834ATH-Part B ATH Flame Retardant Epoxy

MG Chemicals (Head office)

Chemwatch Hazard Alert Code: **4**

Version No: **5.11**
Safety Data Sheet (Conforms to Regulations (EC) No 2015/830)

Issue Date: **04/02/2016**
Print Date: **04/02/2016**
Initial Date: **10/07/2013**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

1.1.Product Identifier

Product name	834ATH-Part B ATH Flame Retardant Epoxy
Synonyms	SDS Code: 834ATH-Part B; Related Part #: 834ATH-375ML, 834ATH-3L, 834ATH-60L
Proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains zinc borate hydrate and triethylenetetramine)
Other means of identification	Not Available

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

Relevant identified uses	Epoxy hardener for use with resins to pot devices or encapsulate components
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals (Head office)	MG Chemicals UK Limited
Address	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada	October House, 17 Dudley Street, Sedgley DY3 1SA Dudley United Kingdom
Telephone	+1 800 201 8822	+44 1663 362888
Fax	+1 800 708 9888	Not Available
Website	www.mgchemicals.com	Not Available
Email	Info@mgchemicals.com	sales@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	Not Available	CHEMTREC
Emergency telephone numbers	Not Available	+(44)-870-8200418
Other emergency telephone numbers	Not Available	+(1) 703-527-3887

SECTION 2 HAZARDS IDENTIFICATION

2.1.Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	Skin Corrosion/Irritation Category 1B, Skin Sensitizer Category 1, Carcinogen Category 2, Chronic Aquatic Hazard Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

2.2. Label elements

CLP label elements	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H314	Causes severe skin burns and eye damage
H317	May cause an allergic skin reaction

Continued...

H351	Suspected of causing cancer
H411	Toxic to aquatic life with long lasting effects

Supplementary statement(s)

EUH066	Repeated exposure may cause skin dryness or cracking

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

2.3. Other hazards

Skin contact may produce health damage*.

Cumulative effects may result following exposure*.

Possible respiratory sensitizer*.

May be harmful to the foetus/ embryo*.

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.68410-23-1 2.Not Available 3.Not Available 4.Not Available	51	C18 fatty acid dimers/ tetraethylenepentamine polyamides	Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, STOT - SE (Resp. Irr.) Category 3; H315, H318, H335 [1]
1.21645-51-2 2.244-492-7 3.Not Available 4.01-2119529246-39-XXXX	17	aluminium hydroxide	Eye Irritation Category 2; H319, EUH066 [1]
1.84852-53-9 2.284-366-9 3.Not Available 4.01-2119474877-18-XXXX	13	decabromodiphenylethane	STOT - SE (Resp. Irr.) Category 3; H335 [1]
1.112-24-3 2.203-950-6 3.612-059-00-5	6	triethylenetetramine	Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1B, Skin Sensitizer Category 1, Chronic Aquatic Hazard Category 3; H312, H314, H317, H412 [3]

Continued...

4.Not Available			
1.138265-88-0 2.Not Available 3.Not Available 4.Not Available	6	zinc borate hydrate	Chronic Aquatic Hazard Category 1; H410 [1]
1.1309-64-4 2.215-175-0 3.051-005-00-X 4.01-2119475613-35-XXXX	2	antimony trioxide	Carcinogen Category 2; H351 [3]
1.108-65-6 2.203-603-9, 283-152-2 3.607-195-00-7 4.01-2119475791-29-XXXX	1	propylene glycol monomethyl ether acetate, alpha-isomer	Flammable Liquid Category 3; H226 [3]
1.64741-65-7. 2.265-067-2 3.649-275-00-4 4.01-2119850115-46-XXXX	1	naphtha petroleum, heavy alkylate	Flammable Liquid Category 3, STOT - SE (Narcosis) Category 3, Aspiration Hazard Category 1; H226, H336, H304 [1]
1.8052-41-3. 2.265-149-8, 265-185-4, 232-489-3 3.649-422-00-2, 649-330-00-2, 649-345-00-4 4.01-2119484819-18-XXXX, 01-2119942421-46-XXXX, 01-2119490979-12-XXXX	0.6	Stoddard Solvent	Flammable Liquid Category 3, STOT - SE (Narcosis) Category 3, Aspiration Hazard Category 1; H226, H336, H304, EUH066 [1]
1.1333-86-4 2.215-609-9 3.Not Available 4.01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX	0.5	carbon black	Carcinogen Category 2; H351 [1]
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI 4. Classification drawn from C&L*		

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

General

If skin or hair contact occurs:
- Immediately flush body and clothes with large amounts of water, using safety shower if available.
- Quickly remove all contaminated clothing, including footwear.
- Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.
- Transport to hospital, or doctor.

For amines:
- In case of major exposure to liquid amine, promptly remove any contaminated clothing, including rings, watches, and shoe, preferably under a safety shower.
- Wash skin for 15 to 30 minutes with plenty of water and soap. Call a physician immediately.
- Remove and dry-clean or launder clothing soaked or soiled with this material before reuse. Dry cleaning of contaminated clothing may be more effective than normal laundering.
- Inform individuals responsible for cleaning of potential hazards associated with handling contaminated clothing.
- Discard contaminated leather articles such as shoes, belts, and watchbands.
- Note to Physician: Treat any skin burns as thermal burns. After decontamination, consider the use of cold packs and topical antibiotics.

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.

For amines:
- If liquid amines come in contact with the eyes, irrigate immediately and continuously with low pressure flowing water, preferably from an eye wash fountain, for 15 to 30 minutes.
- For more effective flushing of the eyes, use the fingers to spread apart and hold open the eyelids. The eyes should then be “rolled” or moved in all directions.
- Seek immediate medical attention, preferably from an ophthalmologist.
- If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested.
- Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- Transport to hospital, or doctor.
- Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema.
- Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).
- As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested.
- Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered.

This must definitely be left to a doctor or person authorised by him/her.

(ICSC13719)

For amines:
- All employees working in areas where contact with amine catalysts is possible should be thoroughly trained in the administration of appropriate first aid procedures.
- Experience has demonstrated that prompt administration of such aid can minimize the effects of accidental exposure.
- Promptly move the affected person away from the contaminated area to an area of fresh air.
- Keep the affected person calm and warm, but not hot.
- If breathing is difficult, oxygen may be administered by a qualified person.

Continued...

	▸ If breathing stops, give artificial respiration. Call a physician at once. ▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ 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. ▸ Transport to hospital or doctor without delay. For amines: ▸ If liquid amine are ingested, have the affected person drink several glasses of water or milk. ▸ Do not induce vomiting. ▸ Immediately transport to a medical facility and inform medical personnel about the nature of the exposure. The decision of whether to induce vomiting should be made by an attending physician.
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. For amines: ▸ If liquid amines come in contact with the eyes, irrigate immediately and continuously with low pressure flowing water, preferably from an eye wash fountain, for 15 to 30 minutes. ▸ For more effective flushing of the eyes, use the fingers to spread apart and hold open the eyelids. The eyes should then be “rolled” or moved in all directions. ▸ Seek immediate medical attention, preferably from an ophthalmologist.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor. For amines: ▸ In case of major exposure to liquid amine, promptly remove any contaminated clothing, including rings, watches, and shoe, preferably under a safety shower. ▸ Wash skin for 15 to 30 minutes with plenty of water and soap. Call a physician immediately. ▸ Remove and dry-clean or launder clothing soaked or soiled with this material before reuse. Dry cleaning of contaminated clothing may be more effective than normal laundering. ▸ Inform individuals responsible for cleaning of potential hazards associated with handling contaminated clothing. ▸ Discard contaminated leather articles such as shoes, belts, and watchbands. ▸ Note to Physician: Treat any skin burns as thermal burns. After decontamination, consider the use of cold packs and topical antibiotics.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor. ▸ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. ▸ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs). ▸ As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested. ▸ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered. **This must definitely be left to a doctor or person authorised by him/her.** (ICSC13719) For amines: ▸ All employees working in areas where contact with amine catalysts is possible should be thoroughly trained in the administration of appropriate first aid procedures. ▸ Experience has demonstrated that prompt administration of such aid can minimize the effects of accidental exposure. ▸ Promptly move the affected person away from the contaminated area to an area of fresh air. ▸ Keep the affected person calm and warm, but not hot. ▸ If breathing is difficult, oxygen may be administered by a qualified person. ▸ If breathing stops, give artificial respiration. Call a physician at once.
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ 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. ▸ Transport to hospital or doctor without delay. For amines: ▸ If liquid amine are ingested, have the affected person drink several glasses of water or milk. ▸ Do not induce vomiting. ▸ Immediately transport to a medical facility and inform medical personnel about the nature of the exposure. The decision of whether to induce vomiting should be made by an attending physician.

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.

- Chelation with British Anti-Lewisite (BAL) for serious antimony exposures should be employed.
- Dialyse as needed. The role of exchange diffusion is not clear.
- Be sure to monitor for dysrhythmias.

[Ellenhorn and Barceloux: Medical Toxicology]

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For acute or short-term repeated exposures to highly alkaline materials:
- Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- Oxygen is given as indicated.
- The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.
INGESTION:
- Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.
- Neutralising agents should never be given since exothermic heat reaction may compound injury.

* Catharsis and emesis are absolutely contra-indicated.
* Activated charcoal does not absorb alkali.
* Gastric lavage should not be used.

Supportive care involves the following:
- Withhold oral feedings initially.
- If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:
- Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]
For amines:
- Certain amines may cause injury to the respiratory tract and lungs if aspirated. Also, such products may cause tissue destruction leading to stricture. If lavage is performed, endotracheal and/or esophagoscopic control is suggested.
- No specific antidote is known.
- Care should be supportive and treatment based on the judgment of the physician in response to the reaction of the patient.

Laboratory animal studies have shown that a few amines are suspected of causing depletion of certain white blood cells and their precursors in lymphoid tissue. These effects may be due to an immunosuppressive mechanism.
Some persons with hyperreactive airways (e.g., asthmatic persons) may experience wheezing attacks (bronchospasm) when exposed to airway irritants.
Lung injury may result following a single massive overexposure to high vapour concentrations or multiple exposures to lower concentrations of any pulmonary irritant material.
Health effects of amines, such as skin irritation and transient corneal edema (“blue haze,” “halo effect,” “glaucopsia”), are best prevented by means of formal worker education, industrial hygiene monitoring, and exposure control methods. Persons who are highly sensitive to the triggering effect of non-specific irritants should not be assigned to jobs in which such agents are used, handled, or manufactured.
Medical surveillance programs should consist of a pre-placement evaluation to determine if workers or applicants have any impairments (e.g., hyperreactive airways or bronchial asthma) that would limit their fitness for work in jobs with potential for exposure to amines. A clinical baseline can be established at the time of this evaluation.
Periodic medical evaluations can have significant value in the early detection of disease and in providing an opportunity for health counseling.
Medical personnel conducting medical surveillance of individuals potentially exposed to polyurethane amine catalysts should consider the following:
- Health history, with emphasis on the respiratory system and history of infections
- Physical examination, with emphasis on the respiratory system and the lymphoreticular organs (lymph nodes, spleen, etc.)
- Lung function tests, pre- and post-bronchodilator if indicated
- Total and differential white blood cell count
- Serum protein electrophoresis

Persons who are concurrently exposed to isocyanates also should be kept under medical surveillance.
Pre-existing medical conditions generally aggravated by exposure include skin disorders and allergies, chronic respiratory disease (e.g. bronchitis, asthma, emphysema), liver disorders, kidney disease, and eye disease.
Broadly speaking, exposure to amines, as characterised by amine catalysts, may cause effects similar to those caused by exposure to ammonia. As such, amines should be considered potentially injurious to any tissue that is directly contacted.
Inhalation of aerosol mists or vapors, especially of heated product, can result in chemical pneumonitis, pulmonary edema, laryngeal edema, and delayed scarring of the airway or other affected organs. There is no specific treatment.
Clinical management is based upon supportive treatment, similar to that for thermal burns.
Persons with major skin contact should be maintained under medical observation for at least 24 hours due to the possibility of delayed reactions.
Polyurethene Amine Catalysts: Guidelines for Safe Handling and Disposal Technical Bulletin June 2000
Alliance for Polyurethanes Industry

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 fire fighting procedures suitable for surrounding area. ‣ **Do not** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use. For amines: ‣ For firefighting, cleaning up large spills, and other emergency operations, workers must wear a self-contained breathing apparatus with full face-piece, operated in a pressure-demand mode. ‣ Airline and air purifying respirators should not be worn for firefighting or other emergency or upset conditions. ‣ Respirators should be used in conjunction with a respiratory protection program, which would include suitable fit testing and medical evaluation of the user.

