	Waste treatment options
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

	 <p>Limited Quantity: 408A-100ML, 408A-125ML, 408A-250ML, 408A-1L</p>
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Land transport (ADR)

14.1.UN number	1307
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14.2. UN proper shipping name	XYLENES	
14.3. Transport hazard class(es)	Class	3
	Subrisk	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Hazard identification (Kemler)	30
	Classification code	F1
	Hazard Label	3
	Special provisions	Not Applicable
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

14.1. UN number	1307	
14.2. UN proper shipping name	Xylenes	
14.3. Transport hazard class(es)	ICAO/IATA Class	3
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	3L
14.4. Packing group	III	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	A3
	Cargo Only Packing Instructions	366
	Cargo Only Maximum Qty / Pack	220 L
	Passenger and Cargo Packing Instructions	355
	Passenger and Cargo Maximum Qty / Pack	60 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y344
	Passenger and Cargo Limited Maximum Qty / Pack	10 L

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1307	
14.2. UN proper shipping name	XYLENES	
14.3. Transport hazard class(es)	IMDG Class	3
	IMDG Subrisk	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	F-E , S-D
	Special provisions	223
	Limited Quantities	5 L

Inland waterways transport (ADN)

14.1. UN number	1307	
14.2. UN proper shipping name	XYLENES	
14.3. Transport hazard class(es)	3	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Classification code	F1
	Special provisions	Not Applicable
	Limited quantity	5 L
	Equipment required	PP, EX, A
	Fire cones number	0

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 European Customs Inventory of Chemical Substances ECICS (English)
 European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
 European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Bulgarian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Czech)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Danish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Dutch)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Estonian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Finnish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Maltese)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Polish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Portuguese)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Romanian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovak)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovenian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Spanish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Swedish)
 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)

ETHYLBENZENE(100-41-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 European Customs Inventory of Chemical Substances ECICS (English)
 European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
 European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Bulgarian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Czech)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Danish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Dutch)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Estonian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Finnish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Maltese)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Polish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Portuguese)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Romanian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovak)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovenian)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Spanish)
 European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Swedish)
 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(119-36-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 European Customs Inventory of Chemical Substances ECICS (English)

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

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments

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15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (methyl salicylate; xylene; ethylbenzene)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (methyl salicylate)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Full text Risk and Hazard codes

H225	Highly flammable liquid and vapour.
H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H317	May cause an allergic skin reaction.
H332	Harmful if inhaled.
H411	Toxic to aquatic life with long lasting effects.

Other information

Ingredients with multiple cas numbers

Name	CAS No
methyl salicylate	119-36-8, 8024-54-2, 9041-28-5

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

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

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

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average

PC—STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit.

IDLH: Immediately Dangerous to Life or Health Concentrations

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

Reason For Change

A-1.02 - Changes to precautionary statements in section 2