8349TFM-B Thermal Adhesive
MG Chemicals UK Limited

Version No: **A-1.00**
Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date: **25/09/2020**
Reision Date: **28/09/2020**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

1.1. Product Identifier

Product name	8349TFM-B
Synonyms	SDS Code: 8349TFM-Part B; 8349TFM-B, 8349TFM-25ML, 8349TFM-50ML
Other means of identification	Thermal Adhesive

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

Relevant identified uses	Thermally conductive adhesive hardener
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

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

1.4. Emergency telephone number

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

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	H318 - Serious Eye Damage Category 1, H315 - Skin Corrosion/Irritation Category 2, H317 - Skin Sensitizer Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

2.2. Label elements

Hazard pictogram(s)	
UFI:	3GQ0-G0G5-G00R-QK4A
Signal word	**Danger**

Hazard statement(s)

H318	Causes serious eye damage.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

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P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing mist/vapours/spray.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P321	Specific treatment (see advice on this label).
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

2.3. Other hazards

Ingestion may produce health damage*.

Cumulative effects may result following exposure*.

Eye contact may produce serious damage*.

Possible respiratory sensitizer*.

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]**
1.21645-51-2 2.244-492-7 3.Not Available 4.01-2119529246-39-XXXX	53	aluminium hydroxide	Eye Irritation Category 2; H319, EUH066 [1]
1.1344-28-1. 2.215-691-6 3.Not Available 4.01-2119529248-35-XXXX	15	aluminium oxide	Not Applicable
1.100-51-6 2.202-859-9 3.603-057-00-5 4.01-2119492630-38-XXXX|01-2120762094-56-XXXX	3	benzyl alcohol	Acute Toxicity (Oral) Category 4, Acute Toxicity (Inhalation) Category 4; H302, H332 [2]
1.135108-88-2 2.Not Available 3.Not Available 4.01-2119983522-33-XXXX	3	formaldehyde/ benzenamine, hydrogenated	Metal Corrosion Category 1, Skin Corrosion/Irritation Category 1B, Serious Eye Damage Category 1, Acute Toxicity (Oral) Category 4; H290, H314, H318, H302 [1]
1.109-55-7 2.203-680-9 3.612-061-00-6 4.01-2119486842-27-XXXX	2	3-dimethylaminopropylamine	Skin Corrosion/Irritation Category 1B, Acute Toxicity (Oral) Category 4, Flammable Liquid Category 3, Skin Sensitizer Category 1; H314, H302, H226, H317 [2]
1.70700-21-9 2.Not Available 3.Not Available 4.Not Available	1	monomethyl phosphate ethoxylated	Skin Corrosion/Irritation Category 2, Chronic Aquatic Hazard Category 4, Serious Eye Damage Category 1; H315, H413, H318 [1]
1.1333-86-4 2.215-609-9|422-130-0 3.Not Available 4.01-2119384822-32-XXXX|01-2120767622-50-XXXX|01-0000016864-62-XXXX	1	carbon black	Carcinogenicity Category 2; H351 [1]
1.1761-71-3 2.217-168-8 3.Not Available 4.01-2119541673-38-XXXX	0.2	4,4'-methylenebis(cyclohexylamine)	Metal Corrosion Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1A, Chronic Aquatic Hazard Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - repeated exposure Category 2, Serious Eye Damage Category 1; H290, H302, H314, H411, H317, H373, H318 [1]

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1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.108-95-2 2.203-632-7 3.604-001-00-2 4.01-2119471329-32-XXXX|01-2120762102-67-XXXX	0.2	phenol *	Germ cell mutagenicity Category 2, Acute Toxicity (Dermal) Category 3, Acute Toxicity (Oral) Category 3, Acute Toxicity (Inhalation) Category 3, Skin Corrosion/Irritation Category 1B, Specific target organ toxicity - repeated exposure Category 2; H341, H311, H301, H331, H314, H373 [2]
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available*		

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with 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. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ 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.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

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

Treat symptomatically.

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

5.3. Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ 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	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) metal oxides other pyrolysis products typical of burning organic material.

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	When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles. May emit poisonous fumes. May emit corrosive fumes. Aluminium hydroxide is a flame retardant. At around 200 C, aluminium hydroxide (aluminium trihydrate) is decomposed to aluminium oxide (which forms a protective, non-flammable layer on the material surface) and water. The water (as steam) forms a layer of non-flammable gas near the material's surface, inhibiting flames. The reaction is endothermic (absorbs heat energy), thus cooling the material and slowing burning .

SECTION 6 Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ 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	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ 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	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.

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Storage incompatibility	For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate. Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt. ‣ Avoid reaction with oxidising agents

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
aluminium hydroxide	Inhalation 10.76 mg/m³ (Systemic, Chronic) Inhalation 10.76 mg/m³ (Local, Chronic) *Oral 4.74 mg/kg bw/day (Systemic, Chronic)* *	Not Available
aluminium oxide	Dermal 0.84 mg/kg bw/day (Systemic, Chronic) Inhalation 3 mg/m³ (Systemic, Chronic) Inhalation 3 mg/m³ (Local, Chronic) *Dermal 0.3 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 0.75 mg/m³ (Systemic, Chronic)* * *Oral 1.32 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 0.75 mg/m³ (Local, Chronic)* *	74.9 µg/L (Water (Fresh)) 20 mg/L (STP)
benzyl alcohol	Dermal 8 mg/kg bw/day (Systemic, Chronic) Inhalation 22 mg/m³ (Systemic, Chronic) Dermal 40 mg/kg bw/day (Systemic, Acute) Inhalation 110 mg/m³ (Systemic, Acute) *Dermal 4 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 5.4 mg/m³ (Systemic, Chronic)* * *Oral 4 mg/kg bw/day (Systemic, Chronic)* * *Dermal 20 mg/kg bw/day (Systemic, Acute)* * *Inhalation 27 mg/m³ (Systemic, Acute)* * *Oral 20 mg/kg bw/day (Systemic, Acute)* *	1 mg/L (Water (Fresh)) 0.1 mg/L (Water - Intermittent release) 2.3 mg/L (Water (Marine)) 5.27 mg/kg sediment dw (Sediment (Fresh Water)) 0.527 mg/kg sediment dw (Sediment (Marine)) 0.456 mg/kg soil dw (Soil) 39 mg/L (STP)
formaldehyde/ benzenamine, hydrogenated	Dermal 2 mg/kg bw/day (Systemic, Chronic) Inhalation 0.2 mg/m³ (Systemic, Chronic) Dermal 6 mg/kg bw/day (Systemic, Acute) Inhalation 2 mg/m³ (Systemic, Acute)	0.015 mg/L (Water (Fresh)) 0.002 mg/L (Water - Intermittent release) 0.15 mg/L (Water (Marine)) 15 mg/kg sediment dw (Sediment (Fresh Water)) 1.5 mg/kg sediment dw (Sediment (Marine)) 1.8 mg/kg soil dw (Soil) 1.9 mg/L (STP)
3-dimethylaminopropylamine	Inhalation 1.2 mg/m³ (Systemic, Chronic)	0.073 mg/L (Water (Fresh)) 0.007 mg/L (Water - Intermittent release) 0.34 mg/L (Water (Marine)) 0.735 mg/kg sediment dw (Sediment (Fresh Water)) 0.073 mg/kg sediment dw (Sediment (Marine)) 0.104 mg/kg soil dw (Soil) 10 mg/L (STP)
carbon black	Inhalation 1 mg/m³ (Systemic, Chronic) Inhalation 0.5 mg/m³ (Local, Chronic) *Inhalation 0.06 mg/m³ (Systemic, Chronic)* *	1 mg/L (Water (Fresh)) 0.1 mg/L (Water - Intermittent release) 10 mg/L (Water (Marine))
4,4'-methylenebis(cyclohexylamine)	Dermal 0.1 mg/kg bw/day (Systemic, Chronic) Inhalation 0.9 mg/m³ (Systemic, Chronic) *Dermal 0.06 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 0.21 mg/m³ (Systemic, Chronic)* * *Oral 0.06 mg/kg bw/day (Systemic, Chronic)* *	0.08 mg/L (Water (Fresh)) 0.008 mg/L (Water - Intermittent release) 0.08 mg/L (Water (Marine)) 14.6 mg/kg sediment dw (Sediment (Fresh Water)) 1.46 mg/kg sediment dw (Sediment (Marine)) 4.56 mg/kg soil dw (Soil) 3.2 mg/L (STP) 0.556 mg/kg food (Oral)
phenol	Dermal 1.23 mg/kg bw/day (Systemic, Chronic) Inhalation 8 mg/m³ (Systemic, Chronic) Inhalation 16 mg/m³ (Local, Acute) *Dermal 0.4 mg/kg bw/day (Systemic, Chronic)* * *Inhalation 1.32 mg/m³ (Systemic, Chronic)* * *Oral 0.4 mg/kg bw/day (Systemic, Chronic)* *	0.008 mg/L (Water (Fresh)) 0.001 mg/L (Water - Intermittent release) 0.031 mg/L (Water (Marine)) 0.091 mg/kg sediment dw (Sediment (Fresh Water)) 0.009 mg/kg sediment dw (Sediment (Marine)) 0.136 mg/kg soil dw (Soil) 2.1 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: inhalable dust	10 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	aluminium oxide	Aluminium oxides: respirable dust	4 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m3	7 mg/m3	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	phenol	Phenol	2 ppm / 7.8 mg/m3	16 mg/m3 / 4 ppm	Not Available	Sk
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	phenol	Phenol	2 ppm / 8 mg/m3	16 mg/m3 / 4 ppm	Not Available	skin

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
aluminium hydroxide	Aluminum hydroxide	8.7 mg/m3	73 mg/m3	440 mg/m3
aluminium oxide	Aluminum oxide; (Alumina)	15 mg/m3	170 mg/m3	990 mg/m3
benzyl alcohol	Benzyl alcohol	30 ppm	52 ppm	740 ppm
3-dimethylaminopropylamine	Dimethyl-1,3-propanediamine, N,N-; (1-Amino-3-dimethylaminopropane)	1.2 ppm	13 ppm	89 ppm
carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3
phenol	Phenol	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
aluminium hydroxide	Not Available	Not Available
aluminium oxide	Not Available	Not Available
benzyl alcohol	Not Available	Not Available
formaldehyde/ benzenamine, hydrogenated	Not Available	Not Available
3-dimethylaminopropylamine	Not Available	Not Available
monomethyl phosphate ethoxylated	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
4,4'-methylenebis(cyclohexylamine)	Not Available	Not Available
phenol	250 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
aluminium hydroxide	E	≤ 0.01 mg/m³
benzyl alcohol	E	≤ 0.1 ppm
formaldehyde/ benzenamine, hydrogenated	E	≤ 0.1 ppm
3-dimethylaminopropylamine	E	≤ 0.1 ppm
monomethyl phosphate ethoxylated	E	≤ 0.1 ppm
4,4'-methylenebis(cyclohexylamine)	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Fragrance substance with is an established contact allergen in humans.
Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012
For aluminium oxide and pyrophoric grades of aluminium:
Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA.
The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For aluminium oxide:
The experimental and clinical data indicate that aluminium oxide acts as an 'inert' material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.
[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition
Odour Threshold Value for phenol: 0.060 ppm (detection)
NOTE: Detector tubes for phenol, measuring in excess of 1 ppm, are commercially available.
Systemic absorption by all routes may induce convulsions with damage to the lungs and central nervous system.
Exposure at or below the recommended TLV-TWA is thought to protect the worker from respiratory, cardiovascular, hepatic, renal and neurological toxicity. Workers or volunteers exposed at or below 5.2 ppm phenol have experienced no ill-effects. Because phenol as a vapour, liquid or solid can penetrate the skin causing systemic effects, a skin notation is considered necessary. Although ACGIH has not recommended a STEL it is felt that ACGIH excursion limits (15 ppm limited to a total duration of 30
minutes with brief excursions limited to no more than 25 ppm) and NIOSH Ceiling values are sufficiently similar so as to provide the same margin of safety.
Odour Safety Factor(OSF)
OSF=25 (PHENOL)

8.2. Exposure controls

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

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

General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

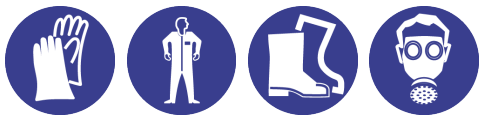
Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low 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)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 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

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

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	· Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

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:
8349TFM-B Adhesive—Thermally Conductive, Flame Retardant

Material	CPI
BUTYL	A
BUTYL/NEOPRENE	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
PVC	C
TEFLON	C
VITON	C
VITON/NEOPRENE	C

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

Respiratory protection

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

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

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

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

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

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	Black

Physical state	Liquid	**Relative density (Water = 1)**	1.74
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available

Continued...

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)	203	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	96	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**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	▸ 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	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.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Acute toxic responses to aluminium are confined to the more soluble forms.
Skin Contact	The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives . Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production. Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure. When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is

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doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.
Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.
There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.
Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.
Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.
After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.
At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.
Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer’s disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.
Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.
Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of 'tau' a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).
Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [*Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995*]
There are reports of lung damage due to excessive inhalation of alumina dust. Ingestion of large amounts of aluminium hydroxide for prolonged periods may cause phosphate depletion, especially if phosphate intake is low. This may cause loss of appetite, muscle weakness, muscular disease and even softening of the bones. These effects have not been reported in people occupationally exposed to aluminium hydroxide.

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TOXICITY	IRRITATION
Not Available	Not Available

aluminium hydroxide

TOXICITY	IRRITATION
Not Available	Eye: no adverse effect observed (not irritating)[1]
	Skin: no adverse effect observed (not irritating)[1]

aluminium oxide

TOXICITY	IRRITATION
Oral (rat) LD50: >5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Skin: no adverse effect observed (not irritating)[1]

Continued...

benzyl alcohol

TOXICITY	IRRITATION
~105 mg/kg[2]	Eye (rabbit): 0.75 mg open SEVERE
~2080 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
~60 mg/kg[2]	Skin (man): 16 mg/48h-mild
>=25<=400 mg/kg[2]	Skin (rabbit):10 mg/24h open-mild
>=25-400 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
>=500<=800 mg/kg[2]	
>400800 mg/kg[2]	
2000 mg/kg[2]	
324 mg/kg[2]	
480 mg/kg[2]	
950 mg/kg[2]	
Inhalation (rat) LC50: >4.178 mg/l/4h[2]	
Oral (rat) LD50: =2080 mg/kg[2]	
Oral (rat) LD50: 1230 mg/kg[2]	

formaldehyde/ benzenamine, hydrogenated

TOXICITY	IRRITATION
Not Available	Skin: adverse effect observed (corrosive)[1]

3-dimethylaminopropylamine

TOXICITY	IRRITATION
Oral (rat) LD50: ~1525 mg/kg[2]	Eye (rabbit): 5 mg - moderate
Oral (rat) LD50: ~922 mg/kg[2]	Eye: adverse effect observed (irreversible damage)[1]
Oral (rat) LD50: 1870 mg/kg[2]	Skin (rabbit): 0.1 mg/24h - open
	Skin: adverse effect observed (corrosive)[1]
	Skin: adverse effect observed (irritating)[1]

monomethyl phosphate ethoxylated

TOXICITY	IRRITATION
Not Available	Not Available

carbon black

TOXICITY	IRRITATION
4 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
7 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
Oral (rat) LD50: >15400 mg/kg[2]	

4,4'-methylenebis(cyclohexylamine)

TOXICITY	IRRITATION
100-1250 mg/kg[2]	Eye (rabbit): 10uL./24h SEVERE
Inhalation (mouse) LC50: 0.4 mg/l/4H[2]	Eye: adverse effect observed (irreversible damage)[1]
Oral (rat) LD50: 380 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit): SEVERE Corrosive **
	Skin: adverse effect observed (corrosive)[1]

phenol

TOXICITY	IRRITATION
=500 mg/kg[2]	Eye(rabbit): 100 mg rinse - mild
=80 mg/kg[2]	Eye(rabbit): 5 mg - SEVERE
Dermal (rabbit) LD50: 850 mg/kg[2]	Skin(rabbit): 500 mg open -SEVERE
Inhalation (rat) LC50: 0.316 mg/l/4H[2]	Skin(rabbit): 500 mg/24hr - SEVERE
Oral (mouse) LD50: =282 mg/kg[2]	
Oral (mouse) LD50: =300 mg/kg[2]	
Oral (rat) LD50: =414 mg/kg[2]	
Oral (rat) LD50: 317 mg/kg[2]	
Oral (rat) LD50: 410-530 mg/kg[2]	

Legend: *1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise*

Continued...

	specified data extracted from RTECS - Register of Toxic Effect of chemical Substances
8349TFM-B Adhesive—Thermally Conductive, Flame Retardant	For aluminium compounds: Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate). Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability. For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe. Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium. The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account Systemic toxicity after repeated exposure No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium. When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose. The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent Reproductive and developmental toxicity: Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI. Genotoxicity Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels. Carcinogenicity. The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons. Neurodegenerative diseases. Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to

Continued...

	determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases.Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment. Contact sensitivity: It has been suggested that the body burden of aluminium may be linked to different iseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome. Aluminium acts not only as an adjuvant,stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitisers causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response.Once inside the skin, the metal ions must bind to proteins to become immunologically reactive.The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminium Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste
BENZYL ALCOHOL	For benzyl alkyl alcohols: Unlike benzylic alcohols, the beta-hydroxyl group of the members of this cluster is unlikely to undergo phase II metabolic activation. Instead, the beta-hydroxyl group is expected to contribute to detoxification via oxidation to hydrophilic acid. Despite structural similarity to carcinogenic ethyl benzene, only a marginal concern has been assigned to phenethyl alcohol due to limited mechanistic analogy. For benzoates: **Acute toxicity:** Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol. The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds. Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye. **Sensitisation:** The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers. **Repeat dose toxicity:** For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed. For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur. **Mutagenicity:** All chemicals showed no mutagenic activity in *in vitro* Ames tests. Various results were obtained with other *in vitro* genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity *in vivo.* While some mixed and/or equivocal *in vitro* chromosomal/chromatid responses have been observed, no genotoxicity was observed in the *in vivo* cytogenetic, micronucleus, or other assays. The weight of the evidence of the *in vitro* and *in vivo* genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies. In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts. **Developmental toxicity:** In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed. A member or analogue of a group of 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 substances in this group: · contain a benzene ring substituted with a reactive primary oxygenated functional group or can be hydrolysed to such a functional group · the major pathway of metabolic detoxification involves hydrolysis and oxidation to yield the corresponding benzoic acid derivate which is excreted either as the free acid or the glycine conjugate · they show a consistent pattern of toxicity in both short- and long- term studies and · they exhibit no evidence of genotoxicity in standardised batteries of in vitro and in vivo assays. The benzyl derivatives are rapidly absorbed through the gut, metabolised primarily in the liver, and excreted in the urine as glycine conjugates of benzoic acid derivatives. In general, aromatic esters are hydrolysed in vivo through the catalytic activity of carboxylesterases, the most important of which are the A-esterases. Hydrolysis of benzyl and benzoate esters to yield corresponding alcohols and carboxylic acids and hydrolysis of

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	acetals to yield benzaldehyde and simple alcohols have been reported in several experiments. The alcohols and aldehydes are rapidly oxidised to benzoic acid while benzoate esters are hydrolysed to benzoic acid. Flavor and Extract Manufacturers Association (FEMA) The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles. The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity. At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin. The potential for eye irritation is minimal. With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low. NOAELs for maternal and developmental toxicity are far in excess of current human exposure levels. No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity in vitro bacterial assays, and in vitro mammalian cell assays. All in vivo micronucleus assays were negative. It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients The Research Institute for Fragrance Materials (RIFM) Expert Panel
FORMALDEHYDE/ BENZENAMINE, HYDROGENATED	Amine adducts have much reduced volatility and are less irritating to the skin and eyes than amine hardeners. However commercial amine adducts may contain a percentage of unreacted amine and all unnecessary contact should be avoided. Amine adducts are prepared by reacting excess primary amines with epoxy resin.
3-DIMETHYLAMINOPROPYLAMINE	for 3-dimethylaminopropylamine (syn 3-aminopropyldimethylamine, DMPA) **Acute toxicity:** DMPA was been found to be harmful following oral administration to rats. In a field study workers showed impaired respiration (wheezy breath, constricted chest, irritation of mucosa of the eyes, nose and pharynx) as a result of occupational exposure to DMPA (2.34 – 5.87 mg/m3= 0.55 – 1.38 ppm). Based on the results of the sensitisation test on the skin DMPA has been classified as having a sensitising effect. DMPA showed strong irritating or corrosive effects. **Repeat dose toxicity**: In a oral 28-day subchronic toxicity study with rats, the no-observed-adverse effect-level (NOAEL) was 50 mg /kg bw/day. In the oral reproduction/developmental toxicity screening test the no-observed-adverse effect-level (NOAEL) was 200 mg/kg bw/day. **Genotoxicity**: DMPA was not mutagenic in the Ames Test and in a mouse micronucleus assay.
MONOMETHYL PHOSPHATE ETHOXYLATED	for alkyl alcohol alkoxylate phosphate (AAAPD) surfactants (alkyl or alcohol ether phosphates): **Acute toxicity:** This group of surfactants exhibits similar effects to the alcohol ether sulfates (AAASDs) (typically sodium lauryl ether sulfate - SLES - CAS RN 68891-38-3). They are likely to be skin/ eye irritants (R36/38) in their undiluted forms but not acutely toxic. The reported oral LD50 values were higher than 1600 mg/kg for the alkyl ether phosphates family described by CAS RN: 9046-01-9. No effects were found at any concentration tested dermally. Commercial products may contain excess phosphoric acid and may produce serious eye irritation (R41) or may even be classified as corrosive, acidic substances. **Subchronic toxicity:** Data for sulfate derivatives has been identified in the public domain**.** Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2(CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction. SLES was evaluated for effects on the reproduction and prenatal/postnatal development of the rat when administered orally via the drinking water through two successive generations. Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives **Genotoxicity:** Alcohol ether phosphates are unlikely to be genotoxic by analogy with their alcohol ether sulfate equivalents. **Carcinogenicity:** Chronic dietary studies conducted with rats on sulfate derivatives showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).] **Reproductive and developmental toxicity:** Studies with sulfate derivatives showed little to no toxicity in dams or pups with the NOEL in a developmental toxicity study in rats with SLES at the limit dose of 1000 mg/kg/day and a reproductive NOAEL of 0.3% in drinking water (equivalent to 300 mg/kg/day), the highest dose tested in a two-generation reproduction study. In studies with phosphate derivatives, the reproductive/ developmental NOAEL for an OECD 422 study with CAS 681340-47-2 was 800 mg/kg/day, the highest dose tested, and for CAS RN 78330-24-2 the NOEL was 200 mg/kg/day. An NOAEL of 200 mg/kg/day was selected as the toxicological endpoint for he chronic risk assessment for phosphate derivatives by the US EPA. Both alcohol ether sulfates and phosphates have been evaluated in acute, subchronic, developmental and reproductive studies capable of detecting effects on endocrine mediated events. The results of these studies did not give any indication of a treatment-related effect on the oestrogen receptor or endocrine system**.** **Metabolic fate:** For compounds of comparable C16 carbon chain, the metabolites of the lower molecular weight ethoxylated (POE n=3) alcohol ether sulfate surfactants are readily absorbed and excreted primarily in the urine whereas the C16 surfactants with increased ethoxylation (POE n=9) are poorly absorbed and excreted primarily in the faeces There was also no evidence of hydrolysis of the sulfate group from C16 POE n= 3 and C16 POE n=9 or of metabolism of the ethoxylate portion of the molecule. With C11 POE n=3 and C12 POE n=3 metabolic studies in rats confirmed that the alkyl chain is extensively metabolised by beta- or omega oxidation leaving the ethoxysulfate, which is excreted directly. By analogy alcohol ether phosphate esters may initially undergo metabolism to generate the corresponding alkyl alcohol alkoxylate and POE (or POE/POP - polyoxypropylene) phosphate glycol; the dephosphoralyted metabolite should be hydrolysed to the POE (or POE/POP) polyalkoxylate glycols and linear branched saturated and unsaturated alkyl alcohol metabolites. The resultant alkyl alcohol metabolites would be oxidised in fatty acid oxidation pathways. The polyalkoxylate glycols may either be conjugated and excreted unchanged or hydrolysed/ oxidised to various degraded metabolites before bring conjugated and excreted **Sensitising potential:** Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their

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	susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69 Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners. PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology http://doi.org/10.5487/TR.2015.31.2.105
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
4,4'-METHYLENEBIS(CYCLOHEXYLAMINE)	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence). The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.
PHENOL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
8349TFM-B Adhesive—Thermally Conductive, Flame Retardant & BENZYL ALCOHOL & 3-DIMETHYLAMINOPROPYLAMINE & 4,4'-METHYLENEBIS(CYCLOHEXYLAMINE)	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
8349TFM-B Adhesive—Thermally Conductive, Flame Retardant & BENZYL ALCOHOL	Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to 'perfume mix'. The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits. Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly

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exposed to the fragrance products, being four brands of cologne and one brand of toilet water.
Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.
Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.
Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.
Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.
Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic.
Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.
Pigmentary anomalies: The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.
Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.
General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.
Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or as a prohapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an 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

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	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
ALUMINIUM HYDROXIDE & ALUMINIUM OXIDE & FORMALDEHYDE/ BENZENAMINE, HYDROGENATED & CARBON BLACK	No significant acute toxicological data identified in literature search.
BENZYL ALCOHOL & 4,4'-METHYLENEBIS(CYCLOHEXYLAMINE)	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
FORMALDEHYDE/ BENZENAMINE, HYDROGENATED & 3-DIMETHYLAMINOPROPYLAMINE & 4,4'-METHYLENEBIS(CYCLOHEXYLAMINE) & PHENOL	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.
3-DIMETHYLAMINOPROPYLAMINE & 4,4'-METHYLENEBIS(CYCLOHEXYLAMINE)	While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects. ▸ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis. ▸ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient. Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion. **Inhalation:** Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies. While most polyurethane amine catalysts are not sensitisers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease. Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema. **Skin Contact:** Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis. Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient. **Eye Contact:** Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. Direct contact with the liquid amine may cause severe irritation and tissue injury, and the “burning” may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling. The corneal swelling may manifest itself in visual disturbances such as blurred or “foggy” vision with a blue tint (“blue haze”) and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation. **Ingestion:** The oral toxicity of amine catalysts varies from moderately to very toxic. Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus,and gastrointestinal tract. Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs. Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death. **Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000 Alliance for Polyurethanes Industry**

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

Continued...

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

SECTION 12 Ecological information

12.1. Toxicity

8349TFM-B Adhesive—Thermally Conductive, Flame Retardant

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

aluminium hydroxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	0.001-0.134mg/L	2
EC50	48	Crustacea	0.7364mg/L	2
EC50	72	Algae or other aquatic plants	0.001-0.05mg/L	2
NOEC	240	Crustacea	0.001-0.1002mg/L	2

aluminium oxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	0.001-0.134mg/L	2
EC50	48	Crustacea	0.7364mg/L	2
EC50	72	Algae or other aquatic plants	0.001-0.799mg/L	2
NOEC	240	Crustacea	0.001-0.1002mg/L	2

benzyl alcohol

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	10mg/L	2
EC50	48	Crustacea	230mg/L	2
EC50	96	Algae or other aquatic plants	76.828mg/L	2
NOEC	336	Fish	5.1mg/L	2

formaldehyde/ benzenamine, hydrogenated

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	63mg/L	2
EC50	48	Crustacea	15.4mg/L	2
EC50	72	Algae or other aquatic plants	43.94mg/L	2
EC10	72	Algae or other aquatic plants	1.2mg/L	2
NOEC	96	Fish	40mg/L	2

3-dimethylaminopropylamine

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	=100mg/L	1
EC50	48	Crustacea	59.46mg/L	2
EC50	72	Algae or other aquatic plants	7-120mg/L	2
EC10	528	Crustacea	5.65mg/L	2
NOEC	528	Crustacea	3.64mg/L	2

monomethyl phosphate ethoxylated

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

carbon black

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	>100mg/L	2
EC50	72	Algae or other aquatic plants	>10-mg/L	2
EC10	72	Algae or other aquatic plants	>10-mg/L	2
NOEC	96	Fish	>=1-mg/L	2

4,4'-methylenebis(cyclohexylamine)

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	68mg/L	2
EC50	48	Crustacea	6.84mg/L	2
EC50	72	Algae or other aquatic plants	2-164mg/L	2
EC0	48	Crustacea	2.5mg/L	2

Continued...

	NOEC	504	Crustacea	4mg/L	2

phenol

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	5.02mg/L	2
EC50	48	Crustacea	3.1mg/L	2
EC50	72	Algae or other aquatic plants	1.91mg/L	2
NOEC	1440	Fish	0.077mg/L	2

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

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
For aluminium and its compounds and salts:
Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals.Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.
Environmental fate:
Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.
Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.
As an element, aluminum cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminum in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminum can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminum in the environment will depend on the ligands present and the pH.
The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion, [Al(H2O)6]3+, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H2O)5(OH)]2+, [Al(H2O)4(OH)2]+). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)2+ and Al(OH)2+, while the solid Al(OH)3 is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)4- is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)3, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.
Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.
Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.
The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.
Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.
Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.
Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.
The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.
Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail.
Bioconcentration of aluminum has also been reported for aquatic insects.
Ecotoxicity:
Freshwater species pH >6.5
Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar)* - 106 mg/L; Chronic NOEC (8-28 d): 7 spp,NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.
Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L
Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L
Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L
Freshwater species pH <6.5 (all between pH 4.5 and 6.0)
Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L
Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)
Alga: 1 sp NOEC growth 2.0 mg/L
Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.
The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4.5.4, with a maximum toxicity occurring around pH 5.0.5.2. The inorganic single unit aluminium species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.
Drinking Water Standards:
aluminium: 200 ug/l (UK max.)
200 ug/l (WHO guideline)
chloride: 400 mg/l (UK max.)
250 mg/l (WHO guideline)
fluoride: 1.5 mg/l (UK max.)

Continued...

1.5 mg/l (WHO guideline)
nitrate: 50 mg/l (UK max.)
50 mg/l (WHO guideline)
sulfate: 250 mg/l (UK max.)
Soil Guideline: none available.
Air Quality Standards: none available.
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
benzyl alcohol	LOW	LOW
3-dimethylaminopropylamine	HIGH	HIGH
4,4'-methylenebis(cyclohexylamine)	HIGH	HIGH
phenol	LOW (Half-life = 10 days)	LOW (Half-life = 0.95 days)

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
benzyl alcohol	LOW (LogKOW = 1.1)
3-dimethylaminopropylamine	LOW (LogKOW = -0.4502)
4,4'-methylenebis(cyclohexylamine)	LOW (LogKOW = 3.2649)
phenol	LOW (BCF = 17.5)

12.4. Mobility in soil

Ingredient	Mobility
benzyl alcohol	LOW (KOC = 15.66)
3-dimethylaminopropylamine	LOW (KOC = 73.36)
4,4'-methylenebis(cyclohexylamine)	LOW (KOC = 672.4)
phenol	LOW (KOC = 268)

12.5.Results of PBT and vPvB assessment

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

12.6. Other adverse effects

No data available

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 Transport information

Continued...

Land transport (ADR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. **UN number**	Not Applicable
14.2. **UN proper shipping name**	Not Applicable
14.3. **Transport hazard class(es)**	Class: Not Applicable Subrisk: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Hazard identification (Kemler): Not Applicable Classification code: Not Applicable Hazard Label: Not Applicable Special provisions: Not Applicable Limited quantity: Not Applicable Tunnel Restriction Code: Not Applicable

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. **UN number**	Not Applicable
14.2. **UN proper shipping name**	Not Applicable
14.3. **Transport hazard class(es)**	ICAO/IATA Class: Not Applicable ICAO / IATA Subrisk: Not Applicable ERG Code: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: Not Applicable Cargo Only Packing Instructions: Not Applicable Cargo Only Maximum Qty / Pack: Not Applicable Passenger and Cargo Packing Instructions: Not Applicable Passenger and Cargo Maximum Qty / Pack: Not Applicable Passenger and Cargo Limited Quantity Packing Instructions: Not Applicable Passenger and Cargo Limited Maximum Qty / Pack: Not Applicable

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. **UN number**	Not Applicable
14.2. **UN proper shipping name**	Not Applicable
14.3. **Transport hazard class(es)**	IMDG Class: Not Applicable IMDG Subrisk: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	EMS Number: Not Applicable Special provisions: Not Applicable Limited Quantities: Not Applicable

Inland waterways transport (ADN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. **UN number**	Not Applicable
14.2. **UN proper shipping name**	Not Applicable
14.3. **Transport hazard class(es)**	Not Applicable: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Classification code: Not Applicable Special provisions: Not Applicable Limited quantity: Not Applicable

Continued...

Equipment required	Not Applicable
Fire cones number	Not Applicable

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

aluminium hydroxide is found on the following regulatory lists

Europe EC Inventory

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

aluminium oxide is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

Europe EC Inventory

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

UK Workplace Exposure Limits (WELs)

benzyl alcohol is found on the following regulatory lists

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

Europe EC Inventory

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

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

formaldehyde/ benzenamine, hydrogenated is found on the following regulatory lists

Not Applicable

3-dimethylaminopropylamine is found on the following regulatory lists

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

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

Europe EC Inventory

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

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

monomethyl phosphate ethoxylated is found on the following regulatory lists

Not Applicable

carbon black is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

Europe EC Inventory

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

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B : Possibly carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

UK Workplace Exposure Limits (WELs)

4,4'-methylenebis(cyclohexylamine) is found on the following regulatory lists

Europe EC Inventory

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

phenol is found on the following regulatory lists

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

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

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

Europe EC Inventory

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

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

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

UK Workplace Exposure Limits (WELs)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2015/830; Regulation (EC) No 1272/2008 as updated through ATPs.

15.2. Chemical safety assessment

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

National Inventory Status

National Inventory	Status
Australia - AIIC	No (monomethyl phosphate ethoxylated)
Australia - Non-Industrial Use	No (aluminium hydroxide; aluminium oxide; benzyl alcohol; formaldehyde/ benzenamine, hydrogenated; 3-dimethylaminopropylamine; monomethyl phosphate ethoxylated; carbon black; 4,4'-methylenebis(cyclohexylamine); phenol)
Canada - DSL	No (monomethyl phosphate ethoxylated)
Canada - NDSL	No (aluminium hydroxide; aluminium oxide; benzyl alcohol; formaldehyde/ benzenamine, hydrogenated; 3-dimethylaminopropylamine; carbon black; 4,4'-methylenebis(cyclohexylamine); phenol)
China - IECSC	No (monomethyl phosphate ethoxylated)
Europe - EINEC / ELINCS / NLP	No (formaldehyde/ benzenamine, hydrogenated; monomethyl phosphate ethoxylated)

Continued...

National Inventory	Status
Japan - ENCS	No (formaldehyde/ benzenamine, hydrogenated; monomethyl phosphate ethoxylated)
Korea - KECI	No (monomethyl phosphate ethoxylated)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (monomethyl phosphate ethoxylated)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (formaldehyde/ benzenamine, hydrogenated; monomethyl phosphate ethoxylated; 4,4'-methylenebis(cyclohexylamine))
Vietnam - NCI	Yes
Russia - ARIPS	No (formaldehyde/ benzenamine, hydrogenated; monomethyl phosphate ethoxylated)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	25/09/2020
Initial Date	26/09/2020

Full text Risk and Hazard codes

H226	Flammable liquid and vapour.
H290	May be corrosive to metals.
H301	Toxic if swallowed.
H302	Harmful if swallowed.
H311	Toxic in contact with skin.
H314	Causes severe skin burns and eye damage.
H319	Causes serious eye irritation.
H331	Toxic if inhaled.
H332	Harmful if inhaled.
H341	Suspected of causing genetic defects.
H351	Suspected of causing cancer.
H373	May cause damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.
H413	May cause long lasting harmful effects to aquatic life.

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.
For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:
EN 166 Personal eye-protection
EN 340 Protective clothing
EN 374 Protective gloves against chemicals and micro-organisms
EN 13832 Footwear protecting against chemicals
EN 133 Respiratory protective devices

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
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-1.00 - New Release