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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) nitrogen oxides (NOx) other pyrolysis products typical of burning organic materialMay emit corrosive fumes.

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

▸ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.
▸ Check regularly for spills and leaks.

Small spills should be covered with inorganic absorbents and disposed of properly. Organic absorbents have been known to ignite when contaminated with amines in closed containers. Certain cellulosic materials used for spill cleanup such as wood chips or sawdust have shown reactivity with ethyleneamines and should be avoided. Ethyleneamine leaks will frequently be identified by the odor (ammoniacal) or by the formation of a white, solid, waxy substance (amine carbamates). Inorganic absorbents or water may be used to clean up the amine waste.

▸ 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.

for amines:

▸ If possible (i.e., without risk of contact or exposure), stop the leak.
▸ Contain the spilled material by diking, then neutralize.
▸ Next, absorb the neutralized product with clay, sawdust, vermiculite, or other inert absorbent and shovel into containers.
▸ Store the containers outdoors.
▸ Brooms and mops should be disposed of, along with any remaining absorbent, in accordance with all applicable federal, state, and local regulations and requirements.
▸ Decontamination of floors and other hard surfaces after the spilled material has been removed may be accomplished by using a 5% solution of acetic acid, followed by very hot water
▸ Dispose of the material in full accordance with all federal, state, and local laws and regulations governing the disposal of chemical wastes.
▸ Waste materials from an amine catalyst spill or leak may be “hazardous wastes” that are regulated under various laws.

Major Spills

Chemical Class: bases
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross-linked polymer - particulate	1	shovel	shovel	R,W,SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R, I, P
foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT
expanded minerals - particulate	3	shovel	shovel	R, I, W, P, DGC
foamed glass - particulate	4	shovel	shovel	R, W, P, DGC,
LAND SPILL - MEDIUM				
cross-linked polymer -particulate	1	blower	skiploader	R,W, SS
sorbent clay - particulate	2	blower	skiploader	R, I, P
expanded mineral - particulate	3	blower	skiploader	R, I,W, P, DGC
cross-linked polymer - pillow	3	throw	skiploader	R, DGC, RT
foamed glass - particulate	4	blower	skiploader	R, W, P, DGC
foamed glass - pillow	4	throw	skiploader	R, P, DGC., RT

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

▸ Clear area of personnel and move upwind.
▸ 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.

Continued...

	▸ Consider evacuation (or protect in place). ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services. For amines: ▸ First remove all ignition sources from the spill area. ▸ Have firefighting equipment nearby, and have firefighting personnel fully trained in the proper use of the equipment and in the procedures used in fighting a chemical fire. ▸ Spills and leaks of polyurethane amine catalysts should be contained by diking, if necessary, and cleaned up only by properly trained and equipped personnel. All others should promptly leave the contaminated area and stay upwind. ▸ Protective equipment for cleanup crews should include appropriate respiratory protective devices and impervious clothing, footwear, and gloves. ▸ All work areas should be equipped with safety showers and eyewash fountains in good working order. ▸ Any material spilled or splashed onto the skin should be quickly washed off. ▸ Spills or releases may need to be reported to federal, state, and local authorities. This reporting contingency should be a part of a site's emergency response plan. ▸ Protective equipment should be used during emergency situations whenever there is a likelihood of exposure to liquid amines or to excessive concentrations of amine vapor. "Emergency" may be defined as any occurrence, such as, but not limited to, equipment failure, rupture of containers, or failure of control equipment that results in an uncontrolled release of amine liquid or vapor. ▸ Emergency protective equipment should include: ▸ • Self-contained breathing apparatus, with full face-piece, operated in positive pressure or pressure-demand mode. ▸ • Rubber gloves ▸ • Long-sleeve coveralls or impervious full body suit ▸ • Head protection, such as a hood, made of material(s) providing protection against amine catalysts ▸ Firefighting personnel and other on-site Emergency Responders should be fully trained in Chemical Emergency Procedures. However back-up from local authorities should be sought

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. ▸ **WARNING:** To avoid violent reaction, **ALWAYS** add material to water and **NEVER** water to material. ▸ 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. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin
Fire and explosion protection	See section 5
Other information	for bulk storages: ▸ If slight coloration of the ethyleneamine is acceptable, storage tanks may be made of carbon steel or black iron, provided they are free of rust and mill scale. However, if the amine is stored in such tanks, color may develop due to iron contamination. If iron contamination cannot be tolerated, tanks constructed of types 304 or 316 stainless steel should be used. (Note: Because they are quickly corroded by amines, do not use copper, copper alloys, brass, or bronze in tanks or lines.) ▸ This product should be stored under a dry inert gas blanket, such as nitrogen, to minimize contamination resulting from contact with air and water ▸ Store in original containers. ▸ Keep containers securely sealed. ▸ 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. ▸ **DO NOT** store near acids, or oxidising agents ▸ No smoking, naked lights, heat or ignition sources.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	▸ **DO NOT** use aluminium, galvanised or tin-plated containers ▸ Lined metal can, lined metal pail/ can. ▸ Plastic pail. ▸ Polyliner drum. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks. For low viscosity materials ▸ Drums and jerricans must be of the non-removable head type. ▸ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ▸ Removable head packaging; ▸ Cans with friction closures and

Continued...

	▸ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	Triethylenetetramine (TETA): ▸ aqueous solutions are strong organic bases ▸ reacts with nitrogen containing compounds; may cause violent decomposition ▸ reacts violently with strong oxidisers, nitroparaffins, nitrogen tetroxide, permanganates, peroxides, ammonium persulfate, bromine dioxide, sulfuric acid, nitric acid ▸ is incompatible with organic anhydrides (eg maleic anhydride), acrylates, alcohols, aldehydes, alkylene oxides, substituted allyls, cellulose nitrate, cresols, caprolactam solutions, epichlorohydrin, ethylene dichloride, glycols, halons, halogenated hydrocarbons, isocyanates, ketones, methyl trichloroacetate, nitrates, phenols, urea, vinyl acetate ▸ increases the explosive sensitivity of nitromethane ▸ attacks aluminium, cobalt, copper, lad, nickel, tin zinc, and their alloys, and some plastics, rubber and coatings ▸ reacts with halon fire extinguishers ▸ Avoid contact with copper, aluminium and their alloys. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ▸ 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

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	antimony trioxide	Antimony and compounds except stibine (as Sb)	0.5 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl acetate	274 mg/m3 / 50 ppm	548 mg/m3 / 100 ppm	Not Available	Sk
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	propylene glycol monomethyl ether acetate, alpha-isomer	2-Methoxy-1-methylethylacetate	275 mg/m3 / 50 ppm	550 mg/m3 / 100 ppm	Not Available	Skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl-2-acetate	275 mg/m3 / 50 ppm	550 mg/m3 / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	Stoddard Solvent	Cobalt and Cobalt compounds (as Co)	0.1 mg/m3	Not Available	Not Available	Carc (cobalt dichloride andsulphate), Sen
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m3	7 mg/m3	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
C18 fatty acid dimers/ tetraethylenepentamine polyamides	C-18 Unsaturated fatty acid, dimers, reaction products with polyethylenepolyamines; (Versamid 140 polyamide resin; Versamid 125)	30 mg/m3	330 mg/m3	2000 mg/m3
aluminium hydroxide	Aluminum hydroxide	3 mg/m3	79 mg/m3	120 mg/m3
triethylenetetramine	Triethylenetetramine	3 ppm	5.7 ppm	83 ppm
antimony trioxide	Antimony oxide	0.6 mg/m3	0.6 mg/m3	96 mg/m3
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
Stoddard Solvent	Stoddard solvent; (Mineral spirits, 85% nonane and 15% trimethyl benzene)	100 ppm	350 ppm	29500 ppm
carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
C18 fatty acid dimers/ tetraethylenepentamine polyamides	Not Available	Not Available
aluminium hydroxide	Not Available	Not Available
decabromodiphenylethane	Not Available	Not Available

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triethylenetetramine	Not Available	Not Available
zinc borate hydrate	Not Available	Not Available
antimony trioxide	80 mg/m3	50 mg/m3
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
naphtha petroleum, heavy alkylate	Not Available	Not Available
Stoddard Solvent	29,500 mg/m3	20,000 mg/m3
carbon black	N.E. mg/m3 / N.E. ppm	1,750 mg/m3

MATERIAL DATA

Polyamide hardeners have much reduced volatility, toxicity and are much less irritating to the skin and eyes than amine hardeners. However commercial polyamides may contain a percentage of residual unreacted amine and all unnecessary contact should be avoided.
for propylene glycol monomethyl ether acetate (PGMEA)
Saturated vapour concentration: 4868 ppm at 20 C.
A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternabral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

The wide-ranging effects of antimony compounds have made it difficult to recommend an exposure standard which characterises the toxicology of these substances. One criteria, reflecting the irritant properties of antimony pentachloride, produced a calculated value of 5.0 mg/m3 (as antimony), which on the basis of experience was felt to be too high but did act as an "out-rider". The present value reflects this thinking.
NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP
NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

8.2. Exposure controls

8.2.1. Appropriate engineering controls

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

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed 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

Continued...

Eye and face protection	▸ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ▸ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ▸ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ▸ Alternatively a gas mask may replace splash goggles and face shields. ▸ 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] For amines: SPECIAL PRECAUTION: ▸ Because amines are alkaline materials that can cause rapid and severe tissue damage, wearing of contact lenses while working with amines is strongly discouraged. Wearing such lenses can prolong contact of the eye tissue with the amine, thereby causing more severe damage. ▸ Appropriate eye protection should be worn whenever amines are handled or whenever there is any possibility of direct contact with liquid products, vapors, or aerosol mists. CAUTION: ▸ Ordinary safety glasses or face-shields will not prevent eye irritation from high concentrations of vapour. ▸ In operations where positive-pressure, air-supplied breathing apparatus is not required, all persons handling liquid amine catalysts or other polyurethane components in open containers should wear chemical workers safety goggles. ▸ Eyewash fountains should be installed, and kept in good working order, wherever amines are used.
Skin protection	See Hand protection below
Hands/feet protection	▸ Elbow length PVC gloves ▸ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. **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. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: ▸ frequency and duration of contact, ▸ chemical resistance of glove material, ▸ glove thickness and ▸ dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). ▸ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ▸ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ▸ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. ▸ Contaminated gloves should be replaced. 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. ▸ When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butatoluene rubber), boots and aprons. ▸ **DO NOT** use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber or polyethylene gloves (which absorb the resin). ▸ **DO NOT** use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use. For amines: ▸ 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 ▸ Where there is a possibility of exposure to liquid amines skin protection should include: rubber gloves, (neoprene, nitrile, or butyl). ▸ DO NOT USE latex.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
834ATH-Part B ATH Flame Retardant Epoxy

Material	CPI
BUTYL	A
NEOPRENE	A
NITRILE	A
PE/EVAL/PE	A
VITON	A

Respiratory protection

Type AK-P 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	AK-AUS / Class 1 P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	AK-3 P2	-

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* 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.

100+ x ES	-	Air-line**	-

* - Continuous-flow; ** - Continuous-flow or positive pressure demand
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)

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)**	1.258
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	10000
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	>185	**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 (g/L)	Miscible	**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	Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales. The material is not thought to produce adverse health effects following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation of amine vapours may cause irritation of the mucous membranes of the nose and throat and lung irritation with respiratory distress and cough. Single exposures to near lethal concentrations and repeated exposures to sublethal concentrations produces tracheitis, bronchitis, pneumonitis and pulmonary oedema. Aliphatic and alicyclic amines are generally well absorbed from the respiratory tract. Systemic effects include headache, nausea, faintness and anxiety. These effects are thought to be transient and are probably related to the pharmacodynamic action of the amines. Histamine release by aliphatic amines may produce bronchoconstriction and wheezing. Inhalation of antimony and its compounds may produce respiratory and gastrointestinal tract discomfort with sore throat, shallow respiration, coughing,

Continued...

	headaches, breathing difficulties, dizziness, weight loss, gingivitis, anaemia, eosinophilia and enzyme inhibition. Inflammation of the upper and lower respiratory tract may occur. Pulmonary congestion and oedema may also occur. Other symptoms include rhinitis, eye irritation, vomiting and diarrhoea, weight loss, dysomnia, hair loss and haematological disorders. Death due to circulatory failure has been described, with pathology showing acute congestion of the heart (myocardial failure), liver and kidneys.
Ingestion	Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation. Symptoms of borate poisoning include nausea, vomiting, diarrhoea, epigastric pain. These may be accompanied headache, weakness and a distinctive red skin rash. In severe cases there may be shock, increased heart rate and the skin may appear blue. Vomiting (which may be violent) is often persistent and vomitus and faeces may contain blood. Weakness, lethargy, headache, restlessness, tremors and intermittent convulsions may also occur. Poisoning produces central nervous system stimulation followed by depression, gastrointestinal disturbance (haemorrhagic gastro-enteritis), erythematous skin eruptions (giving rise to a boiled lobster appearance) and may also involve kidneys (producing oliguria, albuminuria, anuria) and, rarely, liver (hepatomegaly, jaundice). Toxic symptoms may be delayed for several hours. Ingested borates are readily absorbed and do not appear to be metabolised via the liver. Excretion occurs mainly through the kidneys in the urine with about half excreted in the first 12 hours and the remainder over 5-12 days. Borates are excreted primarily in the urine regardless of the route of administration. The borates (tetra-, di-, meta, or ortho- salts, in contrast to perborates) once solubilised in the acid of gastric juices, cannot be distinguished from each other on chemical or toxicological grounds. In humans acute gastroenteric (or percutaneous absorption of as little as 1 gm of sodium borate can result in severe gastrointestinal irritation, kidney damage. In adults the mean lethal dose of sodium borate or boric acid probably exceeds 30 gms (Gosselin) and death occurs due to vascular collapse in the early stages or to central nervous system depression in later stages. Children are thought to be more susceptible to the effects of borate intoxication. Aliphatic and alicyclic amines are generally well absorbed from the gut. Corrosive action may cause tissue damage throughout the gastrointestinal tract. Detoxification is thought to occur in the liver, kidney and intestinal mucosa with the enzymes, monoamine oxidase and diamine oxidase (histaminase) having a significant role. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. Ingestion of antimony compounds may produce violent irritation of the nose, throat, stomach and gastrointestinal tract, vomiting, purging with bloody stools, slow shallow respiration, pulmonary oedema, convulsions, loss of consciousness, coma, and death due to circulatory or respiratory failure. Early signs of antimony intoxication include: fatigue, muscle weakness, myopathy, nausea, low back pain, headache, and metallic taste. Later symptoms include blood disorders (haemolytic anemia, myoglobinuria, haematuria) and renal failure. The substance may cause cough, salivation, nausea, and diarrhoea. It may also cause dizziness, laryngitis, anaemia, muscular and neuralgic pains. Other symptoms of overexposure may include tightness of the chest, pain, swelling of the cervical glands, pustular eruptions ("antimony spots"), particularly on the scrotum, difficult urination, sexual disorders, nervousness, loss of sleep, leukopenia, unconsciousness and death. [NIOSHTIC] The minimum lethal dose in man, of antimony, is 130 mg (although 15000 mg has been survived). Antimony is a strong irritant and emetic but the emetic dose (30 mg by mouth) is dangerously high if vomiting fails to occur. Trivalent compounds are generally more lethal than pentavalent derivatives. The trivalent antimony compounds are cardiotoxic. The insoluble salts however are less likely to produce significant toxic effects. Antimony salts are poorly absorbed from the gastrointestinal tract. Most trivalent compounds of antimony are slowly excreted in the urine and bile. Antimony compounds are used medicinally, but therapeutic dose is close to the toxic dose. Antimony poisoning closely parallels arsenic poisoning although vomiting is probably more prominent as absorption from the gastrointestinal tract is generally lower. Like arsenic, antimony has a high affinity for sulfhydryl groups on many enzymes. Antimony is conjugated with glutathione and excreted in urine and feces. Therefore, excessive exposure to antimony has the potential to deplete intracellular glutathione pools. Temporary changes in heart rhythm occurs amongst humans while poisoned animals exhibit severe heart damage. Periodic medical examinations covering lungs, skin, nervous system, heart and gastro-intestinal tract are recommended for occupationally exposed workers. [ILO Encyclopedia]
Skin Contact	The material can produce severe chemical burns following direct contact with the skin. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep. Volatile amine vapours produce primary skin irritation and dermatitis. Direct local contact, with the lower molecular weight liquids, may produce skin burns. Percutaneous absorption of simple aliphatic amines is known to produce lethal effects often the same as that for oral administration. Cutaneous sensitisation has been recorded chiefly due to ethyleneamines. Histamine release following exposure to many aliphatic amines may result in "triple response" (white vasoconstriction, red flare and wheal) in human skin. Skin contact with antimony compounds may result in redness and severe irritation with the formation of itchy papules, pustules, skin lesions/ small septic blisters (antimony spots) within a few hours. Rhinitis may also result from dermal contact. 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	Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight. Vapours of volatile amines cause eye irritation with lachrymation, conjunctivitis and minor transient corneal oedema which results in "halos" around lights (glaucopsia, "blue haze", or "blue-grey haze"). Vision may become misty and halos may appear several hours after workers are exposed to the substance This effect generally disappears spontaneously within a few hours of the end of exposure, and does not produce physiological after-effects. However oedema of the corneal epithelium, which is primarily responsible for vision disturbances, may take more than one or more days to clear, depending on the severity of exposure. Photophobia and discomfort from the roughness of the corneal surface also may occur after greater exposures. Although no detriment to the eye occurs as such, glaucopsia predisposes an affected individual to physical accidents and reduces the ability to undertake skilled tasks such as driving a vehicle. Direct local contact with the liquid may produce eye damage which may be permanent in the case of the lower molecular weight species.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Continued...

Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.
Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.
Secondary amines may react in the acid conditions of the stomach with oxidants or preservatives) to form potentially carcinogenic N-nitrosamines. The formation of nitrosamines from such amines has not only been observed in animals models but, at least for certain compounds, in the workplace. The amine-containing substances and end products handled at work can themselves be contaminated to a degree with corresponding nitrosamines. Under conditions encountered in practice nitrosation is to be expected with secondary amines and to a limited extent with primary and tertiary amines. Nitrogen oxides are the most probable nitrosating agents. Nitrosyl chloride, nitrite esters, metal nitrites and nitroso compounds may also be involved. Several factors such as pH, temperature, catalysts and inhibitors influence the extent of nitrosation. Two precautionary measures are therefore necessary when handling amines at the workplace.

- Simultaneous exposure to nitrosating agents should be reduced to minimum. This can be out into practice by eliminating nitrosating agents or, if they play a role in the actual process, replacing them with substances that do not lead to the formation of carcinogenic nitrosamines. In particular the level of nitrogen oxides at the workplace should be monitored and reduced when necessary.
- The levels of nitrosamines in the workplace and in substances containing amines should be monitored.

Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Report No. 31, DFG, 1995
In animal experiments the oesophagus is shown to be the most important target organ for nitrosamines, independent of the route of application. The mechanism of this organotrophy cannot be explained sufficiently. The high oesophageal epithelium metabolic activation of nitrosamines, together with a comparatively low DNA repair, probably plays the most important role. In addition chronic stress factors, which lead to high stimulation of epithelial turnover, are a pacemaker for malignant progression. In some countries, the traditional consumption of extremely hot drinks leads to constant burns of the oesophagus, which increases the risk. Mate, a non-alcoholic brew, frequently consumed as tea in Uruguay, appears to be a high risk factor for oesophageal cancer
Chronic intoxication with ionic bromides, historically, has resulted from medical use of bromides but not from environmental or occupational exposure; depression, hallucinosis, and schizophreniform psychosis can be seen in the absence of other signs of intoxication. Bromides may also induce sedation, irritability, agitation, delirium, memory loss, confusion, disorientation, forgetfulness (aphasias), dysarthria, weakness, fatigue, vertigo, stupor, coma, decreased appetite, nausea and vomiting, diarrhoea, hallucinations, an acne like rash on the face, legs and trunk, known as bronchoderma (seen in 25-30% of case involving bromide ion), and a profuse discharge from the nostrils (coryza). Ataxia and generalised hyperreflexia have also been observed. Correlation of neurologic symptoms with blood levels of bromide is inexact. The use of substances such as brompheniramine, as antihistamines, largely reflect current day usage of bromides; ionic bromides have been largely withdrawn from therapeutic use due to their toxicity.
In test animals, brominated vegetable oils (BVOs), historically used as emulsifiers in certain soda-based soft drinks, produced damage to the heart and kidneys in addition to increasing fat deposits in these organs. In extreme cases BVO caused testicular damage, stunted growth and produced lethargy and fatigue.
Brominism produces slurred speech, apathy, headache, decreased memory, anorexia and drowsiness, psychosis resembling paranoid schizophrenia, and personality changes
Several cases of foetal abnormalities have been described in mothers who took large doses of bromides during pregnancy.
Reproductive effects caused by bromide (which crosses the placenta) include central nervous system depression, brominism, and bronchoderma in the newborn.
Repeated or prolonged exposure to antimony and its compounds may produce stomatitis, dry throat, metallic taste, gingivitis, septal and laryngeal perforation, laryngitis, headache, dyspnea, indigestion, nausea, vomiting, diarrhoea, anorexia, anaemia, weight loss, pain and chest tightness, sleeplessness, muscular pain and weakness, dizziness, pharyngitis,tracheitis, bronchitis, pneumonitis, benign pneumoconiosis (with obstructive lung disease and emphysema) and haematological disorders. Degenerative changes of the liver and kidney may occur. Symptoms can be variable, and may including fatigue, myopathy (muscle aches and inflammation), hypotension, angina and immune dysregulation and hypertrophy of splenic follicles.
Antimony's deposition in body tissues and its detrimental effects depend upon the oxidation state of the element. The trivalent antimony compounds are cardiotoxic. Trivalent antimony affects liver functions, impairs enzymes, and may interfere with sulfur chemistry. If antimony impairs phosphofructokinase (PFK), then purine metabolism may be disrupted, resulting in elevated blood and/or urine levels of hypoxanthine, uric acid and possibly ammonia. Pentavalent antimony deposits in bone, kidney, and in organs of the endocrine system.
Chronic exposure to antimony compounds may result in itchiness, papules and pustules around sweat and sebaceous glands ("antimony spots"), but rarely around the face, and dermatitis.
Collapse and sudden death due to anaphylactic-type reactions have occurred. Therapeutic doses given intravenously cause nausea, vomiting, cough and abdominal pain and diarrhoea. Other side-effects include anorexia, chest, muscle and joint pains, pruritus, skin rashes, dizziness and oedema. Renal and hepatic damage occur rarely and haemolytic anaemia has been reported. Continuous treatment with small doses of antimony may give rise to subacute poisoning similar to chronic arsenic poisoning.
Smelter workers often show skin rashes on the forearms and thighs resembling chicken pox pustules.
Workers exposed to inorganic antimony compounds show a benign pneumoconiosis and obstructive lung disease - these are probably non-specific. Woman appear to more susceptible to systemic effects following exposure. Antimony crosses the placenta, is present in amniotic fluids, and is excreted in breast milk. There are suggestions that exposure may produce an increased incidence of spontaneous late abortions, premature births, and gynecological problems among female antimony smelter workers. An excess of deaths from lung cancer has been reported in smelter workers with more than 7 years exposure to relatively high levels of dust and fume. Animal studies demonstrate that the dust may produce pathological changes in cardiac muscle and may produce an interstitial pneumonitis and endogenous pneumonia. One animal study has also suggested that inhalation of the dust by rats induced a significantly increased incidence of carcinogenic tumours of the lungs and thorax. Increased chromosome defects occur when human lymphocytes are incubated with a soluble antimony salt.

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.
Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).
There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:
- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

834ATH-Part B ATH Flame Retardant Epoxy

TOXICITY	IRRITATION
Not Available	Not Available

C18 fatty acid dimers/ tetraethylenepentamine polyamides

TOXICITY	IRRITATION
dermal (rat) LD50: >5000 mg/kg*d[2]	* [Epoxylite]
Oral (rabbit) LD50: 800 mg/kg**][2]	

aluminium hydroxide

TOXICITY	IRRITATION
Oral (rat) LD50: >2000 mg/kg[1]	Not Available

Continued...

decabromodiphenylethane	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg*[2]	Not Available
	Oral (rat) LD50: >5000 mg/kg*[2]	
triethylenetetramine	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 805 mg/kgE[2]	Eye (rabbit):20 mg/24 h - moderate
	Oral (rat) LD50: 2500 mg/kgE[2]	Eye (rabbit); 49 mg - SEVERE
		Skin (rabbit): 490 mg open SEVERE
		Skin (rabbit): 5 mg/24 SEVERE
zinc borate hydrate	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
antimony trioxide	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >8000 mg/kg[1]	Nil reported
	Oral (rat) LD50: >600 mg/kg[1]	
propylene glycol monomethyl ether acetate, alpha-isomer	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	* [CCINFO]
	Inhalation (rat) LC50: 4345 ppm/6h[2]	Nil reported
	Oral (rat) LD50: >14.1 ml[1]	
naphtha petroleum, heavy alkylate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >5000 mg/kg[2]	Not Available
	Inhalation (rat) LC50: >3.83 mg/L/4H[2]	
	Oral (rat) LD50: >25000 mg/kg[2]	
Stoddard Solvent	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye (hmn) 470 ppm/15m irrit.
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye (rabbit) 500 mg/24h moderate
	Inhalation (rat) LC50: >1400 ppm/8H[2]	
	Oral (rat) LD50: >4500 mg/kg[1]	
	Oral (rat) LD50: >5000 mg/kg[1]	
carbon black	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3000 mg/kg[2]	Not Available
	Oral (rat) LD50: >8000 mg/kg[1]	
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

834ATH-Part B ATH Flame Retardant Epoxy	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. No significant acute toxicological data identified in literature search. For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides) The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented. Some typical applications of FND Amides are: masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers. The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing

Continued...

gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health.
The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals
The Fatty nitrogen-derived amides (FND amides) comprise four categories:

- Subcategory I: Substituted Amides
- Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components)
- Subcategory III: Imidazole Derivatives
- Subcategory IV: FND Amphoterics

Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies
Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II.
Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low
order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories.
Genetic Toxicity *in vitro*: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the *Salmonella* reverse mutation assay exist for all of the subcategories.
Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II.
In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (*in vitro* bacterial and mammalian cells as well as *in vivo* studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.
Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds.
Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines.
In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
The primary health concerns revolve around the potential of polybrominated fire retardants (PBFRs) to act as carcinogens, endocrine disruptors and neurodevelopmental toxicants based on data for some members of this class of chemicals. In addition, their structural similarities to the polychlorinated diphenyl ethers (PCDEs), nitrofen and polychlorinated biphenyls (PCBs) lends further support to concerns for health effects exerted by these chemicals.
Three PBFRs, the penta-, octa- and decabromodipheyl ethers (BDPE)s, have been and remain of significant commercial interest.
Nonetheless, the field of PBFRs is expanding and a diverse range of these chemicals are now available . Emphasis on the health effects of PBFRs is directed to certain chemical compounds within this class, namely decabromodipehyl ether (DBDPE), pentabromodipheyl ether (PeBDPE), octabromodiphemyl ether (OBDPE) and hexabromocyclododecane (HBCD). Also discussed are the polybrominated biphenyls (PCBs) and tris(2,3-dibromopropylphosphate (TDBPP), though no longer used, due to their significant adverse health effects.
The PBFRs are a structurally diverse group of chemical compounds, some of which share similarities in chemical structure while others vary significantly. Pharmacokinetic studies are limited for most of the chemicals . However, the available information indicates that some brominated flame retardants such as tetrabromodiphenyl ether (TBDPE), HBCD, TDBPP and PBBs are readily absorbed via the gastrointestinal tract. Data available for the polybrominated diphenyl ethers (PBDPE)s and PBBs indicate that the degree of gastrointestinal absorption is inversely proportional to the level of bromination. Dermal absorption has also been reported for TDBPP.
They are generally of low acute toxicity with no or slight and transient irritation to the skin and eyes of experimental animals. Inhalation studies in animals revealed that exposure to PBDPEs caused transient respiratory difficulties.
Like the PBDPEs, tetrabromobisphenol A (TBBPA) and its derivatives have low acute and repeated dose toxicity. They are neither skin or eye irritants nor skin sensitisers in experimental animals. Reversible respiratory effects were reported following inhalation exposure.
With a few exceptions, mutagenicity studies indicate that the majority of the PBRs are neither mutagenic to microbial or eukaryotic organisms nor genotoxic in experimental *in vivo* and *in vitro* systems. TBDPE and HBCD caused an increase in the recombination frequency in some cell lines.
Of the commercially and commonly used PBFRs, penta- and tetra-bromodiphenyl ethers appear to be of greatest significance where health effects are concerned.
Evidence indicates that the liver, and possibly the thyroid, are the organs most sensitive to these chemicals. According to available data, they are endocrine disruptors and neurodevelopmental toxicants in experimental animals. Whether neurodevelopmental effects are a consequence of changes in thyroid hormone levels or are caused by direct neurotoxicity remain to be elucidated. The absence of clinical, physiological and biochemical correlates precludes any conclusions as to the nature of the mechanisms involved. PeBDPE has been classified as a hazardous chemical, Harmful- Danger of Serious Damage to Health by Prolonged Exposure in Contact with Skin and if Swallowed. A similar toxicity profile is apparent for TBDPE. OBDPE is another chemical of concern due to its adverse effects on reproduction in experimental animals.
The two other groups with significant adverse health effects are TDBPP and PBBs. Although both have relatively low acute toxicity in experimental animals, evidence for carcinogenicity, endocrine disruption and reproductive effects exists. Little human data is available, however, epidemiological reports and follow up studies indicate that PBDPE, TDBPP and PBBs are absorbed and can be detected in the serum, adipose tissue and breast milk of directly and/or indirectly exposed individuals. The available evidence indicates that, in some countries, levels of these chemicals are increasing in animal and human tissues (including breast milk), which suggests they are bioaccumulative and persistent. Thyroid effects appear to be the major adverse health effect, with hypothyroidism seen in animals (e.g. OBDPE and PeBDPE, HBCD and PBB) and humans (e.g. DBDPE and deca-BB), although some PBFRs (e.g. DBDPE, TDBPP, HBCD and PBB) elicit carcinogenic effects in animal studies.
Blooming potential: Blooming is defined as the migration (or more appropriately, diffusion) of an ingredient (e.g., plasticiser or flame retardant) in rubber or plastic material to the outer surface after curing. It is sometimes incorrectly referred to as "leaching" or "degassing". Diffusion is generally considered to be a slow process. Blooming has been identified as a source of potential exposure (human and environmental) to PBFRs, particularly for low molecular weight additive PBFRs.
It is generally accepted that "reactive", PBFRs such as TBBPA (and derivatives) and esters of acrylic (propenoic) acid, which are directly incorporated into polymers (e.g., polyester or epoxy resins) via chemical reaction (i.e., covalent binding) have a low or negligible blooming potential, although such chemicals can also be used as non-reactive (i.e., additive) ingredients.

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So-called "additive" PBFRs (e.g., PBDPEs, PBBs, HBCD) are more likely to be subject to blooming, as these compounds are not chemically bound to the polymer backbone. Additive PBFRs reside within the polymer matrix as discrete molecules, but may be subject to weak Van der Waals and electrostatic interaction both between PBFR molecules and with the polymer backbone. High molecular weight polymeric additive flame retardants such as brominated polystyrene are more likely to remain within the matrix due to the slow rate of diffusion. Other PBFRs may undergo both reactive and/or additive reactions with polymer matrices e.g., tetrabromophthalic anhydride and brominated polystyrenes. Increased temperature is also associated with an increase in the rate of PBFR migration. Release of PBFRs or degradation products may occur at high temperatures during thermal processing or recycling e.g. PBDPEs emissions have been reported during thermal recycling activities.
For alkyl polyamines:
The alkyl polyamines cluster consists of organic compounds containing two terminal primary amine groups and at least one secondary amine group.Typically these substances are derivatives of ethylenediamine, propylenediamine or hexanediamine. The molecular weight range for the entire cluster is relatively narrow, ranging from 103 to 232
Acute toxicity of the alkyl polyamines cluster is low to moderate via oral exposure and a moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin sensitisers in experimental animals. Repeated exposure in rats via the oral route indicates a range of toxicity from low to high hazard. Most cluster members gave positive results in tests for potential genotoxicity.
Limited carcinogenicity studies on several members of the cluster showed no evidence of carcinogenicity. Unlike aromatic amines, aliphatic amines are not expected to be potential carcinogens because they are not expected to undergo metabolic activation, nor would activated intermediates be stable enough to reach target macromolecules.
Polyamines potentiate NMDA induced whole-cell currents in cultured striatal neurons

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.
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
Triethylenetetramine (TETA) is a severe irritant to skin and eyes and induces skin sensitisation.
TETA is of moderate acute toxicity: LD50(oral, rat) > 2000 mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure to saturated vapour via inhalation was tolerated without impairment. Exposure to to aerosol leads to reversible irritations of the mucous membranes in the respiratory tract.
Following repeated oral dosing via drinking water only in mice but not in rats at concentration of 3000 ppm there were signs of impairment. The NOAEL is 600 ppm [92 mg/kg bw (oral, 90 days)]. Lifelong dermal application to mice (1.2 mg/mouse) did not result in tumour formation.
There are differing results of the genetic toxicity for TETA. The positive results of the in vitro tests may be the result of a direct genetic action as well as a result of an interference with essential metal ions. Due to this uncertainty of the in vitro tests, the genetic toxicity of TETA has to be assessed on the basis of in vivo tests.
The in vivo micronucleus tests (i.p. and oral) and the SLRL test showed negative results.
There are no human data on reproductive toxicity (fertility assessment). The analogue diethylenetriamine had no effects on reproduction. TETA shows developmental toxicity in animal studies if the chelating property of the substance is effective. The NOEL is 830 mg/kg bw (oral).

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	Experience with female patients suffering from Wilson´s disease demonstrated that no miscarriages and no foetal abnormalities occur during treatment with TETA.. In rats, there are several studies concerning developmental toxicity. The oral treatment of rats with 75, 375 and 750 mg/kg resulted in no effects on dams and fetuses, except slight increased fetal body weight After oral treatment of rats with 830 or 1670 mg/kg bw only in the highest dose group increased foetal abnormalities in 27/44 fetus (69,2 %) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significant the fetal abnormalities of the highest dose group to 3/51 (6,5 % foetus. These findings suggest that the developmental toxicity is produced as a secondary consequence of the chelating properties of TETA.
C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES	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. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides) The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented. Some typical applications of FND Amides are: masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers. The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health. The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals The Fatty nitrogen-derived amides (FND amides) comprise four categories: · Subcategory I: Substituted Amides · Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components) · Subcategory III: Imidazole Derivatives · Subcategory IV: FND Amphoterics **Acute Toxicity:** The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies **Repeated Dose and Reproductive Toxicity:** Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II. Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories. **Genetic Toxicity *in vitro*:** Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the *Salmonella* reverse mutation assay exist for all of the subcategories. **Developmental Toxicity:** A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II. In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (*in vitro* bacterial and mammalian cells as well as *in vivo* studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole. **[Valspar]
DECABROMODIPHENYLETHANE	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. The primary health concerns revolve around the potential of polybrominated fire retardants (PBFRs) to act as carcinogens, endocrine disruptors and neurodevelopmental toxicants based on data for some members of this class of chemicals. In addition, their structural similarities to the polychlorinated diphenyl ethers (PCDEs), nitrofen and polychlorinated biphenyls (PCBs) lends further support to concerns for health effects exerted by these chemicals. Three PBFRs, the penta-, octa- and decabromodipheyl ethers (BDPE)s, have been and remain of significant commercial interest. Nonetheless, the field of PBFRs is expanding and a diverse range of these chemicals are now available . Emphasis on the health effects of PBFRs is directed to certain chemical compounds within this class, namely decabromodipehyl ether (DBDPE), pentabromodipheyl ether (PeBDPE), octabromodiphemyl ether (OBDPE) and hexabromocyclododecane (HBCD). Also discussed are the polybrominated biphenyls (PCBs) and tris(2,3-dibromopropylphosphate (TDBPP), though no longer used, due to their significant adverse health effects. The PBFRs are a structurally diverse group of chemical compounds, some of which share similarities in chemical structure while others vary significantly. Pharmacokinetic studies are limited for most of the chemicals . However, the available information indicates that some brominated flame retardants such as tetrabromodiphenyl ether (TBDPE), HBCD, TDBPP and PBBs are readily absorbed via the gastrointestinal tract. Data available for the polybrominated diphenyl ethers (PBDPE)s and PBBs indicate that the degree of gastrointestinal absorption is inversely proportional to the level of bromination. Dermal absorption has also been reported for TDBPP. They are generally of low acute toxicity with no or slight and transient irritation to the skin and eyes of experimental animals. Inhalation studies in animals revealed that exposure to PBDPEs caused transient respiratory difficulties.

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	Like the PBDPEs, tetrabromobisphenol A (TBBPA) and its derivatives have low acute and repeated dose toxicity. They are neither skin or eye irritants nor skin sensitisers in experimental animals. Reversible respiratory effects were reported following inhalation exposure. With a few exceptions, mutagenicity studies indicate that the majority of the PBRs are neither mutagenic to microbial or eukaryotic organisms nor genotoxic in experimental *in vivo* and *in vitro* systems. TBDPE and HBCD caused an increase in the recombination frequency in some cell lines. Of the commercially and commonly used PBFRs, penta- and tetra-bromodiphenyl ethers appear to be of greatest significance where health effects are concerned. Evidence indicates that the liver, and possibly the thyroid, are the organs most sensitive to these chemicals. According to available data, they are endocrine disruptors and neurodevelopmental toxicants in experimental animals. Whether neurodevelopmental effects are a consequence of changes in thyroid hormone levels or are caused by direct neurotoxicity remain to be elucidated. The absence of clinical, physiological and biochemical correlates precludes any conclusions as to the nature of the mechanisms involved. PeBDPE has been classified as a hazardous chemical, Harmful- Danger of Serious Damage to Health by Prolonged Exposure in Contact with Skin and if Swallowed. A similar toxicity profile is apparent for TBDPE. OBDPE is another chemical of concern due to its adverse effects on reproduction in experimental animals. The two other groups with significant adverse health effects are TDBPP and PBBs. Although both have relatively low acute toxicity in experimental animals, evidence for carcinogenicity, endocrine disruption and reproductive effects exists. Little human data is available, however, epidemiological reports and follow up studies indicate that PBDPE, TDBPP and PBBs are absorbed and can be detected in the serum, adipose tissue and breast milk of directly and/or indirectly exposed individuals. The available evidence indicates that, in some countries, levels of these chemicals are increasing in animal and human tissues (including breast milk), which suggests they are bioaccumulative and persistent. Thyroid effects appear to be the major adverse health effect, with hypothyroidism seen in animals (e.g. OBDPE and PeBDPE, HBCD and PBB) and humans (e.g. DBDPE and deca-BB), although some PBFRs (e.g. DBDPE, TDBPP, HBCD and PBB) elicit carcinogenic effects in animal studies. **Blooming potential:** Blooming is defined as the migration (or more appropriately, diffusion) of an ingredient (e.g., plasticiser or flame retardant) in rubber or plastic material to the outer surface after curing. It is sometimes incorrectly referred to as "leaching" or "degassing". Diffusion is generally considered to be a slow process. Blooming has been identified as a source of potential exposure (human and environmental) to PBFRs, particularly for low molecular weight additive PBFRs. It is generally accepted that "reactive", PBFRs such as TBBPA (and derivatives) and esters of acrylic (propenoic) acid, which are directly incorporated into polymers (e.g., polyester or epoxy resins) via chemical reaction (i.e., covalent binding) have a low or negligible blooming potential, although such chemicals can also be used as non-reactive (i.e., additive) ingredients. So-called "additive" PBFRs (e.g., PBDPEs, PBBs, HBCD) are more likely to be subject to blooming, as these compounds are not chemically bound to the polymer backbone. Additive PBFRs reside within the polymer matrix as discrete molecules, but may be subject to weak Van der Waals and electrostatic interaction both between PBFR molecules and with the polymer backbone. High molecular weight polymeric additive flame retardants such as brominated polystyrene are more likely to remain within the matrix due to the slow rate of diffusion. Other PBFRs may undergo both reactive and/or additive reactions with polymer matrices e.g., tetrabromophthalic anhydride and brominated polystyrenes. Increased temperature is also associated with an increase in the rate of PBFR migration. Release of PBFRs or degradation products may occur at high temperatures during thermal processing or recycling e.g. PBDPEs emissions have been reported during thermal recycling activities.
TRIETHYLENETETRAMINE	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. Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds. Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines. In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper 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. For alkyl polyamines: The alkyl polyamines cluster consists of organic compounds containing two terminal primary amine groups and at least one secondary amine group.Typically these substances are derivatives of ethylenediamine, propylenediamine or hexanediamine. The molecular weight range for the entire cluster is relatively narrow, ranging from 103 to 232 Acute toxicity of the alkyl polyamines cluster is low to moderate via oral exposure and a moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin sensitisers in experimental animals. Repeated exposure in rats via the oral route indicates a range of toxicity from low to high hazard. Most cluster members gave positive results in tests for potential genotoxicity. Limited carcinogenicity studies on several members of the cluster showed no evidence of carcinogenicity. Unlike aromatic amines, aliphatic amines are not expected to be potential carcinogens because they are not expected to undergo metabolic activation, nor would activated intermediates be stable enough to reach target macromolecules. Polyamines potentiate NMDA induced whole-cell currents in cultured striatal neurons 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. Triethylenetetramine (TETA) is a severe irritant to skin and eyes and induces skin sensitisation. TETA is of moderate acute toxicity: LD50(oral, rat) > 2000 mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure to saturated vapour via inhalation was tolerated without impairment. Exposure to to aerosol leads to reversible irritations of the mucous membranes in the respiratory tract. Following repeated oral dosing via drinking water only in mice but not in rats at concentration of 3000 ppm there were signs of impairment. The NOAEL is 600 ppm [92 mg/kg bw (oral, 90 days)]. Lifelong dermal application to mice (1.2 mg/mouse) did not result in tumour formation. There are differing results of the genetic toxicity for TETA. The positive results of the in vitro tests may be the result of a direct genetic action as well as a result of an interference with essential metal ions. Due to this uncertainty of the in vitro tests, the genetic toxicity of TETA has to be assessed on the

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	basis of in vivo tests. The in vivo micronucleus tests (i.p. and oral) and the SLRL test showed negative results. There are no human data on reproductive toxicity (fertility assessment). The analogue diethylenetriamine had no effects on reproduction. TETA shows developmental toxicity in animal studies if the chelating property of the substance is effective. The NOEL is 830 mg/kg bw (oral). Experience with female patients suffering from Wilson´s disease demonstrated that no miscarriages and no foetal abnormalities occur during treatment with TETA.. In rats, there are several studies concerning developmental toxicity. The oral treatment of rats with 75, 375 and 750 mg/kg resulted in no effects on dams and fetuses, except slight increased fetal body weight After oral treatment of rats with 830 or 1670 mg/kg bw only in the highest dose group increased foetal abnormalities in 27/44 fetus (69,2 %) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significant the fetal abnormalities of the highest dose group to 3/51 (6,5 % foetus. These findings suggest that the developmental toxicity is produced as a secondary consequence of the chelating properties of TETA. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).
ANTIMONY TRIOXIDE	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. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. (intermittent) [CCINFO] Reproductive effector
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I]

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NAPHTHA PETROLEUM, HEAVY ALKYLATE	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. **for petroleum:** This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic. This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents **Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans. **Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays. **Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed. **Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.
STODDARD SOLVENT	**for petroleum:** This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic. This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents **Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans. **Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays. **Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed. **Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.
CARBON BLACK	No significant acute toxicological data identified in literature search. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported
ALUMINIUM HYDROXIDE & ZINC BORATE HYDRATE	No significant acute toxicological data identified in literature search.

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

Legend: ✘ *– Data available but does not fill the criteria for classification*
✔ *– Data required to make classification available*
⊘ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

Continued...

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
C18 fatty acid dimers/ tetraethylenepentamine polyamides	LC50	96	Fish	7.07mg/L	2
C18 fatty acid dimers/ tetraethylenepentamine polyamides	EC50	24	Crustacea	7.07mg/L	2
C18 fatty acid dimers/ tetraethylenepentamine polyamides	EC50	48	Crustacea	5.18mg/L	2
C18 fatty acid dimers/ tetraethylenepentamine polyamides	EC50	72	Algae or other aquatic plants	4.11mg/L	2
C18 fatty acid dimers/ tetraethylenepentamine polyamides	NOEC	72	Algae or other aquatic plants	1.25mg/L	2
aluminium hydroxide	LC50	96	Fish	0.2262mg/L	2
aluminium hydroxide	EC50	168	Crustacea	0.0076mg/L	2
aluminium hydroxide	EC50	48	Crustacea	0.7364mg/L	2
aluminium hydroxide	EC50	96	Algae or other aquatic plants	0.0054mg/L	2
aluminium hydroxide	NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2
decabromodiphenylethane	EC50	48	Crustacea	0.019mg/L	2
triethylenetetramine	EC50	48	Crustacea	31.1mg/L	1
triethylenetetramine	EC10	72	Algae or other aquatic plants	0.67mg/L	1
triethylenetetramine	EC50	72	Algae or other aquatic plants	2.5mg/L	1
triethylenetetramine	NOEC	72	Algae or other aquatic plants	<2.5mg/L	1
triethylenetetramine	LC50	96	Fish	180mg/L	1
antimony trioxide	LC50	96	Fish	0.93mg/L	2
antimony trioxide	NOEC	720	Fish	>0.0075mg/L	2
antimony trioxide	EC50	48	Crustacea	1mg/L	2
antimony trioxide	EC50	96	Crustacea	0.5mg/L	2
antimony trioxide	EC50	96	Algae or other aquatic plants	>=0.00073- <=0.00076mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	96	Algae or other aquatic plants	9.337mg/L	3
propylene glycol monomethyl ether acetate, alpha-isomer	LC50	96	Fish	100mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	NOEC	336	Fish	47.5mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	48	Crustacea	373mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	504	Crustacea	>100mg/L	2
naphtha petroleum, heavy alkylate	EC50	72	Algae or other aquatic plants	=13mg/L	1
naphtha petroleum, heavy alkylate	EC50	72	Algae or other aquatic plants	=30000mg/L	1
naphtha petroleum, heavy alkylate	NOEC	72	Algae or other aquatic plants	=0.1mg/L	1
Stoddard Solvent	LC50	96	Fish	2.2mg/L	4
Stoddard Solvent	NOEC	3072	Fish	=1mg/L	1
Stoddard Solvent	EC50	96	Algae or other aquatic plants	64mg/L	2
carbon black	LC50	96	Fish	>100mg/L	2
carbon black	NOEC	720	Fish	17mg/L	2
carbon black	EC50	48	Crustacea	>100mg/L	2
carbon black	EC50	384	Crustacea	4.9mg/L	2
carbon black	EC50	96	Algae or other aquatic plants	95mg/L	2
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - 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*				

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

Continued...

For Fatty Nitrogen-Derived Amides (FND Amides)

Environmental fate:

As expected for molecules of this size, model predictions for the chemicals with definable structures indicate they are nonvolatile. Predicted or measured Kow values are of limited practical use for the FND Amides. An inherent property of surfactants is that they tend to accumulate at the interface between hydrophobic and hydrophilic phases rather than equilibrate between the two phases. Therefore, the accurate measurement of the Kow of any surfactant is notoriously difficult. The measured values for water solubility of the FND Amides indicate that they are insoluble. The model predictions, however, range from insoluble to moderately soluble. The physical/chemical properties of surfactants often make water solubility data of little practical value in the determination of environmental fate and effects.

Due to the low volatility of the FND Amides atmospheric photodegradation estimates are of no practical use. However, photodegradation was predicted that could be modeled. These predictions indicate that these chemicals would be expected to degrade relatively rapidly upon exposure to light (t1/2 values ranging from approximately 2.2 to 9.5 hours).

Due to the surfactant properties and solubility of the FND Amides, hydrolytic stability is of minimal value for determining
environmental fate or effects.

Biodegradability: There are adequate measured data across Subcategories I, II and IV to allow the conclusion that the these chemicals are readily or inherently biodegradable. Further, the model predictions provide reasonably close estimates to these measured values. Minimal degradability of the one chemical,[CAS RN 68122-86-1], from Subcategory III indicates these chemicals are slowly degraded. The slower degradation of these materials is likely the result of limited water solubility and behavior of the chemicals in aqueous solution. Longer single alkyl group substitutions and/or multiple long-chain alkyl substituents result in slower “inherent” biodegradability.

Ecotoxicity:

The reliable data for acute toxicity to fish and daphnid indicate that the FND Amides like surfactants in general, may adversely affect aquatic organisms (LC50 and EC50 values ranging from 0.2 to 59 mg/l). Many of the ECOSAR model estimates for the acute toxicity endpoints indicate the chemicals are “not toxic at solubility”. However, for surfactants such as the FND Amides the acute aquatic toxicity generally is considered to be related to the effects of the surfactant properties on the organism and not to direct chemical toxicity.

For polybrominated fire retardants (PBFRs):

Environmental Fate: Overall, it has been determined that varying the physico-chemical properties of logKow, water solubility and vapour pressure over a wide range had little effect on distribution to the aquatic and terrestrial compartments. However, a much larger effect was noticed in the atmospheric compartment. Release to the environment will be slow and diffuse over the life of products containing PBFRs. Where released to the environment, some PBFRs are expected to be stable, both microbially and abiotically. When released to land, they should bind strongly to the organic component of soils and be immobile. In the event of release to water, movement from the water column is likely to be rapid with the compounds partitioning to sediments and biota, where bioaccumulation is expected from the commercial pentabromo diphenyl ether compounds (tetra- to hexa-). Bioaccumulation is not anticipated with octabromodiphenyl ethers (OBDPE) and decabromodiphenyl ether (DBDPE) while hexabromocyclododecane (HBCD) has the potential to bioaccumulate. A relatively high bioconcentration factor for tetrabromobisphenol A (TBBPA) is balanced by rapid excretion and the compound has not been found in environmental biological samples. Components of commercial pentabromodiphenyl ether (PeBDPE) may volatilise to the atmosphere from water. It is speculated that they may bind to atmospheric particles with the potential to undergo long-range atmospheric transport.

Atmospheric Fate: The polybrominated diphenyl ethers (PBDPE) commercial products have low vapour pressures, that decrease with increasing bromination. They may be considered slightly to very slightly volatile. Accordingly, PBDPE compounds with a higher level of bromination, when released to land, are more likely to bind to soils than volatilise. Whether other highly brominated flame retardants of low vapour pressure have the same fate, cannot be determined due to the lack of sufficient data. However, this may not be the case where release is to water as the substances have very low water solubilities.

PeBDPE has a rate constant of 1.27 x 10-12 cm3/molecule.sec for reaction with atmospheric hydroxyl radicals. Using the accepted global average atmospheric concentration of hydroxyl radicals as 5 x 105 molecules/cm3, an atmospheric half-life of around 12.6 days can be estimated. This is of sufficient time for long-range atmospheric transport to occur. PeBDPE has been identified in air samples from Swedish background sites on the island of Gotland and in the Scandinavian mountain range. Similarly, HBCD levels up to 5.7 pg/m3 were measured in Sweden during 1990 and 1991 at locations far from known point sources.

Air concentrations of tri- and hexa-BDPE in the range of 7.1 to 53 pg/m3 near metal recycling plants in Taiwan and Japan, and of tetra- and penta-BDPE (combined) in the range 1 to 8 pg/m3 and HBCD of 5.3 to 6.1 pg/m3 in Swedish air samples have been reported.

Aquatic fate: Reports of PBDPEs detected in fish in the northern hemisphere show that aquatic exposure does occur, which may be attributed to local industries. For example, in Virginia, USA, muscle tissue of several fish species contained PBDPEs. While the sample area was not heavily industrialised, it is home to considerable furniture manufacturing activities. In Sweden, fish with detectable PBDPEs and HBCD concentrations were caught downstream of textile industries and sewage treatment plants.

There is little information available on the abiotic degradation of PBDPEs in aqueous solutions. Ethers are not likely to hydrolyse readily in the normal environmental pH range. PeBDPE is reported to be hydrolytically stable under conditions found in the environment.

It is apparent from measured or computed values of the Henry's Law Constant that most of the PBDPEs and HBCD can be considered moderately volatile. The highly brominated compounds may be expected to be less volatile, with the three most highly brominated substances being considered only very slightly volatile from water. This is supported by the long half-lives of volatilisation (from rivers and lakes) for DBDPE, HBCD and TBBPA. This suggests that where release occurs to water and the compounds do not fully partition to sediments or biota, the PBFRs, particularly those with relatively low levels of bromination, may volatilise to the atmosphere and thereby be available for atmospheric transport.

Terrestrial fate: When released to soils, PBFRs may generally be considered to bind strongly and be immobile. Leaching from soil is unlikely to occur. The results show that sorption tendencies increase as the level of bromination increases. This indicates that mobility, albeit very limited, is likely to be greater with the lower brominated compounds.

Degradation: Reductive dehalogenation of PBDPE occurs under some conditions. Although environmental breakdown to lower congeners is also a possibility, no anaerobic biodegradation of DBDPE was seen in sediment for up to 2 years. TBBPA has been shown to partly degrade under both aerobic and anaerobic conditions in a range of soil types and in sediment water. After 64 days approximately 35 to 80% of TBBPA remained in soil under aerobic conditions, with 40 to 90% remaining under anaerobic conditions, with the highest levels measured in sandy loam and lowest in silty loam. A sequential anaerobic/aerobic soil study demonstrated complete degradation of TBBPA after 45 days to the non-brominated bisphenol A, which was resistant to further degradation. The phenolic groups of TBBPA may be methylated in the environment and the resulting metabolite is potentially more lipophilic. This compound has been found in sediment, fish and shellfish. HBCD has been tested and found not ready biodegradable. The biodegradation of HBCD was examined after exposure of samples to bacterial medium for 5, 7 and 15 days. Some biodegradation was indicated.

Bioaccumulation: The bioaccumulation of a commercial PeBDPE product containing TBDPE, PeBDPE (2 isomers) and HBDPE (2 isomers) was studied in carp. An overall log BCF of 4.16 was estimated. The BCF for 2-propenoic acid (pentabromophenyl) methyl ester in carp was measured to be a maximum of 12 at any level (0.2 ppm, 2 ppm) over an 8-week period.

Studies indicate that as bromination levels increase beyond HBDPE, PBDPEs show a decreasing tendency for bioaccumulation . Tetra- and penta-BDPEs, in particular, have a high potential for bioaccumulation. Monitoring data from the Baltic and elsewhere suggest the presence of high concentrations of these compounds higher up in the food chain. Where OBDPE and DBDPE are concerned, no significant bioaccumulation has been demonstrated in fish and BCF varied between about 5 and less than 50. This is due to low uptake. OBDPE and DBDPE are larger molecules and, consequently, are less readily absorbed than PeBDPE.

Studies with aquatic invertebrates and vertebrates with TBBPA indicate bioconcentration factors (BCF) ranging from 20 up to 3200 depending on the test conditions and organisms. Although the BCFs are high, studies indicate that in some species TBBPA is rapidly excreted. Methylated TBBPA have been detected in 2/19 samples of fish and shellfish in Japan.

Environmental effects: While there is a distinct lack of data for avian toxicity, biomagnification in fish eating birds may occur particularly for the tetra- and penta-BDPE, which have been detected in fish as well as in fish eating birds. Due to the very low solubility of the highly used PBDPEs, namely penta-and deca-BDPE, toxicity to aquatic organisms is difficult to determine. Acute toxicity in fish up to the limit of solubility has not been observed. Some chronic effects may occur, but these appear to be limited.

Aquatic invertebrates and algae appear susceptible to PBDPEs based on the limited data available, and PBDPEs may be considered highly toxic to these organisms.

There is only one acute effect available for daphnia following exposure to commercial PeBDPE and evidence suggests the effect may have been physical rather than toxic, so conclusions are uncertain. Based on two test results, TBBPA can be described as moderately to highly toxic to aquatic invertebrates.

Sediment testing conducted as a result of the initial risk assessment conducted on PeBDPE in the EU demonstrated a lack of toxicity to three sediment dwelling organisms. No adverse biological effects resulted from the increased TBBPA body burden in a single sediment organism study.

Based on the commercial PeBDPE, PBDPEs are not toxic to soil micro-organisms, earthworms or plants.

Physical properties: PBDPEs are stable compounds with high boiling points ranging between 310 C and 425 C and low vapour pressures in the range 6.5 x 10-6 to 4.5 x 10-5 Pa at 20 to 25 C. They exhibit poor solubility in water (0.0001 to 0.01 mg/L) and in most organic solvents, with *n*-octanol/water partition coefficients (log Pow) between 4 and 10. No formal fat solubility studies were available for assessment, but pharmacokinetic studies indicate significant differences between congeners e.g., <1% DBDPE was identified in fatty tissue, whereas the majority of TBDPE was retained in adipose tissue.

The chemical stability of the polybromobiphenyls (PBBs) is dependent, in part, on the degree of bromination and the specific substitution patterns. In general, the highly brominated PBBs are more rapidly degraded by UV radiation. Their solubility in water is low and decreases with increasing bromination. Melting points, where determined, range from a low of 72 C to a high of 380 C. Like the PBDPEs, the PBBs have low vapour pressures.

Thermal degradation: Considerable laboratory experimentation has gone into the investigation of the thermal degradation, pyrolysis and combustion products of PBFRs, mainly because of concern that polybromodibenzo-dioxins (PBDD) and -furans (PBDF) might be formed. Close analogies have been drawn with the formation under similar conditions of polychlorodibenzo- dioxins and -furans from organochlorine substances and with the toxicity of these derived "dioxins". Neither the commercial flame retardant DBDPE, nor plastic materials incorporating it, contain measurable amounts of the highly toxic polybromodibenzo-dioxin and -furan contaminants. Partial combustion of the material containing the flame retardant (and usually also antimony trioxide) produced polybromodibenzo-dioxins and -furans, but these were mainly heavily brominated and congeners with the substitution pattern of most concern - 2,3,7,8 -were minor components of the congener mixture. Analysis of 2-propenoic acid (pentabromophenyl) methyl ester; tris (tribromoneopentyl) phosphate and TBBPA bis (2,3-dibromopropyl) ether for contamination of polybrominated p-dibenzodioxins and dibenzofurans indicated that the PBDD/PBDF levels were below the level of quantification specified by US EPA Toxic Substances Control Act (TSCA) 40 CFR 766.27.

Yields of mixed polybromodibenzofuran congeners as high as 90% could be realised by the pyrolysis of neat bromodiphenyl ethers, and lesser yields when the flame retardants were incorporated

Continued...

into polystyrene or polyethylene. Gas-phase pyrolysis of a number of PBFRs, including polybrominated diphenyl ethers, has demonstrated the formation of bromobenzenes, bromophenols and dioxins and furans at intermediate temperatures. However, these were destroyed when the thermal degradation reactions were carried out at 800 C. Similar experiments with decabromodiphenyl and tetrabromobisphenol A showed the presence in the pyrolysates of polybrominated dibenzo-dioxins and -furans, the latter in greater amounts. Only small proportions of these products had the 2,3,7,8-tetrasubstitution pattern, which is associated with the greatest toxicity. Formation of the dioxins and furans was greatest at 600 C. Similar results were reported for thermal degradations conducted in a device, which simulated the operation of a municipal waste incinerator.
For ethyleneamines:
Adsorption of the ethyleneamines correlates closely with both the cation exchange capacity (CEC) and organic content of the soil. Soils with increased CEC and organic content exhibited higher affinities for these amines. This dependence of adsorption on CEC and organic content is most likely due to the strong electrostatic interaction between the positively charged amine and the negatively charged soil surface.
Drinking Water Standards: hydrocarbon total: 10 ug/l (UK max.).
For alkyl polyamines:
All members of this cluster are miscible or soluble in water. The estimated value of log Kows-range from 3.67 to 1.8 is consistent with the available experimental water solubilities. Vapour pressures range from 1.1x 10-6 hPa to 0.31 hPa. Estimated and experimental pKbs are in a relatively narrow range of 9.68 to 10.7.

Environmental fate:

Members of this cluster are expected to have varying degrees of mobility in the soil. Low vapor pressure and Henry's Law Constants suggest that these compounds are not expected to be in the vapor phase. Modeling suggests that all members of this cluster are likely to react rapidly with photochemically produced hydroxyl radials with half-lives on the order of an hour, but with little material in the vapor phase, it is not expected to be a predominant removal pathway for these chemicals. Experimental data and results from estimation models indicate that all members of this cluster have the potential to biodegrade aerobically under environmental conditions. Fugacity models indicate that the members of this cluster are likely to partition predominately to soil and water. All chemicals in this cluster are expected to have low environmental persistence. Measured and estimated bioconcentration factors for members of this cluster indicate a low potential for bioaccumulation.

Ecotoxicity:

Evaluation of the available experimental and estimated aquatic toxicity data indicate acute toxicity to fish is low. Daphnia aquatic toxicity is generally low. Algae appear to be the most sensitive organism with several members of the cluster having measured or estimated toxicity values indicative of moderate toxicity. Chronic toxicity for all cluster members is estimated; it is generally low for fish and algae, but high for daphnia.
Antimony exists in the atmosphere in low concentrations. Urban air contains 0.05 to 0.06 ppm of antimony. There are very low concentrations in water due to minimal solubility. Volatilisation from water is not likely. The soil usually contains 0.1 to 10 mg/kg dry weight. Antimony concentrations in freshwater fish are low, approximately 3 mg/kg wet weight.
Little is known of the adsorptive behavior of antimony, its compounds, and ions in soils and sediments. The binding of antimony to soil is determined by the nature of the soil and the form of antimony deposited on the soil. Some forms of antimony may bind to inorganic and organic ligands. On the other hand, a mineral form would be unavailable for binding. Some studies suggest that antimony is fairly mobile under diverse environmental conditions, while others suggest that it is strongly adsorbed to soil.Since antimony has an anionic character (e.g. Sb(OH)i), it is expected to have little affinity for organic carbon. It is not expected that cation exchange, which generally dominates adsorption to clay, would be important for anionic antimony. Antimony is known to form coprecipitates with hydrous iron, manganese, and aluminum oxides in soil and sediment. Antimony adsorbs strongly to colloidal material in soil. The partition coefficient of antimony to 0.05-0.003 um colloids was 1,300. Antimony adsorbed to such material can be transported with the colloids in groundwater.
Leaching experiments performed with river sediment samples from a mining district in Idaho indicated that Sb(V) was the major species released during leaching. The fraction of antimony leached from sediment with deionized water after 10 days was highly correlated with the free iron and manganese oxide content of the sediment. The release of antimony from the sediment increased at low pH and increased sharply at high pH. The form of released antimony was also sensitive to pH. At pH 2.7, the bulk of antimony released was as Sb(II1); at pH 4.3, the concentrations of tri- and pentavalent antimony were comparable; and at pH 6.3 and above, Sb(V) was the predominant species.
Antimony does not appear to bioconcentrate appreciably in fish and aquatic organisms. No detectable bioconcentration occurred during a 28-day test in bluegills (EPA 1980). Only low levels of antimony have been reported in fish and aquatic organisms collected off the coast of Africa, Australia, and the Danube River in Austria. Bioconcentration factors for antimony ranged from 0.15 to 390. A study of the distribution of antimony around a smelter site indicated that antimony occurring in plants results from surface deposition. Uptake from soil is minor and appears to be correlated with the amount of available antimony (that which is soluble or easily exchangeable).Antimony bioconcentration was measured in voles, shrews, rabbits, and invertebrates around a smelter. Analysis of antimony in organs of the small mammals, compared with estimates of their antimony intake from food, showed that, although the amount of antimony in the organs was elevated, it was low compared to the amount ingested. The results suggest that antimony does not biomagnify from lower to higher trophic levels in the food chain.
Thermodynamically, most dissolved antimony in natural waters under aerobic conditions should be present in the +5 oxidation state as antimonate species. At 0.001 M total antimony, the dominant species were Sb(OH)6? and Sb(OH)5 0. A small quantity of polymeric hydroxy species were found, but these will be less significant when the total antimony concentration is low, such as in natural water. While industrial inputs will commonly contain antimony in the +3 oxidation state (e.g., antimony trioxide), it is not known how fast antimonite would oxidize to antimonate under natural conditions. Under reducing conditions, trivalent species such as Sb(OH) 3 0, Sb(OH) 4? , and Sb2S4 4- may be significant.
Antimony compounds may undergo photochemical reactions, but these do not appear to be significant in determining their aquatic fate. Antimony trioxide suspensions strongly absorb ultraviolet radiation below 325 nm and darken. The process is reversible, and when the light is removed, the white color slowly returns. The effect is believed to be due to peroxide radical formation on the crystal surface. Both water and oxygen seem to be necessary for the reoxidation of the reduced antimony.
Antimony can be reduced and methylated by microorganisms in the aquatic environment, similar to arsenic, and become mobilized. This reaction is most likely to occur in reducing environments, such as in bed sediment. In the case of arsenic, this reaction may be mediated by fungi and bacteria, but it is not known whether this is the case with antimony. The resulting trimethylstibine is initially oxidized by atmospheric oxygen to a mixture of trimethylstilbine oxide ((CH3)3SbOH) and trimethylstibinic acid ((CH3)2SbO3H), and then to antimony oxides and insoluble polymers. The rate constant is estimated to be of the order of 0.1 to 0.2 L/molsec. Trimethylstibine has a high vapor pressure, 103 mmHg at 25 deg C, and might volatilize before it is completely oxidized. The oxidation product, (CH3)3SbO, is much more soluble than trimethylstibine; therefore, oxidation will reduce volatilization. Oxidation of trimethylstibine in the gas phase is very rapid; the rate is 0.11/mmHg-sec or 2000 L/mol-sec. Trimethylstibine has been shown to react with alkyl iodides and bromides; this results in the formation of quaternary salts. Should antimony occur in a landfill with alkyl halides, the formation of quaternary salts should greatly enhance antimony's mobility.
There is evidence that phytoplankton can reduce Sb(V) to the Sb(III). Sb(III) decreases to very low levels at the base of the seasonal thermocline and remains low down to the sediment where increasing levels are again observed. Sb(III) only accounts for 44% of the inorganic antimony in the anoxic zone, and speciation in this region is unclear. Thermodynamically, the antimony should be in the trivalent state. Thiocomplexes are thought to account for some of the antimony in this zone. Methylated antimony species existed throughout the water column and made up 10% of total antimony. Monomethyl antimony species were more abundant in surface waters and in the anoxic zone. There was no sharp increase in methyl antimony near the sediment, which would be expected if these species were formed biosynthetically. Since the highest antimony concentration is at the surface, it is unlikely that antimony is taken up by phytoplankton, as is the case with arsenic. A decrease in antimony concentration with depth suggests scavenging by particulate matter and, at lower depths, by iron hydroxyoxides.
for triethylenetetramine (TETA):

Environmental fate:

TETA is completely miscible with water forming an alkaline solution (pH 10 at 10 g/l). The technical product has a vapour pressure of ca. 1 Pa at 20 C. The calculated Log Pow (unprotonated form) amounts to ca. -1.4 and indicates a low potential for bioaccumulation. There are no measured Koc -values available. For ethylenediamine (CAS Nr. 107- 15-3) and diethylenetriamine (CAS Nr. 111-40- 0), Koc -values of 4766 and 19111 were measured respectively. The high adsorption is most likely due to electrostatic interaction. A comparable Koc can be expected for TETA, which would suggest a high potential for geoaccumulation.
TETA is not readily biodegradable (0% after 20 days, OECD GL 301 D; same result with adapted inoculum). Also, in a test on inherent biodegradability with industrial sludge,
TETA was not degraded (0 % DOC removal after 28 days, OECD GL 302 B). TETA has therefore to be regarded as non-biodegradable**.** Adsorption onto sewage sludge was not observed. In a test on hydrolysis, TETA was not found to have undergone hydrolysis after 36 days.
Direct photolysis of TETA in the hydrosphere is not to be expected (molar extinction coefficient < 10 l / (mol.cm) at > 240 nm). The half - life due to photooxidative degradation by OH-radicals in the atmosphere is estimated to be 1.7 hours. As TETA does have a low tendency to pass from water to air, this does not represent a significant removal process from the environment.
Based upon the physical-chemical and biodegradation properties of TETA, no elimination in waste water treatment plants is assumed.

Ecotoxicity:

Fish LC50 (96 h): *Poecilia reticulata* 570 mg/l
Other test results with *Leuciscus idus* and *Pimephales promelas*, which could not be validated, are in the same order of magnitude.
Daphnia magna EC50 (48 h): 31.1 - 33.9 mg/l (immobilisation several tests); (21 d) >3.2- <10 mg/l; NOEC 1 mg/l (immobilisation of parental organisms was the most sensitive effect parameter).
Concentrations of 293 - 7313 mg/l had no teratogenic effects on sea-urchin (*Paracen trotus lividus*) eggs. The larvae were most sensitive and showed delay of development at 293 mg/l
Algal *Scenedesmus subspicatus* EBC50 (72 h) 2.5 mg/l; EBC10 0.67 mg/l;EuC50 >= 100 mg/l; EuC10 0.95 mg/l
Effect: growth inhibition (B = biomass; u = growth rate)
Algal *Selenastrum capricornutum* EC50 (72 h) 20 mg/l Effect: growth inhibition (biomass) ; NOEC < 2.5 mg/l; EC50 (96 h) 3,7 mg/l
Microorganisms *Pseudomonas fluorescens* EC0 (24 h): 500 mg/l Effect: growth inhibition (biomass)
Bird acute LD50 (18 h): redwinged blackberry >101 mg/kg
Prevent, by any means available, spillage from entering drains or water courses.
DO NOT discharge into sewer or waterways.

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12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
triethylenetetramine	LOW	LOW
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
triethylenetetramine	LOW (LogKOW = -2.6464)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
Stoddard Solvent	LOW (BCF = 159)

12.4. Mobility in soil

Ingredient	Mobility
triethylenetetramine	LOW (KOC = 309.9)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)

12.5.Results of PBT and vPvB assessment

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

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	▸ 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. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Treat and neutralise at an approved treatment plant. ▸ Treatment should involve: Neutralisation with suitable dilute acid followed by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

	 LIMITED QUANTITY: Part B of 834ATH-375ML, 834ATH-3L kits

Continued...

Marine Pollutant	
HAZCHEM	2X

Land transport (ADR)

14.1.UN number	2735
14.2.Packing group	II
14.3.UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains zinc borate hydrate and triethylenetetramine)
14.4.Environmental hazard	Not Applicable
14.5. Transport hazard class(es)	Class: 8 Subrisk: Not Applicable
14.6. Special precautions for user	Hazard identification (Kemler): 80 Classification code: C7 Hazard Label: 8 Special provisions: 274 Limited quantity: 1 L

Air transport (ICAO-IATA / DGR)

14.1. UN number	2735
14.2. Packing group	II
14.3. UN proper shipping name	Amines, liquid, corrosive, n.o.s. *; Polyamines, liquid, corrosive, n.o.s. * (contains zinc borate hydrate and triethylenetetramine)
14.4. Environmental hazard	Not Applicable
14.5. Transport hazard class(es)	ICAO/IATA Class: 8 ICAO / IATA Subrisk: Not Applicable ERG Code: 8L
14.6. Special precautions for user	Special provisions: A3A803 Cargo Only Packing Instructions: 855 Cargo Only Maximum Qty / Pack: 30 L Passenger and Cargo Packing Instructions: 851 Passenger and Cargo Maximum Qty / Pack: 1 L Passenger and Cargo Limited Quantity Packing Instructions: Y840 Passenger and Cargo Limited Maximum Qty / Pack: 0.5 L

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	2735
14.2. Packing group	II
14.3. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains zinc borate hydrate and triethylenetetramine)
14.4. Environmental hazard	Marine Pollutant
14.5. Transport hazard class(es)	IMDG Class: 8 IMDG Subrisk: Not Applicable
14.6. Special precautions for user	EMS Number: F-A, S-B Special provisions: 274 Limited Quantities: 1 L

Inland waterways transport (ADN)

14.1. UN number	2735
14.2. Packing group	II

Continued...

14.3. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains zinc borate hydrate and triethylenetetramine)
14.4. Environmental hazard	Not Applicable
14.5. Transport hazard class(es)	8 Not Applicable
14.6. Special precautions for user	Classification code: C7 Special provisions: 274 Limited quantity: 1 L Equipment required: PP, EP Fire cones number: 0

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

Source	Ingredient	Pollution Category
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk	triethylenetetramine	Y
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk	propylene glycol monomethyl ether acetate, alpha-isomer	Z
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk	Stoddard Solvent	Y

SECTION 15 REGULATORY INFORMATION

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

C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES(68410-23-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

ALUMINIUM HYDROXIDE(21645-51-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

DECABROMODIPHENYLETHANE(84852-53-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

TRIETHYLENETETRAMINE(112-24-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Customs Inventory of Chemical Substances ECICS (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

ZINC BORATE HYDRATE(138265-88-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

ANTIMONY TRIOXIDE(1309-64-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Customs Inventory of Chemical Substances ECICS (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Carcinogenic Substances
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
UK Workplace Exposure Limits (WELs)

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER(108-65-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Bulgarian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Czech)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Danish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Dutch)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Estonian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Finnish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Maltese)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Polish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Portuguese)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Romanian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovak)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovenian)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Spanish)
European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Swedish)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
UK Workplace Exposure Limits (WELs)

NAPHTHA PETROLEUM, HEAVY ALKYLATE(64741-65-7.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 2) Carcinogens: category 1B (Table 3.1)/category 2 (Table 3.2)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Carcinogenic Substances
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Mutagenic Substances
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

STODDARD SOLVENT(8052-41-3.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 2) Carcinogens: category 1B (Table 3.1)/category 2 (Table 3.2)
European Customs Inventory of Chemical Substances ECICS (English)
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Carcinogenic Substances
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Mutagenic Substances
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)

CARBON BLACK(1333-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

European Customs Inventory of Chemical Substances ECICS (English)
European List of Notified Chemical Substances (ELINCS)
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
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 - : 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

15.2. Chemical safety assessment

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

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
C18 fatty acid dimers/ tetraethylenepentamine polyamides	68410-23-1	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2, Eye Irrit. 2	GHS07, Wng	H315, H319
2	Skin Irrit. 2, Skin Sens. 1A, Eye Dam. 1, Aquatic Chronic 2, Eye Irrit. 2, Skin Sens. 1, Skin Corr. 1C, Aquatic Chronic 1, Aquatic Acute 1, Not Classified	GHS09, GHS05, Dgr, Wng, GHS06	H317, H318, H314, H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
aluminium hydroxide	21645-51-2	Not Available	01-2119529246-39-XXXX

Continued...

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Eye Irrit. 2, Not Classified	GHS07, Wng	H319
2	Eye Irrit. 2, Not Classified, Skin Irrit. 2, STOT SE 3	GHS07, Wng	H319, H315, H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
decabromodiphenylethane	84852-53-9	Not Available	01-2119474877-18-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Aquatic Chronic 4	Not Available	Not Available
2	Not Classified, Aquatic Chronic 4	Not Available	Not Available

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
triethylenetetramine	112-24-3	612-059-00-5	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3	GHS07, GHS05, Dgr	H312, H314, H317
2	Acute Tox. 4, Skin Corr. 1B, Skin Sens. 1, Aquatic Chronic 3, Eye Dam. 1, Acute Tox. 3, Resp. Sens. 1, STOT SE 3, Aquatic Chronic 2, Not Classified	GHS05, Dgr, GHS06, GHS08, GHS09	H314, H317, H318, H302, H311, H334, H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
zinc borate hydrate	138265-88-0	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Aquatic Acute 1, Aquatic Chronic 1	GHS09, Wng	H361
2	Aquatic Acute 1, Aquatic Chronic 1, Repr. 2, Aquatic Chronic 2, Not Classified	GHS09, Wng, GHS08	H361

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
antimony trioxide	1309-64-4	051-005-00-X	01-2119475613-35-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Carc. 2	GHS08, Wng	H351
2	Carc. 2, Repr. 1A, STOT RE 1, Aquatic Chronic 3, Eye Dam. 1, Acute Tox. 4, Aquatic Chronic 2, Eye Irrit. 2, Skin Irrit. 2, STOT RE 2	GHS08, Dgr, Wng, GHS05, GHS09	H360, H372, H317, H318, H332, H302, H350

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6	607-195-00-7	01-2119475791-29-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Flam. Liq. 3, Eye Irrit. 2, Eye Dam. 1, Not Classified, STOT SE 3	GHS02, Wng, GHS03, GHS05, Dgr	H226, H319, H335, H336

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
naphtha petroleum, heavy alkylate	64741-65-7.	649-275-00-4	01-2119850115-46-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Asp. Tox. 1, Muta. 1B, Carc. 1B	GHS08, Dgr	H304, H340, H350
2	Asp. Tox. 1, Muta. 1B, Carc. 1B, Flam. Liq. 3, Aquatic Chronic 4, Aquatic Chronic 2, Not Classified, Acute Tox. 3, STOT SE 3, Flam. Liq. 1, Skin Irrit. 2, Flam. Liq. 2, Repr. 2, Aquatic Chronic 3	GHS08, Dgr, GHS02, GHS09, GHS06, Wng	H304, H340, H350, H331, H336, H224, H315, H361

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
Stoddard Solvent	8052-41-3.	649-422-00-2, 649-330-00-2, 649-345-00-4	01-2119484819-18-XXXX, 01-2119942421-46-XXXX, 01-2119490979-12-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Asp. Tox. 1, Muta. 1B, Carc. 1B	GHS08, Dgr	H304, H340, H350

Continued...

2	Asp. Tox. 1, Flam. Liq. 3, Skin Irrit. 2, STOT SE 3, Aquatic Chronic 2, STOT RE 2, Aquatic Chronic 3, STOT SE 1, Not Classified, Acute Tox. 4, Skin Corr. 1B, Muta. 1B, Carc. 1B, Flam. Liq. 2, Flam. Liq. 1, Repr. 2, STOT RE 1, Aquatic Chronic 1, Eye Irrit. 2, Acute Tox. 3, Carc. 1A, Aquatic Acute 1	GHS08, Dgr, GHS09, GHS02, GHS05, Wng, GHS06	H304, H336, H335, H373, H302, H312, H314, H332, H340, H350, H225, H224, H315, H361, H372, H319, H331, H318

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
carbon black	1333-86-4	Not Available	01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Not Classified	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332
2	Not Classified, Carc. 2, STOT SE 3, Eye Irrit. 2, STOT RE 2, STOT RE 1, Aquatic Chronic 4, Self-heat. 1, Self-heat. 2, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Flam. Sol. 2, Acute Tox. 4	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332
2	Not Classified, Carc. 2, STOT SE 3, Eye Irrit. 2, STOT RE 2, STOT RE 1, Aquatic Chronic 4, Self-heat. 1, Self-heat. 2, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Flam. Sol. 2, Acute Tox. 4	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H335, H319, H372, H251, H315, H228, H370, H332

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

National Inventory	Status
Australia - AICS	N (decabromodiphenylethane)
Canada - DSL	N (zinc borate hydrate; decabromodiphenylethane)
Canada - NDSL	N (propylene glycol monomethyl ether acetate, alpha-isomer; antimony trioxide; zinc borate hydrate; Stoddard Solvent; C18 fatty acid dimers/ tetraethylenepentamine polyamides; naphtha petroleum, heavy alkylate; aluminium hydroxide; carbon black; triethylenetetramine; decabromodiphenylethane)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (zinc borate hydrate; C18 fatty acid dimers/ tetraethylenepentamine polyamides)
Japan - ENCS	N (zinc borate hydrate; C18 fatty acid dimers/ tetraethylenepentamine polyamides; naphtha petroleum, heavy alkylate; decabromodiphenylethane)
Korea - KECI	N (zinc borate hydrate; decabromodiphenylethane)
New Zealand - NZIoC	Y
Philippines - PICCS	N (zinc borate hydrate)
USA - TSCA	N (zinc borate hydrate)
Legend:	*Y = All ingredients are on the inventory* *N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Full text Risk and Hazard codes

H224	Extremely flammable liquid and vapour
H225	Highly flammable liquid and vapour
H226	Flammable liquid and vapour
H228	Flammable solid
H251	Self-heating; may catch fire
H302	Harmful if swallowed
H304	May be fatal if swallowed and enters airways
H311	Toxic in contact with skin
H312	Harmful in contact with skin
H315	Causes skin irritation
H318	Causes serious eye damage
H319	Causes serious eye irritation
H331	Toxic if inhaled
H332	Harmful if inhaled
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled
H335	May cause respiratory irritation
H336	May cause drowsiness or dizziness
H340	May cause genetic defects
H350	May cause cancer
H360	May damage fertility or the unborn child
H361	Suspected of damaging fertility or the unborn child
H370	Causes damage to organs
H372	Causes damage to organs through prolonged or repeated exposure
H373	May cause damage to organs.
H410	Very toxic to aquatic life with long lasting effects

Continued...

H412	Harmful to aquatic life with long lasting effects

Other information

Ingredients with multiple cas numbers

Name	CAS No
aluminium hydroxide	12252-70-9, 1302-29-0, 1330-44-5, 21645-51-2, 51330-22-4
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 142300-82-1, 84540-57-8
Stoddard Solvent	64742-47-8, 8052-41-3.

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.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net

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