



ARDEX EG15 Resin Part B Improved Formula

ARDEX (Ardex Australia)

Chemwatch Hazard Alert Code: 4

Chemwatch: 5646-60

Version No: 2.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: 22/11/2023

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L.GHS.AUS.EN.E

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

Product Identifier

| | |
|-------------------------------|--|
| Product name | ARDEX EG15 Resin Part B Improved Formula |
| Chemical Name | Not Applicable |
| Synonyms | Not Available |
| Proper shipping name | CORROSIVE LIQUID, N.O.S. (contains isophorone diamine and tall oil/ tetraethylenepentamine polyamides) |
| Chemical formula | Not Applicable |
| Other means of identification | Not Available |

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

| | |
|--------------------------|--|
| Relevant identified uses | Epoxy hardener for epoxy grout. Use according to manufacturer's directions. |
|--------------------------|--|

Details of the manufacturer or supplier of the safety data sheet

| | |
|-------------------------|--|
| Registered company name | ARDEX (Ardex Australia) |
| Address | 2 Buda Way Kempers Creek NSW 2147 Australia |
| Telephone | 1300 788 780 |
| Fax | 1300 780 102 |
| Website | www.ardexaustralia.com |
| Email | technical.services@ardexaustralia.com |

Emergency telephone number

| | |
|-----------------------------------|---------------------------------|
| Association / Organisation | ARDEX (ARDEX Australia) |
| Emergency telephone numbers | 1800 224 070 (Mon-Fri, 9am-5pm) |
| Other emergency telephone numbers | Not Available |

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

| | |
|--------------------|---|
| Poisons Schedule | S5 |
| Classification [1] | Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1A, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 2 |
| Legend: | 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI |

Label elements

| | |
|---------------------|--------|
| Hazard pictogram(s) | |
| Signal word | Danger |

Hazard statement(s)

| | |
|--------|---|
| H302 | Harmful if swallowed. |
| H312 | Harmful in contact with skin. |
| H314 | Causes severe skin burns and eye damage. |
| H317 | May cause an allergic skin reaction. |
| H335 | May cause respiratory irritation. |
| H336 | May cause drowsiness or dizziness. |
| H360Df | May damage the unborn child. Suspected of damaging fertility. |
| H411 | Toxic to aquatic life with long lasting effects. |
| AUH019 | May form explosive peroxides. |

Precautionary statement(s) Prevention

| | |
|------|--|
| P201 | Obtain special instructions before use. |
| P260 | Do not breathe mist/vapours/spray. |
| P264 | Wash all exposed external body areas thoroughly after handling. |
| P271 | Use only outdoors or in a well-ventilated area. |
| P280 | Wear protective gloves, protective clothing, eye protection and face protection. |
| P270 | Do not eat, drink or smoke when using this product. |
| 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. If more than 15 mins from Doctor, INDUCE VOMITING (if conscious). |
| 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. |
| P302+P352 | IF ON SKIN: Wash with plenty of water. |
| 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. |
| P301+P312 | IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell. |
| P304+P340 | IF INHALED: Remove person to fresh air and keep comfortable for breathing. |

Precautionary statement(s) Storage

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|-----------|--|
| P405 | Store locked up. |
| P403+P233 | Store in a well-ventilated place. Keep container tightly closed. |

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

| CAS No | %[weight] | Name |
|----------------|--|--|
| 100-51-6 | 20-50 | <u>benzyl alcohol</u> |
| 2855-13-2 | 10-40 | <u>isophorone diamine</u> |
| 135108-88-2 | 10-20 | <u>formaldehyde/ benzenamine, hydrogenated</u> |
| 68953-36-6 | 10-20 | <u>tall oil/ tetraethylenepentamine polyamides</u> |
| 25620-58-0 | 5-20 | <u>trimethylhexamethylene diamine</u> |
| 912342-92-8 | 5-20 | <u>cycloaliphatic amine</u> |
| 112-57-2 | 1-5 | <u>tetraethylenepentamine</u> |
| Legend: | 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available | |

SECTION 4 First aid measures**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. |
|--------------------|---|

Continued...

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| | |
|---------------------|---|
| | <ul style="list-style-type: none"> ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| 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. |
| 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. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay. ▶ 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> |
| 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. |

Indication of any immediate medical attention and special treatment needed

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]

Treat symptomatically.

SECTION 5 Firefighting measures**Extinguishing media**

- ▶ Foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.
- ▶ Water spray or fog - Large fires only.

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

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. |
| Fire/Explosion Hazard | <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) aldehydes nitrogen oxides (NO_x)</p> |

| | |
|---------|--|
| | other pyrolysis products typical of burning organic material. May emit corrosive fumes. |
| HAZCHEM | 2X |

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

| | |
|--------------|--|
| Minor Spills | <p>Slippery when spilt.</p> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal. |
| Major Spills | <p>Slippery when spilt.</p> <ul style="list-style-type: none"> ▶ 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. |

Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

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|-------------------|---|
| Safe handling | <ul style="list-style-type: none"> ▶ DO NOT USE brass or copper containers / stirrers ▶ DO NOT allow clothing wet with material to stay in contact with skin <p>The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example.</p> <p>Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.</p> <ul style="list-style-type: none"> ▶ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date. ▶ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▶ Unopened containers received from the supplier should be safe to store for 18 months. ▶ Opened containers should not be stored for more than 12 months. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ WARNING: To avoid violent reaction, ALWAYS add material to water and NEVER water to material. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. |
| Other information | <ul style="list-style-type: none"> ▶ DO NOT store near acids, or oxidising agents ▶ No smoking, naked lights, heat or ignition sources. ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. |

Conditions for safe storage, including any incompatibilities

| | |
|--------------------|--|
| Suitable container | <ul style="list-style-type: none"> · Liquid epoxy curing agents will corrode certain common structural metals. · If slight colouration of the curing agent is acceptable, storage tanks may be made of carbon steel or black iron, provided they are free of rust and mill scale. However, if the amine is stored in such tanks for three or four months or longer, colour may develop due to iron contamination. If iron contamination cannot be tolerated, tanks constructed of types 304 or 316 stainless steel should be used. (Note: Because they are quickly corroded by amines, do not use aluminum, copper, copper alloys, brass, or bronze in tanks or lines.) · Although horizontal tanks may be used, vertical tanks are suggested because they are usually more economical to install, occupy less space, and provide more accurate tank gauging. <p>(Note: In accordance with National Fire Protection Association Rule 30.17, item 2-1.31 (b), a vertical tank, designed to American Petroleum Institute (API) Standard 650, is suggested for curing agents. European equivalents include flat bottomed tank design DIN 4119, Parts 1 and</p> |
|--------------------|--|

2, and horizontal, vertical DIN 6600-6625.)

To ensure safe and orderly delivery, the capacity of the storage tank should be large enough to hold the amount of epoxy curing agent normally shipped in a maximum capacity tank car or tank truck, plus additional working inventory. Also, consider oversizing the tank sufficiently to create a space to accommodate bubbles which may be created by the gas flow used to clear the piping. (Note: If a suction heater is used in the tank, additional capacity should be allowed for the heel.) Also, when calculating tank size, allow sufficient freeboard for liquid expansion while heating. A suitable foundation is required for all curing agent storage tanks.

Temperatures, necessary for transfer and ease of handling of liquid epoxy amine hardeners, generally can be achieved with little or no heating, especially if the storage tank is located in a warm or temperate climate, is well insulated, or is stored indoors in a heated or insulated building. However, product stored outdoors in an uninsulated tank, especially in cold climates, may require some degree of heating. These temperatures can generally be achieved by using some combination of heat tracing and insulated jacketing or external low-pressure steam or hot water heaters, heating coils or a suction heater.

In the design of heaters for storage tanks, all factors pertinent to a particular application must be considered. These include: desired rate of tank warm-up; heat losses to the atmosphere; temperature and capacity of heat source available; amount of agitation available for tank contents; the geometry and space limitations of the proposed installation; etc. Internal pipe coil heaters may be appropriate where quick heat-up is not necessary and where heat losses are small or limited by tank insulation. The coils should be located near the bottom of the tank and should be sized to give sufficient heat transfer surface, both to provide the required heat-up rate and to take care of heat losses to the atmosphere. Uniform temperature of the tank contents can be achieved by using the tank pump to circulate the contents over the tank heating coil.

Finally, for quick tank heat-up and good mixing within the tank, consider the use of tank eductors, particularly for mixing when starting with cold material. Proper sizing of the pump and the eductors is necessary to ensure sufficient velocity through the eductors. The more economical eductor models are 1.5 inches (38 mm) in size. The recycle flow is usually varied to give sufficient volume to effectively mix the material in the tank. The vendor can usually supply curves of pressure drop and pumping capacity. If an agitator is required, a top-entering agitator should be used. In all heated tank systems, it is recommended that the design include provisions to keep pipe coils or suction heater coils submerged at all times. Heaters should be mounted as low in the tank as possible. Again, in areas where temperatures are not expected to drop below freezing for extended periods, one inch of dense glass-fiber insulation is generally sufficient; however, in colder climates, heat tracing of the lines and at least two inches of insulation should be used. Also, the insulation should be covered with an aluminum weather barrier.

- ▶ **DO NOT use aluminium or galvanised containers**
- ▶ Lined metal can, lined metal pail/ can.
- ▶ Plastic pail.
- ▶ Polyliner drum.
- ▶ Packing as recommended by manufacturer.
- ▶ Check all containers are clearly labelled and free from leaks.

For low viscosity materials

- ▶ Drums and jerricans must be of the non-removable head type.
- ▶ Where a can is to be used as an inner package, the can must have a screwed enclosure.

For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):

- ▶ Removable head packaging;
- ▶ Cans with friction closures and
- ▶ low pressure tubes and cartridges

may be used.

-

Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.

Storage incompatibility

- ▶ Avoid strong acids, bases.
- ▶ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air.
- ▶ Avoid contact with copper, aluminium and their alloys.
- ▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

| Ingredient | Original IDLH | Revised IDLH |
|---|---------------|---------------|
| benzyl alcohol | Not Available | Not Available |
| isophorone diamine | Not Available | Not Available |
| formaldehyde/ benzenamine, hydrogenated | Not Available | Not Available |
| tall oil/ tetraethylenepentamine polyamides | Not Available | Not Available |
| trimethylhexamethylene diamine | Not Available | Not Available |
| cycloaliphatic amine | Not Available | Not Available |
| tetraethylenepentamine | Not Available | Not Available |

Occupational Exposure Banding

| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|---|-----------------------------------|----------------------------------|
| benzyl alcohol | E | ≤ 0.1 ppm |
| isophorone diamine | D | > 0.1 to ≤ 1 ppm |
| formaldehyde/ benzenamine, hydrogenated | E | ≤ 0.1 ppm |
| tall oil/ tetraethylenepentamine polyamides | E | ≤ 0.1 ppm |
| trimethylhexamethylene diamine | E | ≤ 0.1 ppm |
| cycloaliphatic amine | 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...

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| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|------------------------|---|----------------------------------|
| tetraethylenepentamine | E | ≤ 0.1 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

Exposure controls

| Appropriate engineering controls | <p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>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.</p> <p>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.</p> <table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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> <p>CARE: Use of a quantity of this material in confined space or poorly ventilated area, where rapid build up of concentrated atmosphere may occur, could require increased ventilation and/or protective gear</p> | 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.) | 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 |
|---|--|----------------------|------------|--|------------------------------|---|----------------------------|--|----------------------------|--|------------------------------|------------------------|------------------------|---|---------------------------------|--|----------------------------------|----------------------------------|-------------------------------|---|----------------------------------|
| 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.) | | | | | | | | | | | | | | | | | | | | |
| 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 | | | | | | | | | | | | | | | | | | | | |
| Individual protection measures, such as personal protective equipment |  | | | | | | | | | | | | | | | | | | | | |
| Eye and face protection | <p>Epoxy amine hardeners may produce eye discomfort, irritation, or even injury; thus, all eye contact with either the liquid or solid products (including vapours, mists, aerosols, or dusts) should be strictly avoided through the use of appropriate eye protection, including chemical workers goggles (or monogoggles), a face shield that allows the use of chemical workers goggles, or a full-face respirator, depending on the degree of potential exposure.</p> <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. [AS/NZS 1337.1, EN166 or national equivalent] ▶ 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]. | | | | | | | | | | | | | | | | | | | | |
| 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>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>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> | | | | | | | | | | | | | | | | | | | | |

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

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

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

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

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

· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

· Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

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

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

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

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

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

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

When handling liquid-grade epoxy resins wear chemically protective gloves, boots and aprons.

The performance, based on breakthrough times, of:

- 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

As defined in ASTM F-739-96

- Excellent breakthrough time > 480 min
- Good breakthrough time > 20 min
- Fair breakthrough time < 20 min
- Poor glove material degradation

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)

· **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.**

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

Body protection

See Other protection below

Other protection

- ▶ Overalls.
- ▶ PVC Apron.
- ▶ PVC protective suit may be required if exposure severe.
- ▶ Eyewash unit.
- ▶ Ensure there is ready access to a safety shower.

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:

ARDEX EG15 Resin Part B Improved Formula

| Material | CPI |
|----------------|-----|
| BUTYL | A |
| VITON | A |
| NATURAL RUBBER | C |
| NEOPRENE | C |

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

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

| Required minimum protection factor | Maximum gas/vapour concentration present in air p.p.m. (by volume) | Half-face Respirator | Full-Face Respirator |
|------------------------------------|--|----------------------|----------------------|
| up to 10 | 1000 | AK-AUS / Class1 P2 | - |
| up to 50 | 1000 | - | AK-AUS / Class 1 P2 |
| up to 50 | 5000 | Airline * | - |
| up to 100 | 5000 | - | AK-2 P2 |
| up to 100 | 10000 | - | AK-3 P2 |
| 100+ | | | Airline** |

* - Continuous Flow ** - Continuous-flow or positive pressure demand
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO =

Continued...

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

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SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

| | | | |
|---|---------------------------|--|----------------|
| Appearance | Brownish alkaline liquid. | | |
| Physical state | Liquid | Relative density (Water = 1) | Not Available |
| 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 (°C) | Not Available |
| Melting point / freezing point (°C) | Not Applicable | Viscosity (cSt) | Not Available |
| Initial boiling point and boiling range (°C) | Not Available | Molecular weight (g/mol) | Not Applicable |
| Flash point (°C) | Not Available | Taste | Not Available |
| Evaporation rate | Not Available | Explosive properties | Not Available |
| Flammability | Not Available | Oxidising properties | Not Available |
| Upper Explosive Limit (%) | Not Available | Surface Tension (dyn/cm or mN/m) | Not Available |
| Lower Explosive Limit (%) | Not Available | Volatile Component (%vol) | Not Available |
| Vapour pressure (kPa) | Not Available | Gas group | Not Available |
| Solubility in water | Not Available | pH as a solution (1%) | Not Available |
| Vapour density (Air = 1) | Not Available | VOC g/L | Not Available |
| Heat of Combustion (kJ/g) | Not Available | Ignition Distance (cm) | Not Available |
| Flame Height (cm) | Not Available | Flame Duration (s) | Not Available |
| Enclosed Space Ignition Time Equivalent (s/m3) | Not Available | Enclosed Space Ignition Deflagration Density (g/m3) | Not Available |

SECTION 10 Stability and reactivity

| | |
|---|--|
| Reactivity | See section 7 |
| Chemical stability | <ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur. |
| Possibility of hazardous reactions | See section 7 |
| Conditions to avoid | See section 7 |
| Incompatible materials | See section 7 |
| Hazardous decomposition products | See section 5 |

SECTION 11 Toxicological information

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 vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</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.</p> |
|----------------|---|

Continued...

| | |
|---------------------|--|
| | <p>Systemic effects include headache, nausea, faintness and anxiety. These effects are thought to be transient and are probably related to the pharmacodynamic action of the amines. Histamine release by aliphatic amines may produce bronchoconstriction and wheezing. Inhalation of 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.</p> <p>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 benzyl alcohol may affect respiration (paralysis of the respiratory center, respiratory depression, gasping respirations), cardiovascular system (hypotension)</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> |
| Ingestion | <p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>The material can produce severe chemical burns within the oral cavity and gastrointestinal tract following ingestion.</p> |
| Skin Contact | <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>A 30% fatty acid amide (cocoamide DEA solution) was a moderate skin irritant in rabbits. Test sites were scored for irritation according to Draize, and the Primary Irritation Index (PII) was 3.1 (maximum irritation is indicated by the score of 8). In products intended for prolonged contact with the skin, the concentration of cocoamide DEA should not exceed 5%. Fatty acid diethanolamides (C8-C18) and monoethanolamides are classified by CESIO as irritating.</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>1% solutions of many cationic surfactants produce dermal irritation and 10% solutions may be corrosive producing chemical burns. Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>The material can produce severe chemical burns following direct contact with the skin.</p> |
| Eye | <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.</p> <p>The material can produce severe chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating.</p> |
| Chronic | <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.</p> <p>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.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers. Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:</p> <ul style="list-style-type: none"> - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. <p>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>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.</p> <p>Imidazole is structurally related to histamine and has been used as an antagonist to counteract the effects of excess histamine found in certain induced physiological conditions (it therefore acts as an antihistamine).</p> <p>Imidazoles have been reported to disrupt male fertility through disruption of testicular function.</p> <p>Certain imidazole fungicides provoke histamine release by a non-immunological mechanism, induce airway constriction in guinea-pigs and hence may be harmful to spray operators who might inhale fungicide aerosols used for plant protection.</p> <p>Imidazole fungicides inhibit the cytochrome P450 (CYP) complex, including the 14alpha-demethylase (CYP51) enzyme required for ergosterol biosynthesis, in fungal cell membranes. In addition, intracellular accumulation of toxic methylated sterols occurs and the synthesis of triglycerides and phospholipids is altered. Disturbances in oxidative and peroxidative enzyme activities lead to an intracellular toxic concentration of hydrogen peroxide. As a result, intracellular organelle destruction then leads to cell necrosis.</p> <p>2-Methylimidazole decreased luteinising hormone secretion and tissue interstitial fluid testosterone concentration two hours after injection into Sprague Dawley rats.</p> <p>Imidazoles bind to cytochrome P450 haeme, resulting in inhibition of catalysis. However, 2-substituted imidazoles are considered to be poor inhibitors. Imidazole is probably an inducer of cytochrome P4502E1. In general, inducers of this isozyme stabilise the enzyme by preventing phosphorylation of a serine which leads to haeme loss.</p> <p>Several drugs containing an imidazole moiety were retained and bound in connective tissue when administered to laboratory animals. The bound material was primarily recovered from elastin (70%) and the collagen. It is postulated that reaction with aldehydes gives an aldol condensation pro</p> <p>Allergic reactions to benzoic acid have been reported. Of 100 patients with asthma undergoing provocation tests with benzoic acid, 47 showed positive reactions. In another study, of 75 patients with recurrent urticaria (skin eruptions) and angio-oedema (a deep dermal condition characterised by large wheals) of more than 4 months duration, 44 were found to be sensitive to sodium benzoate or p-hydroxybenzoic acid (paraben), alone or in conjunction with aspirin or azo- dyes, or both. In a further work there was no significant objective or subjective skin response to two 500-mg daily doses of benzoic acid or lactic acid in a double blind study of 150 dermatological patients</p> <p>Secondary amines may react in the acid conditions of the stomach with oxidants or preservatives) to form potentially carcinogenic N-nitrosamines. The formation of nitrosamines from such amines has not only been observed in animals models but, at least for certain compounds, in the workplace. The amine-containing substances and end products handled at work can themselves be contaminated to a</p> |

ARDEX EG15 Resin Part B Improved Formula

degree with corresponding nitrosamines. Under conditions encountered in practice nitrosation is to be expected with secondary amines and to a limited extent with primary and tertiary amines. Nitrogen oxides are the most probable nitrosating agents. Nitrosyl chloride, nitrite esters, metal nitrites and nitroso compounds may also be involved. Several factors such as pH, temperature, catalysts and inhibitors influence the extent of nitrosation. Two precautionary measures are therefore necessary when handling amines at the workplace.

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

Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Report No. 31, DFG, 1995

In animal experiments the oesophagus is shown to be the most important target organ for nitrosamines, independent of the route of application. The mechanism of this organotrophy cannot be explained sufficiently. The high oesophageal epithelium metabolic activation of nitrosamines, together with a comparatively low DNA repair, probably plays the most important role. In addition chronic stress factors, which lead to high stimulation of epithelial turnover, are a pacemaker for malignant progression. In some countries, the traditional consumption of extremely hot drinks leads to constant burns of the oesophagus, which increases the risk. Mate, a non-alcoholic brew, frequently consumed as tea in Uruguay, appears to be a high risk factor for oesophageal cancer

Prolonged or repeated exposure to benzyl alcohol may cause allergic contact dermatitis.

Prolonged or repeated ingestion may affect behavior/central nervous system with symptoms similar to acute ingestion. It may also affect the liver, kidneys, cardiovascular system, and metabolism (weight loss).

Animal studies have shown this compound to cause lung, liver, kidney and CNS disorders. Studies in animals have shown evidence of teratogenicity in the chick embryo. The significance of the information for humans is unknown.

Benzyl alcohol showed no evidence of carcinogenic activity in long-term toxicology and carcinogenesis study.

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.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

| | | |
|---|---|--|
| ARDEX EG15 Resin Part B Improved Formula | TOXICITY | IRRITATION |
| | Not Available | Not Available |
| benzyl alcohol | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 2000 mg/kg ^[2] | Eye (rabbit): 0.75 mg open SEVERE |
| | Inhalation (Rat) LC50: >4.178 mg/L4h ^[2] | Eye: adverse effect observed (irritating) ^[1] |
| | Oral (Rat) LD50: 1230 mg/kg ^[2] | Skin (man): 16 mg/48h-mild |
| | | Skin (rabbit): 10 mg/24h open-mild |
| | | Skin: no adverse effect observed (not irritating) ^[1] |
| isophorone diamine | TOXICITY | IRRITATION |
| | dermal (rat) LD50: >2000 mg/kg ^[1] | Eye: adverse effect observed (irreversible damage) ^[1] |
| | Inhalation (Rat) LC50: >=1.07<=5.01 mg/l4h ^[1] | Skin: adverse effect observed (corrosive) ^[1] |
| | Oral (Rat) LD50: 1030 mg/kg ^[2] | |
| formaldehyde/ benzenamine, hydrogenated | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: >1000 mg/kg ^[1] | Not Available |
| | Oral (Rat) LD50: >50<300 mg/kg ^[1] | |
| tall oil/ tetraethylenepentamine polyamides | TOXICITY | IRRITATION |
| | Oral (Rat) LD50: >5000 mg/kg ^[2] | Eyes (rabbit) (-) moderate |
| | | Skin (rabbit) (-) moderate |
| trimethylhexamethylene diamine | TOXICITY | IRRITATION |
| | Oral (Rat) LD50: 910 mg/kg ^[2] | Eye (rabbit): Corrosive *Sensitiser ** [* = Manufacturer CG] [** = Manufacturer Degussa] |
| | | Skin (rabbit): Corrosive * |
| cycloaliphatic amine | TOXICITY | IRRITATION |
| | Not Available | Not Available |
| tetraethylenepentamine | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 660 mg/kg ^[2] | Eye (rabbit): 100 mg/24h moderate |
| | Oral (Rat) LD50: 3990 mg/kg ^[2] | Eye (rabbit): 5 mg moderate |
| | | Skin (rabbit): 495 mg SEVERE |
| | | Skin (rabbit): 5 mg/24h SEVERE |

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

BENZYL ALCOHOL

Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and conjugal contact dermatitis occur.

Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.

Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.

Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.

Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual.

Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease. Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.

Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereireae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereireae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

Pigmentary anomalies: The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prohaptens is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohaptens is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prohaptens or as a prohaptens, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.

Prohaptens

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

In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal. The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into

chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation. CYP1A2 is a member of the cytochrome P450 super family, is one of the best characterized. It is responsible for the metabolism of commonly drugs belonging to classes such as antidepressants, antipsychotics, mood stabilizers, beta blockers and sedative/hypnotics. CYP1A2 also metabolises a number of procarcinogens (such as those in cigarettes). Cigarette smoking may lead to three fold increase in 1A2 activity, which explains why smokers require higher doses of beta blockers than non-smokers. Drugs that inhibit CYP1A2 will predictably increase the plasma concentrations of the medications or decrease in clearance of substrates. Drugs such as ciprofloxacin, fluvoxamine, verapamil, cimetidine, caffeine and isoniazid are inhibitors of CYP1A2 enzyme. Vegetables such as grape fruit juice, cumic and turmeric are inhibitors of the CYP1A2 enzyme which may lead to increase plasma concentration of psychotropics.

Inhibition of NF- κ B in vivo can be detrimental. NF- κ B controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF- κ B activity.

The same functions that make NF- κ B attractive for developing inhibitors for treating disease also play a role in homeostasis, and disruption of the NF- κ B pathway during development or in adults leads to unfavorable and potentially unhealthy consequences.

NF- κ B plays a role in multiple homeostatic cellular processes including response to stimuli, cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such as p38 and JNK. In addition to mediating proinflammatory responses, NF- κ B may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF- κ B can have adverse consequences such as immune suppression and tissue damage.

Understanding the consequences of lack of NF- κ B activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF- κ B resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF- κ B activation including IKK gamma (NEMO), a subunit of the IKK complex, and I κ Balpha. The IKK gamma mutations result in a defective IKK complex and the I κ Balpha mutation results in an I κ Balpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF- κ B activation and ectodermal dysplasia with immunodeficiency. In general patients with these genetic defects have multiple immunological defects including impaired innate immunity, impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF- κ B pathway will help prepare for potential adverse effects of pharmacologic NF- κ B inhibitors.

The requirement for NF- κ B in the development and maintenance of the immune system is well documented. NF- κ B is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF- κ B or the I κ Bbeta gene results in death during fetal development primarily due to massive liver apoptosis.

Fetal liver stem cells from p65 or I κ Bbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF- κ B for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF- κ B activity. Lymphocyte depletion with chemical or genetic inhibition of NF- κ B have implications for therapeutic potential use in humans. The double-sided nature of NF- κ B inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations. In addition to controlling lymphocyte development, NF- κ B plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF- κ B which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF- κ B through phosphorylation of CARMA1 by PKC theta and PKC beta respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF- κ B plays a role in T-cell response to costimulatory signals. Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs). TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF- κ B leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF- κ B during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF- κ B is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF- κ B activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation. Given the diverse roles NF- κ B plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF- κ B pathway are susceptible to parasitic and bacterial infection.

The role of NF- κ B in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF- κ B deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF- κ B targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator. Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF- κ B activation.

The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles.

The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity.

At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin.

The potential for eye irritation is minimal.

With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low.

NOAELs for maternal and developmental toxicity are far in excess of current human exposure levels.

No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity in vitro bacterial assays, and in vitro mammalian cell assays. All in vivo micronucleus assays were negative.

It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients.

The Research Institute for Fragrance Materials (RIFM) Expert Panel

A member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption, metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances.

All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals. The substances in this group:

- contain a benzene ring substituted with a reactive primary oxygenated functional group or can be hydrolysed to such a functional group
- the major pathway of metabolic detoxification involves hydrolysis and oxidation to yield the corresponding benzoic acid derivative which is excreted either as the free acid or the glycine conjugate
- they show a consistent pattern of toxicity in both short- and long- term studies and
- they exhibit no evidence of genotoxicity in standardised batteries of in vitro and in vivo assays.

The benzyl derivatives are rapidly absorbed through the gut, metabolised primarily in the liver, and excreted in the urine as glycine conjugates of benzoic acid derivatives.

In general, aromatic esters are hydrolysed *in vivo* through the catalytic activity of carboxylesterases, the most important of which are the A-esterases. Hydrolysis of benzyl and benzoate esters to yield corresponding alcohols and carboxylic acids and hydrolysis of acetals to yield benzaldehyde and simple alcohols have been reported in several experiments.

The alcohols and aldehydes are rapidly oxidised to benzoic acid while benzoate esters are hydrolysed to benzoic acid.

Flavor and Extract Manufacturers Association (FEMA)

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

For benzyl alkyl alcohols:

Unlike benzylic alcohols, the beta-hydroxyl group of the members of this cluster is unlikely to undergo phase II metabolic activation.

Instead, the beta-hydroxyl group is expected to contribute to detoxification via oxidation to hydrophilic acid. Despite structural similarity to carcinogenic ethyl benzene, only a marginal concern has been assigned to phenethyl alcohol due to limited mechanistic analogy.

For benzoates:

Acute toxicity: Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol.

The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds.

Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye.

Sensitisation: The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers.

Repeat dose toxicity: For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed.

For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur.

Mutagenicity: All chemicals showed no mutagenic activity in *in vitro* Ames tests. Various results were obtained with other *in vitro* genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity *in vivo*. While some mixed and/or equivocal *in vitro* chromosomal/chromatid responses have been observed, no genotoxicity was observed in the *in vivo* cytogenetic, micronucleus, or other assays. The weight of the evidence of the *in vitro* and *in vivo* genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.

In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxic studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts.

Developmental toxicity: In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL = 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed.

For isophorone diamine

Based on a limited skin irritation study with rabbits and rats, isophorone diamine is deemed to be a strong irritant (duration of the exposure not reported) and corrosive after repeated application. Isophorone diamine is corrosive to the eyes of rabbits when tested according to OECD TG 405. Isophorone diamine was found to induce dermal sensitisation when tested according to OECD TG 406 in guinea pigs. From a number of publications there is evidence that frequent occupational exposure to isophorone diamine may lead to the development of allergic contact dermatitis in humans. No definite conclusion can be currently drawn on respiratory sensitisation.

From two 14-day inhalative exposure studies with rats no NOAEL could be determined. At the first study s LOAEL of 18 mg/m³, degeneration/necrosis in the olfactory epithelium of the nose were observed. Trachea, larynx and lungs were affected at 200 mg/m³ and above (degeneration/necrosis, hyperplasia, squamous metaplasia). At the LOAEL of the follow-up study, i.e. at 2.2 mg/m³, reversible minimal to mild degeneration of respiratory nasal mucosa in the anterior dorsal nose was observed. In a subchronic drinking water study according to OECD TG 408, the administration of 150 mg/kg bw/day led to reduced absolute and relative kidney weights in male and female rats (histopathology being indicative for tubular nephrosis), while 59 mg/kg bw/day (males) and 62 mg/kg bw/day (females) were determined as a NOAEL.

Isophorone diamine was not mutagenic in bacteria and mammalian cell systems *in vitro* (Ames test according to Directive 84/449/EEC B.14 (1984) and HPRT test according to OECD TG 476 (1984)). It did not induce chromosomal aberrations in CHO cells *in vitro* in a test performed in accordance with OECD TG 473. *In vivo* mouse micronucleus tests (one performed according to OECD TG 474 (1983) for the induction of micronucleated polychromatic erythrocytes were clearly negative. From all *in vitro* and *in vivo* tests performed there is no evidence that isophorone diamine has a mutagenic or clastogenic potential.

No studies have been performed on the toxicity of isophorone diamine to reproduction.

Data from an oral 90-day study in rats according to OECD TG 408 did not reveal any adverse effects on the male and female reproductive organs.

Isophorone diamine did not show any teratogenic or embryofetotoxic effects in a gavage study with rats performed in accordance with OECD TG 414 (2001) up to and including the highest tested dose level of 250 mg/kg bw/day. The NOAEL for maternal toxicity was 50 mg/kg bw/day, effects at 250 mg/kg bw/day were reduced food consumption and reduced body weight gain. The NOAEL for developmental toxicity is 250 mg/kg bw/day.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

ISOPHORONE DIAMINE

FORMALDEHYDE/ BENZENAMINE, HYDROGENATED

Amine adducts have much reduced volatility and are less irritating to the skin and eyes than amine hardeners. However commercial amine adducts may contain a percentage of unreacted amine and all unnecessary contact should be avoided.

Amine adducts are prepared by reacting excess primary amines with epoxy resin.

TALL OIL/ TETRAETHYLENAPENTAMINE POLYAMIDES

For imidazoline surfactants (amidoamine/ imidazoline - AAI)

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 polyethyleneamines (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.

ARDEX EG15 Resin Part B Improved Formula

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 tetraethylene-pentamine 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

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)

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.

Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonella reverse mutation assay exist for all of the subcategories.

Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II.

In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.

Some typical applications of FND Amides are:

masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers.

The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health.

The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals.

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.

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 depends on the exposure time and type of skin

Sensitisation: Topical mucosal application of QACs may produce sensitisation. Reports on case stories and patch test have shown that compounds such as benzalkonium chloride, cetalkonium chloride and cetrinide 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 has 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. Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41.

TETRAETHYLENEMPENTAMINE

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

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

TETA is of moderate acute toxicity: LD50(oral, rat) > 2000 mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure to saturated vapour via inhalation was tolerated without impairment. Exposure to 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

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| | <p>formation.</p> <p>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.</p> <p>The in vivo micronucleus tests (i.p. and oral) and the SLRL test showed negative results.</p> <p>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).</p> <p>Experience with female patients suffering from Wilson's disease demonstrated that no miscarriages and no foetal abnormalities occur during treatment with TETA.</p> <p>In rats, there are several studies concerning developmental toxicity. The oral treatment of rats with 75, 375 and 750 mg/kg resulted in no effects on dams and fetuses, except slight increased fetal body weight. After oral treatment of rats with 830 or 1670 mg/kg bw only in the highest dose group increased foetal abnormalities in 27/44 fetus (69,2 %) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significantly the foetal abnormalities of the highest dose group to 3/51 (6,5 %). These findings suggest that the developmental toxicity is produced as a secondary consequence of the chelating properties of TETA.</p> |
| <p>BENZYL ALCOHOL & ISOPHORONE DIAMINE & TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TRIMETHYLHEXAMETHYLENE DIAMINE & TETRAETHYLENEPENTAMINE</p> | <p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> |
| <p>ISOPHORONE DIAMINE & FORMALDEHYDE/ BENZENAMINE, HYDROGENATED & TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TRIMETHYLHEXAMETHYLENE DIAMINE & CYCLOALIPHATIC AMINE & TETRAETHYLENEPENTAMINE</p> | <p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p> |
| <p>ISOPHORONE DIAMINE & TRIMETHYLHEXAMETHYLENE DIAMINE</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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> |
| <p>FORMALDEHYDE/ BENZENAMINE, HYDROGENATED & CYCLOALIPHATIC AMINE</p> | <p>No significant acute toxicological data identified in literature search.</p> |
| <p>TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TETRAETHYLENEPENTAMINE</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>For alkyl polyamines:</p> <p>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</p> <p>Acute toxicity of the alkyl polyamines cluster is low to moderate via oral exposure and a moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin 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.</p> <p>Polyamines potentiate NMDA induced whole-cell currents in cultured striatal neurons</p> <p>Tetraethylenepentamine (TEPA) has a low acute toxicity when administered orally to rats (LD50 = 3250 mg/kg). In an acute inhalation toxicity study with saturated vapor and whole body exposure, the LC50 was calculated to be >9.9 ppm (highest dose tested). TEPA is corrosive to the skin and eyes of rabbits. TEPA is a skin sensitizer in the guinea pig. Dermal acute toxicity LD50 values in the rabbit range from 660 - 1260 mg/kg. The higher toxicity via the dermal route is most likely due to the corrosive nature of TEPA to the skin whereas TEPA would be neutralized by stomach acid.</p> <p>The results of a 28-day repeated dose dermal toxicity study of TEPA indicated a systemic toxicity NOEL of 200 mg/kg/day and a dermal toxicity NOEL (local) of 50 mg/kg/day. The dermal LOAEL was 100 mg/kg/day. In addition, in a repeat dose study of TETA administered in drinking water to male and female rats for 90-92 days, the NOEL was 276 mg/kg/day in males and 352 mg/kg/day in females, the highest dose administered with the NIH-31 diet (several diets were used to study the effects of copper deficiency versus toxicity directly to TEPA). In this same study in mice the NOEL was 487 mg/kg/day in males and 551 mg/kg/day in females, the highest dose administered. A lifetime study was conducted via dermal administration in fifty male mice with a solution of 35% TEPA. There were 20 cases of hyperkeratosis, 13 cases of epidermal necrosis and no evidence of dermal hyperplasia.</p> <p>There were no data available for TEPA for reproductive and developmental toxicity. As a result, data on triethylenetetramine (TETA) was used to address these endpoints. TETA data showed no effects on reproductive organs in rats up to 276 mg/kg/day (males) and 352 mg/kg/day (females) and in mice (up to 500 mg/kg/day) when administered in drinking water. TETA was not considered a developmental toxicant via dermal administration in rabbits at maternally toxic doses up to 125 mg/kg/day but showed developmental toxicity in rats at maternally toxic doses of 830 or 1660 mg/kg/day via drinking water. The maternal and foetal toxicity was most likely due to copper deficiency and zinc toxicity at these levels. Subsequent studies where the diet was supplemented with copper resulted in a decrease of</p> |

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| | foetal abnormalities. There were no standard fertility studies available. However, there were no effects on the gonads observed in a 90-day drinking water study in rats and mice as described above. In the Ames Salmonella assay, TEPA was found to be positive both with and without metabolic activation. TEPA was found to increase sister chromatid exchange in CHO cells and was considered positive in a UDS assay using rat hepatocytes. TEPA was not considered genotoxic in the mouse micronucleus assay and had equivocal results in the two dominant lethal assays in <i>Drosophila melanogaster</i> . Again, it is believed that the positive results are based upon TEPA's ability to chelate copper. |
| TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TRIMETHYLHEXAMETHYLENE DIAMINE & TETRAETHYLENEPENTAMINE | The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. |
| TALL OIL/ TETRAETHYLENEPENTAMINE POLYAMIDES & TRIMETHYLHEXAMETHYLENE DIAMINE | <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 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: Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies. While most polyurethane amine catalysts are not 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. 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: Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis. Skin contact with some amines may result in allergic 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: Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling. The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.</p> <p>Ingestion: The oral toxicity of amine catalysts varies from moderately to very toxic. Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract. Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs. Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.</p> <p>Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000 Alliance for Polyurethanes Industry</p> |

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

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

SECTION 12 Ecological information

Toxicity

| ARDEX EG15 Resin Part B Improved Formula | Endpoint | Test Duration (hr) | Species | Value | Source |
|--|---------------|--------------------|---------------|---------------|---------------|
| | Not Available | Not Available | Not Available | Not Available | Not Available |

| benzyl alcohol | Endpoint | Test Duration (hr) | Species | Value | Source |
|----------------|-----------|--------------------|---------|---------|--------|
| | NOEC(ECx) | 336h | Fish | 5.1mg/l | 2 |
| | LC50 | 96h | Fish | 10mg/l | 2 |

Continued...

| | | | | | |
|---|--|---------------------------|-------------------------------|---------------|---------------|
| | EC50 | 72h | Algae or other aquatic plants | 500mg/l | 2 |
| | EC50 | 48h | Crustacea | 230mg/l | 2 |
| | EC50 | 96h | Algae or other aquatic plants | 76.828mg/l | 2 |
| isophorone diamine | Endpoint | Test Duration (hr) | Species | Value | Source |
| | BCF | 1008h | Fish | <0.3 | 7 |
| | EC50 | 72h | Algae or other aquatic plants | 37mg/l | 1 |
| | EC50 | 48h | Crustacea | 14.6-21.5mg/l | 4 |
| | LC50 | 96h | Fish | 70mg/l | 1 |
| | NOEC(ECx) | 72h | Algae or other aquatic plants | 1.5mg/l | 1 |
| formaldehyde/ benzenamine, hydrogenated | Endpoint | Test Duration (hr) | Species | Value | Source |
| | EC50 | 72h | Algae or other aquatic plants | 43.94mg/l | 2 |
| | EC50 | 48h | Crustacea | 15.4mg/l | 2 |
| | LC50 | 96h | Fish | 63mg/l | 2 |
| | EC10(ECx) | 72h | Algae or other aquatic plants | 1.2mg/l | 2 |
| tall oil/ tetraethylenepentamine polyamides | Endpoint | Test Duration (hr) | Species | Value | Source |
| | EC50 | 72h | Algae or other aquatic plants | 0.638mg/l | 2 |
| | EC50 | 48h | Crustacea | 0.18mg/l | 2 |
| | EC50(ECx) | 48h | Crustacea | 0.18mg/l | 2 |
| | LC50 | 96h | Fish | 0.19mg/l | 2 |
| trimethylhexamethylene diamine | Endpoint | Test Duration (hr) | Species | Value | Source |
| | EC50 | 72h | Algae or other aquatic plants | 29.5mg/l | 1 |
| | EC10(ECx) | 72h | Algae or other aquatic plants | 16.3mg/l | 1 |
| cycloaliphatic amine | Endpoint | Test Duration (hr) | Species | Value | Source |
| | Not Available | Not Available | Not Available | Not Available | Not Available |
| tetraethylenepentamine | Endpoint | Test Duration (hr) | Species | Value | Source |
| | EC50 | 72h | Algae or other aquatic plants | 2.1mg/l | 1 |
| | EC50 | 48h | Crustacea | 24.1mg/l | 1 |
| | NOEC(ECx) | 72h | Algae or other aquatic plants | 0.5mg/l | 1 |
| Legend: | Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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 | | | | |

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

DO NOT discharge into sewer or waterways.

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

Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|--------------------------------|-------------------------|------------------|
| benzyl alcohol | LOW | LOW |
| isophorone diamine | HIGH | HIGH |
| trimethylhexamethylene diamine | HIGH | HIGH |
| tetraethylenepentamine | LOW | LOW |

Bioaccumulative potential

| Ingredient | Bioaccumulation |
|--------------------------------|------------------------|
| benzyl alcohol | LOW (LogKOW = 1.1) |
| isophorone diamine | LOW (BCF = 3.4) |
| trimethylhexamethylene diamine | LOW (LogKOW = 1.6347) |
| tetraethylenepentamine | LOW (LogKOW = -3.1604) |



Mobility in soil

| Ingredient | Mobility |
|--------------------------------|-----------------------|
| benzyl alcohol | LOW (Log KOC = 15.66) |
| isophorone diamine | LOW (Log KOC = 340.4) |
| trimethylhexamethylene diamine | LOW (Log KOC = 1101) |
| tetraethylenepentamine | LOW (Log KOC = 1098) |

SECTION 13 Disposal considerations**Waste treatment methods**

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|-------------------------------------|--|
| 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. ▶ 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. |
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SECTION 14 Transport information**Labels Required**

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| Marine Pollutant |  |
| HAZCHEM | 2X |

Land transport (ADG)

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|------------------------------------|--|----------------|
| 14.1. UN number or ID number | 1760 | |
| 14.2. UN proper shipping name | CORROSIVE LIQUID, N.O.S. (contains isophorone diamine and tall oil/ tetraethylenepentamine polyamides) | |
| 14.3. Transport hazard class(es) | Class | 8 |
| | Subsidiary Hazard | