



9200-B Structural Epoxy Adhesive

MG Chemicals UK Limited

Version No: A-2.00
Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

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L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	9200-B
Synonyms	SDS Code: 9200-B; 9200-25ML, 9200-50ML, 9200-1.7L
Other means of identification	Structural Epoxy Adhesive

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

Relevant identified uses	Epoxy adhesive hardener for use with resins
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

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

1.4. Emergency telephone number

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

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments [1]	H314 - Skin Corrosion/Irritation Category 1B, H411 - Chronic Aquatic Hazard Category 2, H361 - Reproductive Toxicity Category 2, H317 - Skin Sensitizer Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

2.2. Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H314	Causes severe skin burns and eye damage.
H411	Toxic to aquatic life with long lasting effects.
H361	Suspected of damaging fertility or the unborn child.
H317	May cause an allergic skin reaction.

Supplementary statement(s)

Not Available

Precautionary statement(s) Prevention

9200-B Structural Epoxy Adhesive

P201	Obtain special instructions before use.
P260	Do not breathe 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 [or 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.
P321	Specific treatment (see advice on this label).
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.
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Precautionary statement(s) Disposal

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

Ingestion may produce serious health damage*.

Cumulative effects may result following exposure*.

Limited evidence of a carcinogenic effect*.

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] and amendments
1.68683-29-4 2.Not Available 3.Not Available 4.Not Available	32	<u>acrylonitrile/ butadiene copolymer amine terminated</u>	Acute Toxicity (Inhalation) Category 4; H332, EUH032 [1]
1.7727-43-7 2.231-784-4 3.Not Available 4.01-2119491274-35-XXXX	30	<u>barium sulfate</u>	Not Applicable
1.68410-23-1 2.Not Available 3.Not Available 4.01-2119972323-38-XXXX	24	<u>C18 fatty acid dimers/ tetraethylenepentamine polyamides</u>	Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Serious Eye Damage Category 1, Skin Corrosion/Irritation Category 2; H335, H318, H315 [1]
1.68082-29-1 2.500-191-5 3.Not Available 4.01-2119972320-44-XXXX	7	<u>tall oil/ triethylenetetramine polyamides</u>	Acute Toxicity (Oral and Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Chronic Aquatic Hazard Category 2, Skin Sensitizer Category 1, Respiratory Sensitizer Category 1, Serious Eye Damage Category 1; H302+H332, H315, H411, H317, H334, H318 [1]
1.112-24-3 2.203-950-6 3.612-059-00-5 4.Not Available	3	<u>triethylenetetramine</u>	Acute Toxicity (Dermal) Category 4, Chronic Aquatic Hazard Category 3, Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 1B; H312, H412, H317, H314 [2]
1.140-31-8 2.205-411-0 3.612-105-00-4 4.01-2119471486-30-XXXX	2	<u>N-aminoethylpiperazine</u>	Acute Toxicity (Dermal) Category 4, Chronic Aquatic Hazard Category 3, Skin Sensitizer Category 1, Skin Corrosion/Irritation Category 1B, Acute Toxicity (Oral) Category 4; H312, H412, H317, H314, H302 [2]

9200-B Structural Epoxy Adhesive

Legend: 1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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. <p>For amines:</p> <ul style="list-style-type: none"> ▶ 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	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ 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. <p>For amines:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prosthesis such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay. ▶ 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. <p>This must definitely be left to a doctor or person authorised by him/her. (ICSC13719)</p> <p>For amines:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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. <p>For amines:</p> <ul style="list-style-type: none"> ▶ 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.

For cyanide intoxication (and for certain nitriles which produce cyanide ion)

- ▶ Signs symptoms of acute cyanide poisoning reflect cellular hypoxia and are often non-specific.
- ▶ Cyanosis may be a late finding.
- ▶ A *bradycardic*, hypertensive and tachypneic patient suggests poisoning especially if CNS and cardiovascular depression subsequently occurs.
- ▶ Immediate attention should be directed towards assisted ventilation, administration of 100% oxygen, insertion of intravenous lines and institution of cardiac monitoring.

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9200-B Structural Epoxy Adhesive

- ▶ Obtain an arterial blood gas immediately and correct any severe metabolic acidosis (pH below 7.15).
- ▶ Mildly symptomatic patients generally require supportive care alone. Nitrites should not be given indiscriminately - in all cases of moderate to severe poisoning, they should be given in conjunction with thiosulfate. As a temporizing measure supply amyl nitrite perles (0.2ml inhaled 30 seconds every minute) until intravenous lines for sodium nitrite are established. 10 ml of a 3% solution is administered over 4 minutes to produce 20% methaemoglobin in adults. Follow directly with 50 ml of 25% sodium thiosulfate, at the same rate, IV. If symptoms reappear or persist within 1/2-1 hour, repeat nitrite and thiosulfate at 50% of initial dose. As the mode of action involves the metabolic conversion of the thiosulfate to thiocyanate, renal failure may enhance thiocyanate toxicity.
- ▶ Methylene blue is not an antidote. [Ellenhorn and Barceloux: Medical Toxicology]

If amyl nitrite intervention is employed then Medical Treatment Kits should contain the following:

- ▶ One box containing one dozen amyl nitrite ampoules
- ▶ Two sterile ampoules of sodium nitrite solution (10 mL of a 3% solution in each)
- ▶ Two sterile ampoules of sodium thiosulfate solution (50 mL of a 25% solution in each)
- ▶ One 10 mL sterile syringe. One 50 mL sterile syringe. Two sterile intravenous needles. One tourniquet.
- ▶ One dozen gauze pads.
- ▶ Latex gloves
- ▶ A 'Biohazard' bag for disposal of bloody/contaminated equipment.
- ▶ A set of cyanide instructions on first aid and medical treatment.

- Notes on the use of amyl nitrite:-

- ▶ AN is highly volatile and flammable - do not smoke or use around a source of ignition.
- ▶ If treating patient in a windy or draughty area provide some shelter or protection (shirt, wall, drum, cupped hand etc.) to prevent amyl nitrite vapour from being blown away. Keep ampoule upwind from the nose, the objective is to get amyl nitrite into the patients lungs.
- ▶ Rescuers should avoid AN inhalation to avoid becoming dizzy and losing competence.
- ▶ Lay the patient down. Since AN dilates blood vessels and lowers blood pressure, lying down will help keep patient conscious.
- ▶ **DO NOT overuse - excessive use might put the patient into shock.** Experience at DuPont plants has not shown any serious after-effects from treatment with amyl nitrite.

ADDITIONAL NOTES:

- ▶ Major medical treatment procedures may vary e.g. US (FDA method as recommended by DuPont) uses amyl nitrite as a methaemoglobin generator, followed by treatment with sodium nitrite and then sodium thiosulfate.

MODES OF ACTION: Amyl nitrite (AN) reacts with haemoglobin (HB) to form about 5% methaemoglobin (MHB). Sodium nitrite (NaNO₂) reacts with haemoglobin to form approximately 20-30% methaemoglobin. Methaemoglobin attracts cyanide ions (CN⁻) from tissue and binds with them to become cyanmethaemoglobin (CNMHB). Sodium thiosulfate (Na₂S₂O₃) converts cyanmethaemoglobin to thiocyanate (HSCN) which is excreted by the kidneys. i.e. AN + HB = MHB NaNO₂ + HB = MHB CN + MHB = CNMHB Na₂S₂O₃ + CNMHB + O₂ = HSCN

- ▶ The administration of the antidote salts is intravenous in normal saline, Ringers lactate or other available IV fluid.
- ▶ European practice may use 4-dimethylaminophenol (DMAP) as a methaemoglobin generator. Also hydroxycobalamin (Vitamin B12a) is used. Hydroxycobalamin works by reacting with cyanide to form cyanocobalamin (Vitamin B12) which is excreted in the urine.
- ▶ European and Australian NOHSC (ASCC) propose dicobalt edetate (Kelocyanor) as antidote. This acts by chelating cyanide to form stable cobaltcyanide, which is excreted in the urine. In all cases hyperbaric therapy may increase the efficiency of a cyanide antidote kit.

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 esophagoscopy 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," "glaucompsia"), 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

9200-B Structural Epoxy Adhesive

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.

Polyurethane 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
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5.3. Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ 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. <p>For amines:</p> <ul style="list-style-type: none"> ▶ 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.
Fire/Explosion Hazard	<p>Once acrylic fibre is ignited, an exothermic reaction can occur in the absence of oxygen with evolution of hazardous materials.</p> <ul style="list-style-type: none"> ▶ 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. <p>Combustion products include: carbon dioxide (CO₂) nitrogen oxides (NO_x) sulfur oxides (SO_x) metal oxides other pyrolysis products typical of burning organic material. May emit corrosive fumes.</p>

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	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ 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. ▶ 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. <p>for amines:</p> <ul style="list-style-type: none"> ▶ 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.
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9200-B Structural Epoxy Adhesive

Environmental hazard - contain spillage.

Chemical Class: bases

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

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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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.

▶ 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

Major Spills

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.

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9200-B Structural Epoxy Adhesive

	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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. <p>For low viscosity materials</p> <ul style="list-style-type: none"> ▶ 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. <p>For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):</p> <ul style="list-style-type: none"> ▶ Removable head packaging; ▶ Cans with friction closures and ▶ low pressure tubes and cartridges <p>may be used.</p> <p>-</p> <p>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.</p>
Storage incompatibility	<p>Barium sulfate (barytes)</p> <ul style="list-style-type: none"> ▶ reacts violently with dimethyl sulfoxide, sodium acetylide, finely divided carbon, aluminium, magnesium, zirconium, and possibly other active metals, especially at elevated temperatures ▶ is incompatible with potassium, phosphorus (ignites when primed with nitrate-calcium silicide) ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ▶ Avoid contact with copper, aluminium and their alloys. ▶ Avoid reaction with oxidising agents <p>Amines are incompatible with:</p> <ul style="list-style-type: none"> · isocyanates, halogenated organics, peroxides, phenols (acidic), epoxides, anhydrides, and acid halides. · strong reducing agents such as hydrides, due to the liberation of flammable gas. <p>Amines possess a characteristic ammonia smell, liquid amines have a distinctive 'fishy' smell. Amines are formally derivatives of ammonia, wherein one or more hydrogen atoms have been replaced by a substituent such as an alkyl or aryl group. Compounds with a nitrogen atom attached to a carbonyl group, thus having the structure R-CO-NR'R'', are called amides and have different chemical properties from amines. The water solubility of simple amines is enhanced by hydrogen bonding involving these lone electron pairs. Typically salts of ammonium compounds exhibit the following order of solubility in water: primary ammonium (RNH+3) > secondary ammonium (R2NH+2) > tertiary ammonium (R3NH+). Small aliphatic amines display significant solubility in many solvents, whereas those with large substituents are lipophilic. Aromatic amines, such as aniline, have their lone pair electrons conjugated into the benzene ring, thus their tendency to engage in hydrogen bonding is diminished. Their boiling points are high and their solubility in water is low.</p> <p>Like ammonia, amines are bases. Compared to alkali metal hydroxides, amines are weaker.</p> <ul style="list-style-type: none"> · The basicity of amines depends on: <ul style="list-style-type: none"> · The electronic properties of the substituents (alkyl groups enhance the basicity, aryl groups diminish it). · The degree of solvation of the protonated amine, which includes steric hindrance by the groups on nitrogen. <p>Owing to inductive effects, the basicity of an amine might be expected to increase with the number of alkyl groups on the amine. Correlations are complicated owing to the effects of solvation which are opposite the trends for inductive effects. Solvation effects also dominate the basicity of aromatic amines.</p> <p>Solvation significantly affects the basicity of amines. N-H groups strongly interact with water, especially in ammonium ions. Consequently, the basicity of ammonia is enhanced by 10¹⁰ by solvation.</p> <p>Tertiary amines are more basic than secondary amines, which are more basic than primary amines, and finally ammonia is least basic. The order of pK_b's (basicities in water) does not follow this order. Similarly aniline is more basic than ammonia in the gas phase, but ten thousand times less so in aqueous solution.</p> <p>In aprotic polar solvents such as DMSO, DMF, and acetonitrile the energy of solvation is not as high as in protic polar solvents like water and methanol. For this reason, the basicity of amines in these aprotic solvents is almost solely governed by the electronic effect</p>

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

9200-B Structural Epoxy Adhesive

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
barium sulfate	Inhalation 10 mg/m ³ (Systemic, Chronic) Inhalation 10 mg/m ³ (Local, Chronic) <i>Inhalation 10 mg/m³ (Systemic, Chronic) *</i> <i>Oral 13 000 mg/kg bw/day (Systemic, Chronic) *</i>	115 µg/L (Water (Fresh)) 600.4 mg/kg sediment dw (Sediment (Fresh Water)) 207.7 mg/kg soil dw (Soil) 62.2 mg/L (STP)
C18 fatty acid dimers/ tetraethylenepentamine polyamides	Dermal 1.1 mg/kg bw/day (Systemic, Chronic) Inhalation 3.9 mg/m ³ (Systemic, Chronic) <i>Dermal 0.56 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 0.97 mg/m³ (Systemic, Chronic) *</i> <i>Oral 0.56 mg/kg bw/day (Systemic, Chronic) *</i>	0.004 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.041 mg/L (Water (Marine)) 411.01 mg/kg sediment dw (Sediment (Fresh Water)) 41.1 mg/kg sediment dw (Sediment (Marine)) 82.18 mg/kg soil dw (Soil) 3.14 mg/L (STP)
tall oil/ triethylenetetramine polyamides	Dermal 1.1 mg/kg bw/day (Systemic, Chronic) Inhalation 3.9 mg/m ³ (Systemic, Chronic) <i>Dermal 0.56 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 0.97 mg/m³ (Systemic, Chronic) *</i> <i>Oral 0.56 mg/kg bw/day (Systemic, Chronic) *</i>	0.004 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.043 mg/L (Water (Marine)) 434.02 mg/kg sediment dw (Sediment (Fresh Water)) 43.4 mg/kg sediment dw (Sediment (Marine)) 86.78 mg/kg soil dw (Soil) 3.84 mg/L (STP)
N-aminoethylpiperazine	Dermal 3.33 mg/kg bw/day (Systemic, Chronic) Inhalation 10.6 mg/m ³ (Systemic, Chronic) Inhalation 15 µg/m ³ (Local, Chronic) Inhalation 10.6 mg/m ³ (Systemic, Acute) Inhalation 80 mg/m ³ (Local, Acute)	0.058 mg/L (Water (Fresh)) 0.006 mg/L (Water - Intermittent release) 0.58 mg/L (Water (Marine)) 215 mg/kg sediment dw (Sediment (Fresh Water)) 21.5 mg/kg sediment dw (Sediment (Marine)) 1 mg/kg soil dw (Soil) 250 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	barium sulfate	Barium sulphate: respirable dust	4 mg/m ³	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	barium sulfate	Barium sulphate: inhalable dust	10 mg/m ³	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
barium sulfate	Barium sulfate	15 mg/m ³	170 mg/m ³	990 mg/m 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/m ³	330 mg/m ³	2,000 mg/m ³
triethylenetetramine	Triethylenetetramine	3 ppm	14 ppm	83 ppm
N-aminoethylpiperazine	Aminoethylpiperazine, N-	6.4 mg/m ³	71 mg/m ³	420 mg/m ³

Ingredient	Original IDLH	Revised IDLH
acrylonitrile/ butadiene copolymer amine terminated	Not Available	Not Available
barium sulfate	Not Available	Not Available
C18 fatty acid dimers/ tetraethylenepentamine polyamides	Not Available	Not Available
tall oil/ triethylenetetramine polyamides	Not Available	Not Available
triethylenetetramine	Not Available	Not Available
N-aminoethylpiperazine	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
acrylonitrile/ butadiene copolymer amine terminated	E	≤ 0.1 ppm
C18 fatty acid dimers/ tetraethylenepentamine polyamides	E	≤ 0.1 ppm
tall oil/ triethylenetetramine polyamides	E	≤ 0.1 ppm

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

Continued...

9200-B Structural Epoxy Adhesive

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
triethylenetetramine	E	≤ 0.1 ppm
N-aminoethylpiperazine	D	> 0.1 to ≤ 1 ppm
Notes:	<i>Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.</i>	

MATERIAL DATA

For 1,3-butadiene:

Odour Threshold Value: 0.45 ppm (detection), 1.1 ppm (recognition)

Exposure at or below the TLV-TWA is thought to provide significant protection for workers against systemic toxicity including cancer.

US rubber workers reached an accord in 1996 to limit exposure to 1 ppm with a 15-minute, short-term limit of 5 ppm. This TLV-TWA is currently under review in light of a report of animal carcinogenicity at 6.25 ppm.

Odour Safety Factor(OSF)

OSF=1.3 ('1,3-BUTADIENE')

Phenol and catechol produce similar toxic actions although catechol is considerably less toxic than phenol in animal tests following inhalation. By other exposure routes catechol is considerably more toxic. The TLV-TWA is thought to be protective against the significant risk of dermal and upper respiratory tract irritation and central nervous system effects, including convulsion.

Barium sulfate has been identified as a nontoxic dust. However high dust levels have caused benign pneumoconiosis (baritosis). The TLV-TWA is thought to be protective against the risk of eye, nose and upper respiratory tract irritation and perhaps, pneumoconiosis.

TLV TWA: 0.001 mg/m³ (as total proteins) Inhalable fraction skin sensitiser as rubber processing fume:

MEL-TWA: 0.6 mg/m³ as cyclohexane solubles [HSE, UK]

BRMA-TWA: 0.25 mg/m³ as cyclohexane solubles [BRMA Code of Practice]

Rubber fume is a complex and indeterminate mixture of substances and is defined as 'fume evolved in the mixing, milling and blending of natural rubber and synthetic polymers combined with chemicals, and in the processes which convert the resultant blend into finished products or parts thereof, and including any inspection procedures where fume continues to be evolved'.

'Fume' generally describes solid particles generated by chemical reactions, or by condensation from the gaseous state, usually after volatilisation from melted substances, and often accompanied by a chemical reaction such as oxidation or thermal breakdown.

Several chemical agents may occur in rubber fume which are experimental or animal carcinogens, however, given the number of chemicals used or formed during rubber making, difficulties arise in attributing a particular effect to a given exposure.

Stomach cancer has been associated with work in jobs early in the production line; lung and lower oesophagus cancer with all work processes; and lymphomas with jobs where co-exposure to solvents occurs. Other cancers have also been reported with liver tumours appearing as a secondary phenomenon. No no-effect levels have been determined.

Two studies showed no excess of bladder cancer in workers entering the industry after 1950: the excess risk before that date is thought to result from exposure to residual beta-naphthylamines previously used as anti-oxidants.

as rubber process dust:

MEL-TWA: 6 mg/m³ [HSE, UK]

Rubber process dust is a complex, variable mixture of particulates defined as 'dust arising in the stages of rubber manufacture where ingredients are handled, weighed, added to or mixed with natural or synthetic elastomers. It does not include dusts arising from the abrasion of cured rubber but occurs during the preparation of compounds of either synthetic or natural rubber.

There is some evidence that occupational exposure to rubber dusts produces an excess incidence of stomach cancer. HSE data concluded that there was a small but significant excess of stomach cancer associated with the initial processes in rubber manufacture. Stomach cancer shows a marked social class gradient, which may lead to an over-estimation of the risk.

One report from the USA stated that exposure in rubber processing areas produces pulmonary disease but this has not been supported by UK epidemiology nor reports from the industry.

No no-effect level has been determined. The MEL was considered appropriate because it was felt reasonably practical for industry to comply with this value.

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 barium compounds:


The recommended TLV-TWA is based on satisfactory results achieved while employing an internal limit for barium nitrate at a national laboratory. It is not known what degree of added safety this limit incorporates.

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.	
	Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. 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:		

Continued...

9200-B Structural Epoxy Adhesive

	<table border="1" data-bbox="384 203 1090 371"> <thead> <tr> <th data-bbox="384 203 831 232">Lower end of the range</th> <th data-bbox="831 203 1090 232">Upper end of the range</th> </tr> </thead> <tbody> <tr> <td data-bbox="384 232 831 262">1: Room air currents minimal or favourable to capture</td> <td data-bbox="831 232 1090 262">1: Disturbing room air currents</td> </tr> <tr> <td data-bbox="384 262 831 291">2: Contaminants of low toxicity or of nuisance value only.</td> <td data-bbox="831 262 1090 291">2: Contaminants of high toxicity</td> </tr> <tr> <td data-bbox="384 291 831 320">3: Intermittent, low production.</td> <td data-bbox="831 291 1090 320">3: High production, heavy use</td> </tr> <tr> <td data-bbox="384 320 831 349">4: Large hood or large air mass in motion</td> <td data-bbox="831 320 1090 349">4: Small hood-local control only</td> </tr> </tbody> </table> <p data-bbox="384 389 1490 521">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.</p>	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
Lower end of the range	Upper end of the range										
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents										
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3: Intermittent, low production.	3: High production, heavy use										
4: Large hood or large air mass in motion	4: Small hood-local control only										
8.2.2. Personal protection											
Eye and face protection	<ul style="list-style-type: none"> ▶ 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] <p data-bbox="384 999 478 1019">For amines:</p> <p data-bbox="384 1021 580 1041">SPECIAL PRECAUTION:</p> <ul style="list-style-type: none"> ▶ 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. <p data-bbox="384 1133 469 1153">CAUTION:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Elbow length PVC gloves ▶ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. <p data-bbox="384 1373 443 1393">NOTE:</p> <ul style="list-style-type: none"> ▶ 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. <p data-bbox="384 1460 1473 1527">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.</p> <p data-bbox="384 1529 1458 1572">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.</p> <p data-bbox="384 1574 1426 1617">Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p data-bbox="384 1619 1241 1639">Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> - frequency and duration of contact, - chemical resistance of glove material, - glove thickness and - dexterity <p data-bbox="384 1731 1254 1751">Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> - 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. <p data-bbox="384 1908 914 1928">As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> - Excellent when breakthrough time > 480 min - Good when breakthrough time > 20 min - Fair when breakthrough time < 20 min - Poor when glove material degrades <p data-bbox="384 2020 1142 2040">For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p data-bbox="384 2042 1458 2107">It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p data-bbox="384 2110 1414 2152">Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p>										

9200-B Structural Epoxy Adhesive

	<p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> • Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. • Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>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.</p> <p>When handling liquid-grade epoxy resins wear chemically protective gloves, boots and aprons.</p> <p>The performance, based on breakthrough times, of:</p> <ul style="list-style-type: none"> • Ethyl Vinyl Alcohol (EVAL laminate) is generally excellent • Butyl Rubber ranges from excellent to good • Nitrile Butyl Rubber (NBR) from excellent to fair. • Neoprene from excellent to fair • Polyvinyl (PVC) from excellent to poor <p>As defined in ASTM F-739-96</p> <ul style="list-style-type: none"> • Excellent breakthrough time > 480 min • Good breakthrough time > 20 min • Fair breakthrough time < 20 min • Poor glove material degradation <p>Gloves should be tested against each resin system prior to making a selection of the most suitable type. Systems include both the resin and any hardener, individually and collectively)</p> <ul style="list-style-type: none"> • DO NOT use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical 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. <p>Replacement time should be considered when selecting the most appropriate glove. It may be more effective to select a glove with lower chemical resistance but which is replaced frequently than to select a more resistant glove which is reused many times</p> <p>For amines:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower.

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:

9200-B Structural Epoxy Adhesive

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

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as 'feel' or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Type 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 P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - Full-face

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

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

Where engineering controls are not feasible and work practices do not reduce airborne amine concentrations below recommended exposure limits, appropriate respiratory protection should be used. In such cases, air-purifying respirators equipped with cartridges designed to protect against amines are recommended.

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

Continued...

9200-B Structural Epoxy Adhesive

9.1. Information on basic physical and chemical properties

Appearance	amber		
Physical state	Liquid	Relative density (Water = 1)	1.18
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	>20.5
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	>122	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)	<0.001	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>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.</p> <p>Inhalation of epoxy resin amine hardener vapours (including polyamines and amine adducts) may produce bronchospasm and coughing episodes lasting days after cessation of the exposure. Even faint traces of these vapours may trigger an intense reaction in individuals showing 'amine asthma'. The literature records several instances of systemic intoxications following the use of amines in epoxy resin systems. Excessive exposure to the vapours of epoxy amine curing agents may cause both respiratory irritation and central nervous system depression. Signs and symptoms of central nervous system depression, in order of increasing exposure, are headache, dizziness, drowsiness, and incoordination. In short, a single prolonged (measured in hours) or excessive inhalation exposure may cause serious adverse effects, including death.</p> <p>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.</p>
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9200-B Structural Epoxy Adhesive

	<p>Exposure to toxic levels of butadiene has also produced chromosome damage. Human volunteers exposed at 2000-8000 ppm 1,3-butadiene for 6-8 hours showed slight smarting of the eyes, difficulty in focusing on instrument scales and a transient objection to butadiene odour. Characteristics of exposure include dry nose/mouth/throat, fatigue, headache, vertigo, nausea, narcosis, respiratory paralysis, and central nervous system depression. Very high concentrations may cause loss of consciousness or death. Repeated and prolonged exposure to 1,3-butadiene vapour may cause kidney and liver damage. Deep anaesthesia was induced in rabbits in 8 to 10 minutes at 200000 to 250000 ppm. Recovery from brief periods of anaesthesia occurred within two minutes of terminating the exposure.</p> <p>The intensity and time of exposure to hydrogen cyanide determines effects, symptoms. Short term inhalation of 20 to 40 ppm hydrogen cyanide may result in slight symptoms. Higher concentrations can cause death within minutes or hours; a concentration of 270 ppm can be fatal in one minute. Acute exposure to cyanides can cause death by cyanosis, asphyxia. At very low doses, symptoms of hydrogen cyanide exposure may be weakness, headaches, confusion, giddiness, dizziness, confusion, anxiety, nausea and vomiting. Normal blood pressure with rapid pulse is usual in mild cases. The respiratory rate varies with the intensity of exposure: rapid with mild exposure, or slow and gasping with severe exposure. Symptoms of mild exposure to hydrogen cyanide are completely reversed when exposure ceases.</p> <p>In severe cases, breathing is rapid and deep, then becomes slow and gasping. The victim may feel an irregular heartbeat and tightness in the chest. The skin appears bright pink or red. Fluid may fill the lungs and interfere with breathing. Unconsciousness, convulsions and death can quickly follow depending on the degree of exposure. Massive exposures may produce sudden collapse and death. concentration of 270 If death does not result, recovery is usually complete. There have however been a few reports of after-effects. These are similar to those seen in people deprived of oxygen, e.g. near-drowning victims</p> <p>Barium fumes are respiratory irritants. Over-exposure to barium dusts and fume may result in rhinitis, frontal headache, wheezing, laryngeal spasm, salivation and anorexia. Long term effects include nervous disorders and adverse effects on the heart, circulatory system and musculature. Heavy exposures may result in a benign pneumoconiosis.</p>
Ingestion	<p>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.</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Ingestion of amine epoxy-curing agents (hardeners) may cause severe abdominal pain, nausea, vomiting or diarrhoea. The vomitus may contain blood and mucous. If death does not occur within 24 hours there may be an improvement in the patients condition for 2-4 days only to be followed by the sudden onset of abdominal pain, board-like abdominal rigidity or hypo-tension; this indicates that delayed gastric or oesophageal corrosive damage has occurred.</p> <p>All cases of acute oral barium poisoning in adults exhibit gastrointestinal disturbances as the initial symptoms. These include gastric pain, vomiting, and diarrhea.</p> <p>Ingestion of soluble barium compounds may result in ulceration of the mucous membranes of the gastrointestinal tract, tightness in the muscles of the face and neck, gastroenteritis, vomiting, diarrhoea, muscular tremors and paralysis, anxiety, weakness, laboured breathing, cardiac irregularity due to contractions of smooth, striated and cardiac muscles (often violent and painful), slow irregular pulse, hypertension, convulsions and respiratory failure.</p> <p>The predominant musculoskeletal effect observed in cases of barium toxicity in humans is progressive muscle weakness, often leading to partial or total paralysis. In severe cases, the paralysis affects the respiratory system. The likely cause of the muscle weakness was the barium-induced hypokalaemia (low potassium levels) rather than a direct effect on muscles.</p> <p>Numbness and tingling around the mouth and neck were sometimes among the first symptoms of barium toxicity in humans. Occasionally, these neurological symptoms extended to the extremities. Partial and complete paralysis occurred in severe cases, often accompanied by an absence of deep tendon reflexes</p> <p>Toxic effects on the kidneys have been observed in several adult cases of acute barium poisoning. Effects include hemoglobin in the urine (which may be indicative of kidney damage), renal insufficiency, degeneration of the kidneys, and acute renal failure.</p> <p>Studies in animals suggest that the kidney is a critical target of barium toxicity. An increase in relative kidney weight (kidney/brain weight ratio) was observed in male and female rats receiving a single gavage dose of 198 mg barium/kg/day as barium chloride in water.</p> <p>Acute exposure to presumably high doses of barium carbonate, barium sulfate, or barium chloride can result in serious effects on heart rhythm. Barium adversely affects cardiac automaticity resulting in ventricular tachycardia and other disruptions of rhythm. Hypotension has also been reported in some cases. The likely cause of these effects was barium-induced hypokalaemia.</p> <p>Several human studies have investigated a possible association between exposure to low levels of barium and alterations in blood pressure and cardiac rhythms. In a small-scale (11 subjects) study of individuals exposed to 0.1 or 0.2 mg barium/kg/day as barium chloride in drinking water for 4 weeks, no significant alterations in blood pressure or ECG readings were found. There was no significant alteration in blood pressure measurements or alterations in hypertension, heart disease, or stroke among residents of two communities with elevated (0.2 mg barium/kg/day) or low (0.003 mg barium/kg/day) levels of barium in drinking water. Significantly higher mortality rates for cardiovascular disease and heart disease (arteriosclerosis) were found in the elevated barium communities (0.06-0.3 mg barium/kg/day) than in the low barium communities (0.006 mg barium/kg/day). The largest difference between the groups was in individuals 65 years of age and older. These results should be interpreted cautiously because the study did not control for a number of potential confounding variables such as the use of water softeners, which would reduce the amount of barium and increase sodium levels, duration of exposure, or actual barium intakes.</p> <p>Several animal studies have examined potential cardiovascular end points following acute-, intermediate-, or chronic-duration exposures. Significant increases in systolic blood pressure were observed in rats exposed to 8.6 or 11 mg barium/kg/day for 1 or 4 months, respectively; no effect levels were 1.0 and 1.2 mg barium/kg/day. When the duration of exposure was longer (8-16 months), the LOAEL for increased blood pressure was 0.80 mg barium/kg/day and the NOAEL was 0.17 mg barium/kg/day. Depressed rates of cardiac contraction and cardiac conductivity and decreased cardiac ATP levels were observed in another group of rats exposed to 7.2 mg barium/kg/day. In contrast to the findings in this study, a second study could find no significant alterations in blood pressure were observed in rats exposed to up to 150 mg barium/kg/day in drinking water for 16 weeks; it should be noted that the second was conducted in uninephrectomized rats or Dahl salt-sensitive and salt-resistant rats. NTP (1994) also found no significant alterations in blood pressure, heart rate, or ECG readings in rats exposed to 180 mg barium/kg/day for 45 or 90 days. The low metal diet used in the first study may have influenced the study outcome.</p> <p>When evaluating the health effects of barium compounds, it is important to keep in mind that different barium compounds have different solubilities in water and body fluids and therefore serve as variable sources of the Ba²⁺ ion. The Ba²⁺ ion and the soluble compounds of barium (notably chloride, nitrate, and hydroxide) are generally highly toxic to humans and experimental animals. The insoluble barium compounds (notably sulfate) are inefficient sources of the Ba²⁺ ion and therefore are generally nontoxic. Although barium carbonate is insoluble in water, barium ions would be released from ingested barium carbonate in the acid milieu of the stomach.</p>

9200-B Structural Epoxy Adhesive

<p>Skin Contact</p>	<p>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. Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur. Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions. Individuals exhibiting 'amine dermatitis' may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis. NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided.</p> <p>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. The diepoxide of butadiene (1,2:3,4-diepoxbutane), a probable metabolite, has been reported to be a mild skin tumourigen when applied topically to the skin of mice 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.</p> <p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
<p>Eye</p>	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. 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 (glauropsia, '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, glauropsia 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.</p>
<p>Chronic</p>	<p>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. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Acrylonitrile is a skin and respiratory sensitiser. Chronic exposures may produce severe liver inflammation. Chronic effects of occupational exposure include skin and eye irritation, nausea, vomiting, weakness, fatigue, jaundice, anaemia, leukocytosis, bilirubinaemia, increased serum thiocyanate concentrations, and hepatic and renal irritation. When administered orally (by gavage or in drinking water), acrylonitrile induced increased incidences of fore-stomach squamous cell papillomas, central nervous system microgliomas, mammary gland carcinomas and Zymbal gland carcinomas, forestomach papillomas and acanthomas, and central nervous system neoplasms in rats of both sexes. An epidemiological study of textile-plant workers potentially exposed to acrylonitrile and observed for 20 years, showed an increased incidence of cancers of the lung; further follow-up of this cohort revealed a continued excess of lung cancer, although during the actual 5-year follow-up period, there was no excess. This follow-up showed a significant excess of cancer of the prostate. <i>NTP</i> Amongst humans occupationally exposed to 1,3-butadiene several cancer sites with high statistically significant mortality ratios were identified. These included cancer of the testes, cancers of the digestive system (oesophagus, stomach, large intestine), larynx and Hodgkin's disease. Exposure by rats to 1,3-butadiene gas at 1000 ppm/6hrs/day, 5 days /week (105 weeks for females and 111 weeks for males) caused significant increases in the incidence of tumours at various sites; mammary gland adenomas and sarcomas; uterine sarcomas; Zymbal gland carcinomas; thyroid adenomas and pancreatic adenomas. A high incidence of malignant lymphoma was found amongst a group of exposed rats in a second study On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body 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. The toxic properties of cyanide result from its ability to inhibit enzymes required for the respiration of cells within the body. Chronic exposure to cyanides, at levels too low to produce clinical complaints, may cause dermatitis, itching, scarlet rash, perforation of nasal septum, throat irritation, muscular cramps, weight loss and enlargement of the thyroid gland. Workers with pre-existing CNS, heart and lung disorders are at significant risk. A wide range of symptoms are thought to be caused by long-term, low-level (often less than 10 ppm) exposure to cyanides. Symptoms include persistent runny nose, weakness, dizziness, giddiness, headache, nausea, vomiting, abdominal pain, throat irritation, changes in taste and smell, muscle cramps, weight loss, flushing of the face, itching and irritation of the upper respiratory tract, throat and eyes and enlargement of the</p>

9200-B Structural Epoxy Adhesive

thyroid gland. These symptoms are not specific to cyanide exposure; therefore it has been difficult to prove that chronic cyanide toxicity exists. Repeated minor contact with cyanides produces a characteristic scarlet rash with itching, papules (small, superficial raised spots on the skin), perforation of the nasal septum and possible sensitisation. Concerns have been expressed that low-level, long term exposures may result in damage to the nerves of the eye.

Chronic exposure to cyanides and certain nitriles may result in interference to iodine uptake by thyroid gland and its consequent enlargement. This occurs following metabolic conversion of the cyanide moiety to the less toxic thiocyanate which is excreted in the urine. Thyroid insufficiency may also occur as a result of metabolic conversion of cyanides to the corresponding thiocyanate..

A small amount of cyanide is excreted, unchanged, in the breath, sweat and urine.

Workers exposed to barium compounds have been reported to show an increased incidence of hypertension, irritation of the respiratory system, and damage to the spleen, liver and bone marrow. Long term exposure to some barium compounds (especially inorganic species) may produce a condition known as baritosis, a form of benign pneumoconiosis. X-ray may show this when no other abnormal signs are present.

Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. Barium sulfate produces noncollagenous pneumoconiosis identified by minimal stromal reaction, consisting mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible. Miners of ores containing barium sulfate do not show symptoms, abnormal physical signs, an incapacity to work, diminished lung function, an increased likelihood of developing pulmonary or other bronchial infections or other thoracic disease despite the fact that particulate matter may have been retained in the lungs for many years.

No changes in mortality were observed in rats chronically exposed to doses as high as 60 mg barium/kg/day as barium chloride in the drinking water. An increase in mortality, attributable to nephropathy, was observed in mice chronically exposed to 160 mg barium/kg/day as barium chloride in drinking water; the number of deaths was similar to controls in mice exposed to 75 mg barium/kg/day. In male mice exposed to 0.95 mg barium/kg/day as barium acetate in drinking water, a significant decrease in longevity (defined as average lifespan of the last five surviving animals) was observed; however, no significant differences in mean lifespan were observed. Similarly, lifespan was not significantly altered in female mice exposed to 0.95 mg barium/kg/day or male or female rats exposed to 0.7 mg barium/kg/day as barium acetate in drinking water. The potential for barium to induce reproductive and developmental effects has not been well investigated. Decreases in the number of sperm and sperm quality and a shortened estrous cycle and morphological alterations in the ovaries were observed in rats exposed to 2.2 mg barium/m3 and higher in air for an intermediate duration. Interpretation of these data is limited by the poor reporting of the study design and results, in particular, whether the incidence was significantly different from controls. In general, oral exposure studies have not found morphological alterations in reproductive tissues of rats or mice exposed to 180 or 450 mg barium/kg/day, respectively, as barium chloride in drinking water for an intermediate duration. Additionally, no significant alterations in reproductive performance was observed in rats or mice exposed to 200 mg barium/kg/day as barium chloride in drinking water. Decreased pup birth weight and a nonsignificant decrease in litter size have been observed in the offspring of rats exposed to 180/200 mg barium/kg/day as barium chloride in drinking water prior to mating. Several studies have examined the carcinogenic potential of barium following oral exposure and did not find significant increases in the tumour incidence.

9200-B Structural Epoxy Adhesive	TOXICITY	IRRITATION
	Not Available	Not Available
acrylonitrile/ butadiene copolymer amine terminated	TOXICITY	IRRITATION
	dermal (rat) LD50: >3000 mg/kg ^[2]	Eye (rabbit): irritant *
	Inhalation(Rat) LC50 5.61 mg/l/4h ^[2]	Skin: irritant, Draize Score 3.6*
	Oral(Rat) LD50 >15380 mg/kg ^[2]	
barium sulfate	TOXICITY	IRRITATION
	=15000 mg/kg ^[2]	Not Available
	Oral(Mouse) LD50 >3000 mg/kg ^[2]	
C18 fatty acid dimers/ tetraethylenepentamine polyamides	TOXICITY	IRRITATION
	Not Available	Not Available
tall oil/ triethylenetetramine polyamides	TOXICITY	IRRITATION
	Oral(Rat) LD50 >5000 mg/kg ^[2]	Not Available
triethylenetetramine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 805 mg/kg ^[2]	Eye (rabbit):20 mg/24 h - moderate
	Oral(Mouse) LD50 =1600 mg/kg ^[2]	Eye (rabbit); 49 mg - SEVERE
	Oral(Rat) LD50 =2780 mg/kg ^[2]	Skin (rabbit): 490 mg open SEVERE
	Oral(Rat) LD50 =4300 mg/kg ^[2]	Skin (rabbit): 5 mg/24 SEVERE
	Oral(Rat) LD50 2500 mg/kg ^[2]	
N-aminoethylpiperazine	TOXICITY	IRRITATION
	Oral(Rat) LD50 2140 mg/kg ^[2]	Eye (rabbit): 20 mg/24h - mod
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 0.1 mg/24h - mild

9200-B Structural Epoxy Adhesive

Skin (rabbit): 5 mg/24h - SEVERE

Skin: adverse effect observed (corrosive)^[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

<p>9200-B Structural Epoxy Adhesive</p>	<p>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.</p> <ul style="list-style-type: none"> ▶ 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 the amines are usually transient. <p>Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.</p> <p>Inhalation:</p> <p>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.</p> <p>Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.</p> <p>Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.</p> <p>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.</p> <p>While most polyurethane amine catalysts are not sensitizers, 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 sensitized, 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.</p> <p>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.</p> <p>Skin Contact:</p> <p>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.</p> <p>Skin contact with some amines may result in allergic sensitization. Sensitized 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.</p> <p>Eye Contact:</p> <p>Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations.</p> <p>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.)</p> <p>Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.</p> <p>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.</p> <p>Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.</p> <p>Ingestion:</p> <p>The oral toxicity of amine catalysts varies from moderately to very toxic.</p> <p>Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract.</p> <p>Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.</p> <p>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.</p> <p>Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000 Alliance for Polyurethanes Industry</p>
<p>ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED</p>	<p>* B.F. Goodrich</p> <p>The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.</p> <p>Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).</p> <p>The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<p>BARIUM SULFATE</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES</p>	<p>**[Valspar]</p>
<p>TALL OIL/ TRIETHYLENETETRAMINE POLYAMIDES</p>	<p>Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitization, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances.</p> <p>Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).</p> <p>Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.</p>

9200-B Structural Epoxy Adhesive

Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.

For imidazoline surfactants (amidoamine/ imidazoline - AAls)

All substances within the AAI group show the same reactive groups, show similar composition of amide, imidazoline, and some dimer structures of both, with the length of original EA amines used for production as biggest difference. Inherent reactivity and toxicity is not expected to differ much between these substances.

All in vivo skin irritation/corrosion studies performed on AAI substances all indicate them to be corrosive following 4 hour exposure. There do not seem to be big differences in response with the variation on EA length used for the production of the AAI.

The available data available for AAI substances indicate that for AAI based on shorter polyethylenamines (EA), higher toxicity is observed compared to AAI based on longer EA. The forming of imidazoline itself does not seem to play a significant role. For cross-reading in general Fatty acid reaction product with diethylenetriamine (AAI-DETA) therefore represents the worst case. In series of 28-day and combined repeated dose/reproduction screening toxicity studies (OECD 422) AAI-DETA has shown the highest level of toxicity

Acute oral exposure of tall oil + triethylenepentamine (TEPA) show limited acute toxicity, with a LD50 above 2000 mg/kg bw. Hence no classification is required.

Acute dermal testing with corrosive materials is not justified. As a consequence no classification can be made for acute dermal toxicity. Effects will be characterised by local tissue damage. Systemic uptake via skin is likely to be very limited. The low acute oral toxicity indicate a low systemic toxicity.

For dermal exposure no good overall NOAEL can be established as effects are rather characterized by local corrosive effects that are related to duration, quantity and concentration, than by systemic toxicity due to dermal uptake. The mode of action for AAI follows from its structure, consisting of an apolar fatty acid chain and a polar end of a primary amine from the polyethylenamine. The structure can disrupt the cytoplasmic membrane, leading to lyses of the cell content and consequently the death of the cell.

The AAI are protonated under environmental conditions which causes them to strongly adsorb to organic matter. This leads to a low dermal absorption.

No classification for acute dermal toxicity is therefore indicated.

Also for acute inhalation toxicity information for classification is lacking, and is testing not justified. Due to very low vapour pressure is the likelihood of exposure low.

AAI do not contain containing aliphatic, alicyclic and aromatic hydrocarbons and have a relatively high viscosity and so do not indicate an immediate concern for aspiration hazard.

Various studies with different AAI indicate that these substances can cause dermal sensitisation.

All substances within the AAI group show the same reactive groups, show similar composition of amide, imidazoline, and some dimer structures of both, with the length of original EA amines used for production as biggest difference. Inherent reactivity and toxicity is not expected to differ much between these substances, aspects which determine sensitization.

The actual risk of sensitisation is probably low, as AAI are corrosive to skin and consequently exposure will be low due to necessary protective measures to limit dermal exposure.

The likelihood for exposure via inhalation and thus experience respiratory irritation or becoming sensitised to AAI, is very low considering the high boiling point (> 300 deg C) and very low vapour pressure (0.00017 mPa at 25 deg C for diethylenetriamine (DETA) based AAI). In case of high exposure by inhalation, local effects will be more prominent than possible systemic effects considering the low systemic toxicity seen in acute oral toxicity testing

However, some calculations can be made for systemic effects following short-term inhalation exposure by extrapolating information from an OECD 422 study on 'tall oil reaction products with tetraethylenepentamine showing a NOAEL of 300 mg/kg/day. This would certainly be protective for levels of acute inhalation expected to lead to similar systemic exposure levels.

The corrected 8 hr inhalation NOAEC for workers is NOAEL (300 mg/kg) * 1.76 mg/m³ = 529 mg/m³ (assuming no difference in absorption following oral and inhalation exposure). Assessment factors further applied: No interspecies factor is needed due to allometric scaling applied in calculation of corrected NOAEC. Further combined inter-/intra-species for workers AF = 3 (ECETOC concept). As this involves acute exposures, no extrapolation for duration is needed.

This results in a DNEL of 529/3 = 176 mg/m³. A short term/acute exposure at this level can be assumed not to lead to systemic toxicity.

Repeat dose toxicity:

A combined repeated dose/reproduction screening toxicity study according to OECD 422 with Fatty acid reaction products with tetraethylenepentamine resulted to a NOAEL of 300 mg/kg bw/day, the highest dose tested. Also available data from the group of Amidoamine/Imidazoline (AAI) substances, including 90-day studies in rat and dogs on a similar substance, indicate very low toxicity.

Consequently, serious toxicity is not observed at levels requiring consideration classification for STOTS-RE

Genotoxicity:

Tall oil, reaction products with tetraethylenepentamine is not mutagenic in the Salmonella typhimurium reverse mutation assay (based on test with Fatty acids C16-18, C18 unsaturated reaction products with tetraethylenepentamine), is not clastogenic in human lymphocytes, and not mutagenic in the TK mutation test with L5178Y mouse lymphoma cells.

It can therefore be concluded that tall oil, reaction products with tetraethylenepentamine not genotoxic.

Toxicity to reproduction:

The database of relevant studies available for the group of amidoamine/ imidazolines (AAI) include various OECD 422 studies and an OECD 414 study, that all show no concerns regarding reproduction or developmental toxicity. Also all already available data from the group of AAI substances, including a 90-day study in dogs on a similar substance, indicate low toxicity and no adverse effects on reproductive organs.

REACH Dossier

For quaternary ammonium compounds (QACs):

Quaternary ammonium compounds (QACs) are cationic surfactants. They are synthetic organically tetra-substituted ammonium compounds, where the R substituents are alkyl or heterocyclic radicals (where hydrogen atoms remain unsubstituted, the term 'secondary- or 'tertiary-ammonium compounds' is preferred) .

A common characteristic of these synthetic compounds is that one of the R's is a long-chain hydrophobic aliphatic residue

The cationic surface active compounds are in general more toxic than the anionic and non-ionic surfactants. The positively-charged cationic portion is the functional part of the molecule and the local irritation effects of QACs appear to result from the quaternary ammonium cation.

Due to their relative ability to solubilise phospholipids and cholesterol in lipid membranes, QACs affect cell permeability which may lead to cell death. Further QACs denature proteins as cationic materials precipitate protein and are accompanied by generalised tissue irritation.

It has been suggested that the experimentally determined decrease in acute toxicity of QACs with chain lengths above C16 is due to decreased water solubility.

In general it appears that QACs with a single long-chain alkyl groups are more toxic and irritating than those with two such substitutions,

The straight chain aliphatic QACs have been shown to release histamine from minced guinea pig lung tissue. However, studies with benzalkonium chloride have shown that the effect on histamine release depends on the concentration of the solution. When cell suspensions (11% mast cells) from rats were exposed to low concentrations, a decrease in histamine release was seen. When exposed to high concentrations the opposite result was obtained.

In addition, QACs may show curare-like properties (specifically benzalkonium and cetylpyridinium derivatives, a muscular paralysis with no involvement of the central nervous system. This is most often associated with lethal doses. Parenteral injections in rats, rabbits and dogs have resulted in prompt but transient limb paralysis and sometimes fatal paresis of the respiratory muscles. This effect seems to be transient. From human testing of different QACs the generalised conclusion is obtained that all the compounds investigated to date exhibit similar toxicological properties.

Acute toxicity: Studies in rats have indicated poor intestinal absorption of QACs. Acute toxicity of QACs varies with the compound and, especially, the route of administration. For some substances the LD50 value is several hundreds times lower by the i.p. or i.v. than the oral route, whereas toxicities between the congeners only differ in the range of two to five times.

At least some QACs are significantly more toxic in 50% dimethyl sulfoxide than in plain water when given orally

Probably all common QAC derivatives produce similar toxic reactions, but as tested in laboratory animals the oral mean lethal dose varies with the compound .

9200-B Structural Epoxy Adhesive

Oral toxicity: LD50 values for QACs have been reported within the range of 250-1000 mg/kg for rats, 150-1000 mg/kg for mice, 150-300 mg/kg for guinea pigs and about 500 mg/kg b.w. for rabbits and dogs. The ranges observed reflect differences in the study designs of these rather old experiments as well as differences between the various QACs.

The oral route of administration was characterised by delayed deaths, gastrointestinal lesions and respiratory and central nervous system depression. It was also found that given into a full stomach, the QACs lead to lower mortality and fewer gastrointestinal symptoms. This supports the suggestion of an irritating effect.

Dermal toxicity: It has been concluded that the maximum concentration that did not produce irritating effect on intact skin is 0.1%. Irritation became manifest in the 1-10% range. Concentrations below 0.1% have caused irritation in persons with contact dermatitis or broken skin. Although the absorption of QACs through normal skin probably is of less importance than by other routes, studies with excised guinea pig skin have shown that the permeability constants strongly depend on the exposure time and type of skin.

Sensitisation: Topical mucosal application of QACs may produce sensitisation. Reports on case stories and patch tests have shown that compounds such as benzalkonium chloride, cetalkonium chloride and cetrimide may possibly act as sensitisers. However, in general it is suggested that QACs have a low potential for sensitising man. It is difficult to distinguish between an allergic and an irritative skin reaction due to the inherent skin irritating effect of QACs.

Long term/repeated exposure:

Inhalation: A group of 196 farmers (with or without respiratory symptoms) were evaluated for the relationship between exposure to QACs (unspecified, exposure levels not given) and respiratory disorders by testing for lung function and bronchial responsiveness to histamine. After histamine provocation statistically significant associations were found between the prevalence of mild bronchial responsiveness (including asthma-like symptoms) and the use of QACs as disinfectant. The association seems even stronger in people without respiratory symptoms.

Genetic toxicity: QACs have been investigated for mutagenicity in microbial test systems. In Ames tests using *Salmonella typhimurium* with and without metabolic activation no signs of mutagenicity have been observed. Negative results were also obtained in *E. coli* reversion and *B. subtilis* rec assays. However, for benzalkonium chloride also positive and equivocal results were seen in the *B. subtilis* rec assays.

Fatty acid amides (FAA) are ubiquitous in household and commercial environments. The most common of these are based on coconut oil fatty acids alkanolamides. These are the most widely studied in terms of human exposure.

Fatty acid diethanolamides (C8-C18) are classified by Comité Européen des Agents de Surface et de leurs Intermediaires Organiques (CESIO) as Irritating (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes). Fatty acid monoethanolamides are classified as Irritant (Xi) with the risk phrases R41.

Several studies of the sensitization potential of cocoamide diethanolamide (DEA) indicate that this FAA induces occupational allergic contact dermatitis and a number of reports on skin allergy patch testing of cocoamide DEA have been published. These tests indicate that allergy to cocoamide DEA is becoming more common.

Alkanolamides are manufactured by condensation of diethanolamine and the methylester of long chain fatty acids. Several alkanolamides (especially secondary alkanolamides) are susceptible to nitrosamine formation which constitutes a potential health problem. Nitrosamine contamination is possible either from pre-existing contamination of the diethanolamine used to manufacture cocoamide DEA, or from nitrosamine formation by nitrosating agents in formulations containing cocoamide DEA. According to the Cosmetic Directive (2000) cocoamide DEA must not be used in products with nitrosating agents because of the risk of formation of N-nitrosamines. The maximum content allowed in cosmetics is 5% fatty acid dialkanolamides, and the maximum content of N-nitrosodialkanolamines is 50 mg/kg. The preservative 2-bromo-2-nitropropane-1,3-diol is a known nitrosating agent for secondary and tertiary amines or amides. Model assays have indicated that 2-bromo-2-nitropropane-1,3-diol may lead to the N-nitrosation of diethanolamine forming the carcinogenic compound, N-nitrosodiethanolamine which is a potent liver carcinogen in rats (IARC 1978).

Several FAAs have been tested in short-term genotoxicity assays. No indication of any potential to cause genetic damage was seen. Lauramide DEA was tested in mutagenicity assays and did not show mutagenic activity in *Salmonella typhimurium* strains or in hamster embryo cells. Cocoamide DEA was not mutagenic in strains of *Salmonella typhimurium* when tested with or without metabolic activation.

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Miljøministeriet (Danish Environmental Protection Agency)

TRIETHYLENETETRAMINE

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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 moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin sensitizers 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.

Triethylenetetramine (TETA) is a severe irritant to skin and eyes and induces skin sensitization.

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 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).

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 fetuses (69.2%) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significant foetal 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).

9200-B Structural Epoxy Adhesive

<p>N-AMINOETHYLPIPERAZINE</p>	<p>for piperazine: Exposure to piperazine and its salts has clearly been demonstrated to cause asthma in occupational settings. No NOAEL can be estimated for respiratory sensitisation (asthma). Although the LD50 levels indicate a relatively low level of oral acute toxicity (LD50 1-5 g/kg bw), signs of neurotoxicity may appear in humans after exposure to lower doses. Based on exposure levels of up to 3.4 mg/kg/day piperazine base and a LOAEL of 110 mg/kg, there is no concern for acute toxicity In pigs, piperazine is readily absorbed from the gastrointestinal tract, and the major part of the resorbed compound is excreted as unchanged piperazine during the first 48 hours. The principal route of excretion of piperazine and its metabolites is via urine, with a minor fraction recovered from faeces (16%). In humans the kinetics of the uptake and excretion of piperazine and its metabolites with urine appear to be roughly similar to that in the pig, and the nature and extent of conversion to metabolites has not been determined. Piperazine has demonstrated a low acute toxicity (LD50 = 1-5 g/kg bw) by the oral, dermal, and subcutaneous route of administration to rodents, whereas adequate inhalation toxicity data have not been found. However, there are findings of EEG (electroencephalogram) changes in 37% of 89 children administered 90-130 mg/kg piperazine (two doses during one day), corroborated by a proposed GABA (gamma-aminobutyric acid) receptor agonism exerted by piperazine. Since clinical symptoms of neurotoxicity may occur after exposure to higher doses, a LOAEL of 110 mg/kg piperazine base for acute neurotoxicity in humans after acute exposure is proposed. Piperazine, as concentrated aqueous solution, has strongly irritating properties with regard to skin, and should be regarded as corrosive with respect to the eye. Exposure to piperazine and its salts has been demonstrated to cause allergic dermatitis as well as respiratory sensitisation in humans. As shown by the LLNA, piperazine has a sensitising potential in animals. Although piperazine is clearly sensitising, no NOAEL can be set for this effect from the present database. A NOAEL of 25 mg/kg/day of piperazine for liver toxicity in the beagle dog has been chosen after repeated exposure. A LOAEL of 30 mg/kg/day of piperazine for neurotoxicity is proposed based on documentation of (rare cases) of neurotoxicity from human clinical practice. Neurotoxicity also appears in other species (e.g., rabbits, dogs, cats, tigers, and horses), but not in rodents. For reproductive effects of piperazine, there is a NOAEL of 125 mg/kg/day for effects on fertility, i.e., reduced pregnancy index, decreased number of implantation sites, and decreased litter sizes in rats. The teratogenic properties have been investigated in rats and rabbits in adequate studies. In rabbit, such effects may be elicited at a dose level that is also toxic to the dam. The LOAEL is 94 mg/kg/day, and the NOAEL 42 mg/kg/day piperazine base (maternal and embryotoxic). In the rat study, there were decreases in body weight of both dams and offspring at the top dose (2,100 mg/kg/day piperazine base), but there were no signs of any malformations. The genotoxic properties have been investigated both <i>in vitro</i> (in the Ames test, in a nonstandard study on <i>Saccharomyces cerevisiae</i> and in Chinese hamster ovary cells) and <i>in vivo</i>, in a micronuclei assay on mice, all with negative results. There are no solid indications of a carcinogenic effect of piperazine, neither in animal studies, nor from the investigation on humans. In view of lack of genotoxic action, it appears unlikely that piperazine poses a carcinogenic risk. There seems to be an additional cancer risk due to the formation of N-mononitrosopiperazine (NPZ) from piperazine. It is possible to calculate a hypothetical additional cancer risk posed by NPZ after exposure to piperazine, but the calculation would depend on several assumptions. We conclude that there seems to be an additional cancer risk due to the formation of NPZ from piperazine, and although it is difficult to estimate, it is probably small.</p>
<p>9200-B Structural Epoxy Adhesive & ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED & C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES & TRIETHYLENETETRAMINE & N-AMINOETHYLPIPERAZINE</p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>
<p>9200-B Structural Epoxy Adhesive & TALL OIL/ TRIETHYLENETETRAMINE POLYAMIDES & TRIETHYLENETETRAMINE & N-AMINOETHYLPIPERAZINE</p>	<p>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.</p>
<p>9200-B Structural Epoxy Adhesive & ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED</p>	<p>Occupational exposures in the rubber-manufacturing industry are carcinogenic to humans (Group 1). IARC Working Groups There is sufficient evidence in humans for the carcinogenicity of occupational exposures in the rubber-manufacturing industry. Occupational exposures in the rubber-manufacturing industry cause leukaemia, lymphoma, and cancers of the urinary bladder, lung, and stomach. Also, a positive association has been observed between occupational exposures in the rubber-manufacturing industry and cancers of the prostate, oesophagus, and larynx. IARC Working Group. The multiple genetic and cytogenetic effects observed among workers employed in the rubber-manufacturing industry provide strong evidence to support genotoxicity as one mechanism for the observed increase in cancer risks. However, due to the complexity and changing nature of the exposure mixture and the potential interactions between exposures in the rubber-manufacturing industry, other mechanisms are also likely to play a role. While it is clear that exposure to some agents in the rubber-manufacturing industry has been reduced over time, the results of recent cytogenetic studies continue to raise concerns about cancer risks. The rubber-manufacturing industry has used and still uses a wide variety of substances that belong to many different chemical categories, e.g. carbon black, aromatic amines, PAH, N-nitrosamines, mineral oils, other volatile organic compounds from curing fumes, trace amounts of monomers from synthetic rubber like 1,3-butadiene, acetonitrile, styrene, vinyl chloride, ethylene oxide, etc.. For this reason, it has been difficult to relate the observed cancer hazards in the rubber-manufacturing industry to exposure to specific chemicals.</p>
<p>9200-B Structural Epoxy Adhesive & C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES & TALL OIL/ TRIETHYLENETETRAMINE POLYAMIDES</p>	<p>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. 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 Amphoteric 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.</p>

9200-B Structural Epoxy Adhesive

	<p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p>
<p>ACRYLONITRILE/ BUTADIENE COPOLYMER AMINE TERMINATED & C18 FATTY ACID DIMERS/ TETRAETHYLENEPENTAMINE POLYAMIDES & N-AMINOETHYLPIPERAZINE</p>	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p>TALL OIL/ TRIETHYLENETETRAMINE POLYAMIDES & TRIETHYLENETETRAMINE & N-AMINOETHYLPIPERAZINE</p>	<p>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.</p> <p>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.</p> <p>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</p>
<p>TRIETHYLENETETRAMINE & N-AMINOETHYLPIPERAZINE</p>	<p>The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.</p> <p>Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p>

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

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

SECTION 12 Ecological information

12.1. Toxicity

<p>9200-B Structural Epoxy Adhesive</p>	<table border="1"> <thead> <tr> <th>Endpoint</th> <th>Test Duration (hr)</th> <th>Species</th> <th>Value</th> <th>Source</th> </tr> </thead> <tbody> <tr> <td>Not Available</td> <td>Not Available</td> <td>Not Available</td> <td>Not Available</td> <td>Not Available</td> </tr> </tbody> </table>	Endpoint	Test Duration (hr)	Species	Value	Source	Not Available	Not Available	Not Available	Not Available	Not Available															
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9200-B Structural Epoxy Adhesive

C18 fatty acid dimers/ tetraethylenepentamine polyamides	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96	Fish	7.07mg/L	2
	EC50	48	Crustacea	5.18mg/L	2
	EC50	72	Algae or other aquatic plants	4.11mg/L	2
	NOEC	72	Algae or other aquatic plants	1.25mg/L	2
tall oil/ triethylenetetramine polyamides	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96	Fish	7.07mg/L	2
	EC50	48	Crustacea	7.07mg/L	2
	EC50	72	Algae or other aquatic plants	4.34mg/L	2
	EC10	72	Algae or other aquatic plants	1.78mg/L	2
	NOEC	72	Algae or other aquatic plants	0.5mg/L	2
triethylenetetramine	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48	Crustacea	31.1mg/L	1
	EC50	72	Algae or other aquatic plants	2.5mg/L	1
	NOEC	72	Algae or other aquatic plants	<2.5mg/L	1
N-aminoethylpiperazine	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	32mg/L	2
	EC50	72	Algae or other aquatic plants	495mg/L	2
	EC100	48	Crustacea	100mg/L	2
	NOEC	48	Crustacea	10mg/L	2
Legend:	<i>Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data</i>				

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and/or delayed, to the structure and/ or functioning of natural ecosystems.

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.

A team analyzed the effects of washing a number of synthetic materials at different temperatures in domestic washing machines, using different combinations of detergents, to quantify the microfibrils shed. They found that acrylic was responsible for releasing nearly 730,000 tiny synthetic particles (microplastics) per wash, five times more than polyester-cotton blend fabric, and nearly 1.5 times as many as pure polyester.

Fiber wastes has been found in sediments along shorelines around the world, predicted. They have been found to be tiny, synthetic, and all over the coastline, with the greatest concentration near sewage outflows. Of the man-made material found on the shoreline, 85% were microfibers and matched the types of material (such as nylon and acrylic) used in clothing.

Non-ionic polymers with MWs > 1,000 that do not contain reactive functional groups and are comprised of minimal low MW oligomers are estimated to display no effects at saturation (NES). These polymers display NES because the amount dissolved in water is not anticipated to reach a concentration at which adverse effects may be expressed. Guidance for the assessment of aquatic toxicity hazard results in a Low hazard designation for those materials that display NES.

For high molecular weight synthetic polymers: (according to the Sustainable Futures (SF) program (U.S. EPA 2005b; U.S. EPA 2012c) polymer assessment guidance.)

High MW polymers are expected:

- to have low vapour pressure and are not expected to undergo volatilization .
- to adsorb strongly to soil and sediment
- to be non-biodegradable (not anticipated to be assimilated by microorganisms.- therefore, biodegradation is not expected to be an important removal process. However many exceptions exist

High MW polymers are not expected to undergo removal by other degradative processes under environmental conditions

For quaternary ammonium compounds (QACs):

QACs are generally white crystalline powders. Low molecular weight QACs are very soluble in water, but slightly or not at all soluble in solvents such as ether, petrol and benzene. As the molecular weight and chain lengths increases, the solubility in polar solvents (e.g. water) decreases and the solubility in non-polar solvents increases.

Environmental fate

A major part of the QACs is discharged into wastewater and removed in the biological processes of sewage treatment plant. A 90% reduction of the QACs in the water phase of sludge has been reported and alkyl di-/ trimethyl ammonium and alkyl dimethyl benzyl ammonium compounds seem almost completely degraded in sewage sludge.

However, the aerobic and anaerobic biodegradability of QACs is not well investigated. Only sparse data are available concerning stability, solubility and biodegradability. In general, it seems that the biodegradability decreases with increasing numbers of alkyl chains: $R(CH_3)3N^+ > R_2(CH_3)2N^+ > R_3(CH_3)N^+$. Within each category the biodegradability seems inversely proportional to the alkyl chain length. Heterocyclic QACs are less degradable than the non-cyclic.

Investigations have shown that bioaccumulation of considerable dimensions will probably not take place.

Ecotoxicity:

Quaternary ammonium compounds and their polymers may be highly toxic to fish and other aquatic organisms. The toxicity of the quaternary ammoniums is known to be greatly reduced in the environment because of preferential binding to dissolved organics in surface water.

for inorganic sulfates:

Environmental fate:

Data from tap water studies with human volunteers indicate that sulfates produce a laxative effect at concentrations of 1000 - 1200 mg/litre, but no increase in diarrhoea, dehydration or weight loss. The presence of sulfate in drinking-water can also result in a noticeable taste; the lowest taste threshold concentration for sulfate is approximately 250 mg/litre as the sodium salt. Sulfate may also contribute to the corrosion of distribution systems. No health-based guideline value for sulfate in drinking water is proposed. However, there is an increasing likelihood of complaints arising from a noticeable taste as concentrations in water increase above 500 mg/litre.

Sulfates are removed from the air by both dry and wet deposition processes. Wet deposition processes including rain-out (a process that occurs within the clouds) and washout (removal by precipitation below the clouds) contribute to the removal of sulfate from the atmosphere.

9200-B Structural Epoxy Adhesive

In soil, the inorganic sulfates can adsorb to soil particles or leach into surface water and groundwater. Sulfates can be taken up by plants and be incorporated into the parenchyma of the plant.

Sulfate in water can also be reduced by sulfate bacteria (*Thiobacilli*) which use them as a source of energy.

In anaerobic environments sulfate is biologically reduced to (hydrogen) sulfide by sulfate reducing bacteria, or incorporated into living organisms as source of sulfur, and thereby included in the sulfur cycle. Sodium sulfate is not reactive in aqueous solution at room temperature. Sodium sulfate will completely dissolve, ionise and distribute across the entire planetary 'aquasphere'. Some sulfates may eventually be deposited, the majority of sulfates participate in the sulfur cycle in which natural and industrial sodium sulfate are not distinguishable.

The BCF of sodium sulfate is very low and therefore significant bioconcentration is not expected. Sodium and sulfate ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. However some plants (e.g. corn and *Kochia Scoparia*), are capable of accumulating sulfate to concentrations that are potentially toxic to ruminants.

Ecotoxicity:

For sulfate in general:

Fish LC50: toxic from 7000 mg/l

Bacteria: toxic from 2500 mg/l

Algae were shown to be the most sensitive to sodium sulfate; EC50 120 h = 1,900 mg/l. For invertebrates (*Daphnia magna*) the EC50 48 h = 4,580 mg/l and fish appeared to be the least sensitive with a LC50 96h = 7,960 mg/l for *Pimephales promelas*. Activated sludge showed a very low sensitivity to sodium sulfate. There was no effect up to 8 g/l. Sodium sulfate is not very toxic to terrestrial plants. *Picea banksiana* was the most sensitive species, an effect was seen at 1.4 g/l. Sediment dwelling organisms were not very sensitive either, with an LC50 96h = 660 mg/l for *Trycorythus sp.* Overall it can be concluded that sodium sulfate has no acute adverse effect on aquatic and sediment dwelling organisms. Toxicity to terrestrial plants is also low.

No data were found for long term toxicity. The acute studies all show a toxicity of sodium sulfate higher than 100 mg/l, no bioaccumulation is expected,

For barium and its compounds::

Environmental fate:

The length of time that barium will last in air, land, water, or sediments following release of barium into these media depends on the form of barium released. Barium compounds that do not dissolve well in water, such as barium sulfate and barium carbonate, can persist for a long time in the environment. Barium compounds, such as barium chloride, barium nitrate, or barium hydroxide, that dissolve easily in water usually do not last in these forms for a long time in the environment. The barium in these compounds that is dissolved in water quickly combines with sulfate or carbonate that are naturally found in water and become the longer lasting forms (barium sulfate and barium carbonate).

Under natural conditions, barium is stable in the +2 valence state and is found primarily in the form of inorganic complexes. Conditions such as pH, Eh (oxidation-reduction potential), cation exchange capacity, and the presence of sulfate, carbonate, and metal oxides (e.g., oxides of aluminum, manganese, silicon, and titanium) will affect the partitioning of barium and its compounds in the environment. The major features of the biogeochemical cycle of barium include wet and dry deposition to land and surface water, leaching from geological formations to groundwater, adsorption to soil and sediment particulates, and biomagnification in terrestrial and aquatic food chains.

Barium is a highly reactive metal that occurs naturally only in a combined state. The element is released to environmental media by both natural processes and anthropogenic sources.

The general population is exposed to barium through consumption of drinking water and foods, usually at low levels. Most barium released to the environment from industrial sources is in forms that do not become widely dispersed. In the atmosphere, barium is likely to be present in particulate form. Although chemical reactions may cause changes in speciation of barium in air, the main mechanisms for the removal of barium compounds from the atmosphere are likely to be wet and dry deposition.

In aquatic media, barium is likely to precipitate out of solution as an insoluble salt (i.e., as BaSO₄ or BaCO₃). Waterborne barium may also adsorb to suspended particulate matter through the formation of ion pairs with natural anions such as bicarbonate or sulfate in the matter.

Precipitation of barium sulfate salts is accelerated when rivers enter the ocean because of the high sulfate content (905 mg/L) in the ocean. It is estimated that only 0.006% of the total barium input into oceans from freshwater sources remains in solution. Sedimentation of suspended solids removes a large portion of the barium content from surface waters. There is evidence to suggest that the precipitation of barium from the surface of fresh and marine waters occurs, in part, as the result of the barite crystal formation in microorganisms.

Barium in sediments is found largely in the form of barium sulfate (barite). Coarse silt sediment in a turbulent environment will often grind and cleave the barium sulfate from the sediment particles leaving a buildup of dense barites. Estimated soil:water distribution coefficients (K_d) (i.e., the ratio of the quantity of barium sorbed per gram of sorbent to the concentration of barium remaining in solution at equilibrium) range from 200 to 2,800 for sediments and sandy loam soils. The uptake of barium by fish and marine organisms is also an important removal mechanism. Barium levels in sea water range from 2 to 63 µg/L with a mean concentration of about 13 µg/L. Barium was found to bioconcentrate in marine plants by a factor of 400-4,000 times the level present in the water. Bioconcentration factors in marine animals, plankton, and brown algae of 100, 120, and 260, respectively, have been reported. In freshwater, a bioconcentration factor of 129 was estimated in fish where the barium in water was 0.07 mg/L.

Barium added to soils (e.g., from the land farming of waste drilling muds) may either be taken up by vegetation or transported through soil with precipitation. Relative to the amount of barium found in soils, little is typically bioconcentrated by plants. For example, a bioconcentration factor of 0.4 has been estimated for plants in a Virginia floodplain with a barium soil concentration of 104.2 mg/kg. However, there are some plants, such as legumes, forage plants, Brazil nuts, and mushrooms that accumulate barium. Bioconcentration factors from 2 to 20 have been reported for tomatoes and soybeans.

Barium is not very mobile in most soil systems, due to the formation of water-insoluble salts and an inability of the barium ion to form soluble complexes with fulvic and humic acids.

The rate of transportation of barium in soil is dependent on the characteristics of the soil material. Soil properties that influence the transportation of barium to groundwater are cation exchange capacity, calcium carbonate (CaCO₃) content and pH. In soil with a high cation exchange capacity (e.g., fine textured mineral soils or soils with high organic matter content), barium mobility will be limited by adsorption. High CaCO₃ content limits mobility by precipitation of the element as BaCO₃. Barium will also precipitate as barium sulfate in the presence of sulfate ions. Barium is more mobile and is more likely to be leached from soils in the presence of chloride due to the high solubility of barium chloride as compared to other chemical forms of barium. Barium may become more mobile in soils under acid conditions as barium in water-insoluble salts, such as barium sulfate and carbonate, becomes more soluble. Barium complexes with fatty acids (e.g., in acidic landfill leachate) will be much more mobile in the soil due to the lower charge of these complexes and subsequent reduction in adsorption capacity.

The alkali metal cyanides (and other metal cyanides) are very soluble in water. As a result, they readily dissociate into their respective anions and cations when released into water.

Depending on the pH of the water, the resulting cyanide ion may then form hydrogen cyanide or react with various metals in natural water. The proportion of hydrogen cyanide formed from soluble cyanides increases as the water pH decreases. At pH <7, >99% of the cyanide ions in water are converted to hydrogen cyanide. As the pH increases, cyanide ions in the water may form complex metalocyanides in the presence of excess cyanides; however, if metals are prevalent, simple metal cyanides are formed. Volatilization is the dominant mechanism for the removal of free cyanide. At pH >9.2, most of the free cyanide should exist as HCN, a volatile form of cyanide. Wide variations in the rate of volatilization are expected since this process is affected by a number of parameters such as temperature, pH, wind speed, and cyanide concentration. Volatilization of free cyanide from concentrated solutions is most effective under conditions of high temperatures, high dissolved oxygen levels, and at increased concentrations of atmospheric carbon dioxide.

Unlike water-soluble alkali metal cyanides, insoluble metal cyanides are not expected to degrade to hydrogen cyanide. Cyanide occurs most commonly as hydrogen cyanide in water, although it can also occur as the cyanide ion, alkali and alkaline earth metal cyanides (potassium cyanide, sodium cyanide, calcium cyanide), relatively stable metalocyanide complexes (ferricyanide complex [Fe(CN)₆]³⁻), moderately stable metalocyanide complexes (complex nickel and copper cyanide), or easily decomposable metalocyanide complexes (zinc cyanide [Zn(CN)₂], cadmium cyanide [Cd(CN)₂]). Oxidation, hydrolysis, and photolysis (photodegradation) are the three predominant chemical processes that may cause loss of simple cyanides in aquatic media.

Certain cyanides are oxidised to isocyanates by strong oxidising agents; the isocyanates may be further hydrolysed to ammonia and carbon dioxide. However, it has not yet been determined whether such oxidation and subsequent hydrolysis of isocyanate is a significant fate process in natural waters known to contain peroxy radicals. In water, hydrogen cyanide and cyanide ion exist in equilibrium with their relative concentrations primarily dependent on pH and temperature. At pH <8, >93% of the free cyanide in water will exist as undissociated hydrogen cyanide. Hydrogen cyanide can be hydrolysed to formamide, which is subsequently hydrolysed to ammonium and formate ions. However, the relatively slow rates of hydrolysis reported for hydrogen cyanide in acidic solution and of cyanides under alkaline conditions indicate that hydrolysis is not competitive with volatilisation and biodegradation for removal of free cyanide from ambient waters. At pH <9.2, most of the free cyanide in solution should exist as hydrogen cyanide, a volatile cyanide form. On the basis of Henry's law constant and the volatility characteristics associated with various ranges of Henry's law constant, volatilization is a significant and probably dominant fate process for hydrogen cyanide in surface water. The most common alkali metal cyanides (e.g., sodium and potassium cyanide) may also be lost from surface water primarily through volatilization; whereas, the sparingly soluble metal cyanides such as copper (I) cyanide are removed from water predominantly by sedimentation and biodegradation. Because volatilisation is not an important fate process for cyanide in groundwater, cyanide would be expected to persist for considerably longer periods of time in underground aquifers than in surface water.

The significance of photolysis in the fate of cyanides in water has not been fully investigated. Hydrogen cyanide and cyanide ions in aqueous solution have been found to be very resistant to photolysis by natural sunlight, except under heterogeneous photocatalytic conditions. Photocatalytic oxidation may not be significant in natural waters, however, because of significant light reduction at increasingly greater depths. In clear water or at water surfaces, some metalocyanides, such as ferrocyanides and ferricyanides, may decompose to the cyanide ion by photodissociation and subsequently form hydrogen cyanide.

Biodegradation is an important transformation process for cyanide in natural surface waters, and is dependent on such factors as cyanide concentrations, pH, temperature, availability of nutrients, and acclimation of microbes. Although the cyanide ion is toxic to microorganisms at concentrations as low as 5-10 mg/L, acclimation increases tolerance to this compound. Mixed microorganisms in sewage sludge or activated sludge acclimated to cyanide also significantly biodegrade concentrations <=100 mg/L of most simple and complex

9200-B Structural Epoxy Adhesive

cyanides. It is known that there is a natural attenuation of the cyanide ion and thiocyanide concentrations in waste waters, for example those obtained gold mill tails, that is due the acclimation of indigenous microflora in the tailings. A number of microorganisms have been identified that are capable of uptake, conversion, sorption, and/or precipitation of the cyanide ion, cyanate, and thiocyanate, including species of the genera, *Actinomyces*, *Alcaligenes*, *Arthrobacter*, *Bacillus*, *Micrococcus*, *Neisseria*, *Paracoccus*, *Pseudomonas*, and *Thiobacillus*. Some of these species, for example *Pseudomonas*, are capable of using the cyanide ion and thiocyanate as the sole source of carbon and nitrogen and therefore, are particularly effective at cyanide degradation. In fact, *Pseudomonas* is the basis of commercial applications for degrading the cyanide ion to ammonia and carbonate in waste waters generated in mining operations that use the cyanide ion to leach gold and other precious metals for low-grade ores. Sulfur transferases such as rhodanese are involved in substitution reactions that result in the conversion of the cyanide ion to the less toxic thiocyanate, whereas pyridoxal phosphate enzymes are involved in substitution/addition reactions that result in production of nitrile derivatives of α -amino acids. These organic nitriles may then be ultimately degraded via enzyme catalysed hydrolysis to either the corresponding amino acid and ammonia or the carboxylic acid and ammonia. The cyanide hydratase and cyanidase enzymes catalyse the hydrolysis of the cyanide ion to formamide or formic acid and ammonia, respectively. In soil, cyanide present at low concentrations would biodegrade under aerobic conditions with the initial formation of ammonia, which would be converted to nitrite and nitrate in the presence of nitrifying bacteria. Under anaerobic conditions, the cyanides ion will denitrify to gaseous nitrogen. Upper limits of 200 and 2 ppm (mg/kg CN⁻), respectively, have been reported for uninhibited aerobic and anaerobic biodegradation of cyanide in soil; however, these limits have not been confirmed in other studies. Cyanide ions in soil are not involved in oxidation-reduction reactions but may undergo complexation reactions with metal ions in soil.

Cyanides are sorbed by various natural media, including clays, biological solids and sediments. Hydrogen cyanide and the alkali metal cyanides are not likely to be strongly sorbed onto sediments and suspended solids because of their high water solubilities. Soluble metal cyanides may show somewhat stronger sorption than hydrogen cyanide, with the extent of sorption increasing with decreasing pH and increasing iron oxide, clay, and organic material contents of sediment and suspended solids. However, sorption is probably insignificant even for metal cyanides when compared to volatilisation and biodegradation. Cyanides are fairly mobile in soil. Mobility is lowest in soils with low pH and high concentrations of free iron oxides, positively charged particles, and clays (e.g., chlorite, kaolin, gibbsite), and highest in soils with high pH, high concentrations of free CaCO₃ and negatively charged particles, and low clay content. Although cyanide has a low soil sorption capability, it is usually not detected in groundwater, probably because of fixation by trace metals through complexation or transformation by soil microorganisms. In soils where cyanide levels are high enough to be toxic to microorganisms (i.e., landfills, spills), this compound may leach into groundwater. Leaching of cyanide into a shallow aquifer has been demonstrated. Volatilisation of hydrogen cyanide would be a significant loss mechanism for cyanides from soil surfaces at a pH <9.2.

Most cyanide in the atmosphere exists almost entirely as hydrogen cyanide gas, although small amounts of metal cyanides may be present as particulate matter in the air. Hydrogen cyanide is very resistant to photolysis at wavelengths of normal sunlight. The most important reaction of hydrogen cyanide in air is the reaction with photochemically-generated hydroxyl radicals and subsequent rapid oxidation to carbon monoxide (CO) and nitric oxide (NO); photolysis and reaction with ozone are not important transformation processes, and reaction with singlet oxygen is not a significant transformation process except at stratospheric altitudes where singlet oxygen is present in significant concentrations. The rate of hydroxyl radical reaction with hydrogen cyanide in the atmosphere depends on the altitude, and the rate of the reaction is at least an order of magnitude faster at lower tropospheric altitudes (0–8 km) than at upper tropospheric altitudes (10–12 km). Based on a reaction rate constant of 3x10⁻¹⁴ cm³/(molecule-sec) at 25 °C and assuming an average hydroxyl radical concentration of 5x10⁵ molecules/cm³, the residence time for the reaction of hydrogen cyanide vapor with hydroxyl radicals in the atmosphere is approximately 2 years.

There is some evidence that certain metal cyanide complexes bioaccumulate in aquatic organisms. Fish from water with soluble silver and copper cyanide complexes were found to have metal cyanides in their tissues at concentrations ranging up to 168 and 304 µg/g, respectively (wet or dry weight not specified). It is difficult to evaluate the toxicologic significance of bioaccumulation of metal cyanide complexes because these compounds are much less toxic than soluble hydrogen cyanide, sodium cyanide, or potassium cyanide.

There is no evidence of biomagnification of cyanides in the food chain. Accumulation of cyanide in food webs is not expected, considering the rapid detoxification of cyanide by most species and the lethal effects of large doses of cyanide.

For butadiene:

Kow: 1.99

Koc : 72-228

Half-life (hr) air : 4.9

Henry's Pa m³ /mol: 2.57

Henry's atm m³ /mol: 7.24E-02

BCF : 19.1

Environmental fate:

The high volatility of this compound suggests that it will partition predominantly to the atmospheric compartment, where it is not expected to be adsorbed to particulate matter to any significant extent.

Terrestrial Fate: If spilled on land, 1,3-butadiene will predominately volatilise very rapidly due to its very low boiling point. Dissolved in water, it may leach through soil into ground water due to its high water solubility and low estimated soil adsorption coefficient. It will not appreciably hydrolyse but may be subject to biodegradation based on screening tests. 1,3-Butadiene is expected to volatilize rapidly from either moist or dry soil to the atmosphere. This follows from the estimated lack of any appreciable adsorption to soil, and consideration of 1,3-butadiene's calculated Henry's law constant for moist soil or its vapor pressure, 2,100 mm Hg at 25 °C, for dry soil. Both values suggest a rapid rate of volatilisation from their respective media. The calculated soil adsorption coefficient of 288 suggests that 1,3-butadiene may display moderate mobility in soil. However, the expected rapid rate of volatilisation and the possibility of rapid degradation in soil suggest that there is little potential for 1,3-butadiene to leach into groundwater. Methane-utilizing bacteria isolated from the soil of an oil refinery epoxidised 1,3-butadiene under aerobic conditions.

Aquatic Fate: When released into water, 1,3-butadiene will volatilise rapidly with a half-life estimated to be several hours. It will not hydrolyse appreciably, but may be subject to biodegradation, based on screening tests.

Atmospheric Fate: Butadiene is a reactive, electron-rich chemical that is expected to undergo rapid reactions with the electrophilic oxidants typically present in the atmosphere: ozone, photochemically produced hydroxyl radicals, nitrate radicals, and molecular oxygen. Among these, the most rapid reaction in the atmosphere is with photochemically produced hydroxyl radicals. The atmospheric destruction of 1,3-butadiene by photo-initiated processes has been established empirically by early studies. There are four gas-phase pathways that can destroy 1,3-butadiene in the atmosphere. Depending on local conditions, any one or all of these reactions may occur. Destruction of atmospheric 1,3-butadiene by the gas-phase reaction with photochemically produced hydroxyl radicals is expected to be the dominant photo-initiated pathway. Destruction by nitrate radicals is expected to be a significant night-time process in urban areas.

Reaction with hydroxyl radicals is the dominant removal mechanism, with an estimated half-life of several hours. Reaction with ozone and nitrate radicals may also contribute to the degradation of the chemical. Polluted urban atmospheres increase the rate of degradation somewhat during daylight hours as suggested by the detection of the highest atmospheric levels of the chemical in the early morning hours. Acetaldehyde and acrolein have been identified as products of photooxidation. Washout may contribute to removal of 1,3-butadiene from the atmosphere; however, evaporation from the rain may be rapid and the compound returned to the atmosphere relatively quickly unless it leaches into the soil.

Biodegradation: No data concerning the biodegradation of 1,3-butadiene in natural systems could be found in the literature. 1,3-Butadiene was listed in a group of chemicals which should be biodegraded by biological sewage treatment, as long as suitable acclimatization is achieved. Screening tests suggest that 1,3-butadiene may be biodegradable in the environment with 1,2-epoxybutene being a potential product.

Soil Adsorption/Mobility: The range of estimated adsorption coefficients for 1,3-butadiene from the soils and sediments is 72-228 based on its octanol/water partition coefficient or its water solubility and would therefore not be expected to appreciably adsorb in soils and sediments.

Volatilization from Water/Soil: Using the Henry's Law constant, the estimated half-life for evaporation of 1,3-butadiene from a river 1 m deep with a 1 m/sec current and a 3 m/sec wind is 3.8 hours. Due to its low boiling point, 1,3-butadiene would be expected to rapidly evaporate from soils.

Ecotoxicity:

Fish LC50 (24 h): 71.5 mg/L

1,3-Butadiene is moderately toxic to aquatic life in the short term and slightly toxic in the long term. There is not enough information to predict additional short or long-term effects of 1,3-butadiene on plants, birds, or other animals. 1,3-Butadiene is not expected to accumulate in fish. Animal studies have reported development effects such as skeletal abnormalities and decreased foetal weights, and reproductive effects, including an increased incidence of shrinkage of the ovaries and testicles. Animal studies have also reported tumours at a variety of sites from inhalation of 1,3-butadiene.

Soil Guidelines: Dutch Criteria:

free cyanide: 1 mg/kg (target)

20 mg/kg (intervention)

complex cyanide (pH 5): 5 mg/kg (target)

50 mg/kg (intervention)

Air Quality Standards: no safe guidelines recommended due to carcinogenic properties.

Prevent, by any means available, spillage from entering drains or water courses.

9200-B Structural Epoxy Adhesive

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
triethylenetetramine	LOW	LOW
N-aminoethylpiperazine	HIGH	HIGH

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
triethylenetetramine	LOW (BCF = 5)
N-aminoethylpiperazine	LOW (LogKOW = -1.5677)

12.4. Mobility in soil

Ingredient	Mobility
triethylenetetramine	LOW (KOC = 309.9)
N-aminoethylpiperazine	LOW (KOC = 171.7)

12.5. Results of PBT and vPvB assessment

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

12.6. Other adverse effects

No data available


SECTION 13 Disposal considerations

13.1. Waste treatment methods

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

SECTION 14 Transport information

Labels Required

		Limited Quantity: For 9200-25ML, 9200-50ML, 9200-1.7L
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Land transport (ADR-RID)

14.1. UN number	2735
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9200-B Structural Epoxy Adhesive

14.2. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains C18 fatty acid dimers/ tetraethylenepentamine polyamides and triethylenetetramine)	
14.3. Transport hazard class(es)	Class	8
	Subrisk	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Hazard identification (Kemler)	80
	Classification code	C7
	Hazard Label	8
	Special provisions	274
	Limited quantity	1 L
	Tunnel Restriction Code	2 (E)

Air transport (ICAO-IATA / DGR)

14.1. UN number	2735	
14.2. UN proper shipping name	Polyamines, liquid, corrosive, n.o.s. * (contains C18 fatty acid dimers/ tetraethylenepentamine polyamides and triethylenetetramine); Amines, liquid, corrosive, n.o.s. * (contains C18 fatty acid dimers/ tetraethylenepentamine polyamides and triethylenetetramine)	
14.3. Transport hazard class(es)	ICAO/IATA Class	8
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	8L
14.4. Packing group	II	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	A3 A803
	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. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains C18 fatty acid dimers/ tetraethylenepentamine polyamides and triethylenetetramine)	
14.3. Transport hazard class(es)	IMDG Class	8
	IMDG Subrisk	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Marine Pollutant	
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. UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains C18 fatty acid dimers/ tetraethylenepentamine polyamides and triethylenetetramine)	
14.3. Transport hazard class(es)	8	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Classification code	C7
	Special provisions	274
	Limited quantity	1 L
	Equipment required	PP, EP
	Fire cones number	0

9200-B Structural Epoxy Adhesive

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

Not Applicable

SECTION 15 Regulatory information

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

acrylonitrile/ butadiene copolymer amine terminated is found on the following regulatory lists

Not Applicable

barium sulfate is found on the following regulatory lists

Europe EC Inventory

UK Workplace Exposure Limits (WELs)

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

C18 fatty acid dimers/ tetraethylenepentamine polyamides is found on the following regulatory lists

Not Applicable

tall oil/ triethylenetetramine polyamides is found on the following regulatory lists

Europe EC Inventory

triethylenetetramine is found on the following regulatory lists

Europe EC Inventory

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

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

N-aminoethylpiperazine is found on the following regulatory lists

Europe EC Inventory

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

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

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

15.2. Chemical safety assessment

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (acrylonitrile/ butadiene copolymer amine terminated; barium sulfate; C18 fatty acid dimers/ tetraethylenepentamine polyamides; tall oil/ triethylenetetramine polyamides; triethylenetetramine; N-aminoethylpiperazine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (acrylonitrile/ butadiene copolymer amine terminated; C18 fatty acid dimers/ tetraethylenepentamine polyamides)
Japan - ENCS	No (acrylonitrile/ butadiene copolymer amine terminated; tall oil/ triethylenetetramine polyamides)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (acrylonitrile/ butadiene copolymer amine terminated)
Vietnam - NCI	Yes
Russia - ARIPS	No (acrylonitrile/ butadiene copolymer amine terminated; C18 fatty acid dimers/ tetraethylenepentamine polyamides; tall oil/ triethylenetetramine polyamides)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 Other information

Revision Date	21/12/2020
Initial Date	31/03/2016

Full text Risk and Hazard codes

H302	Harmful if swallowed.
H302+H332	Harmful if swallowed or if inhaled.
H312	Harmful in contact with skin.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H332	Harmful if inhaled.

Continued...

9200-B Structural Epoxy Adhesive

H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H412	Harmful to aquatic life with long lasting effects.

SDS Version Summary

Version	Issue Date	Sections Updated
2.7.1.1.1	21/12/2020	Chronic Health, Classification, Physical Properties

Other information

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

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

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

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average

PC—STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit.

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

Reason for Change

A-2.00 - Update to the classification.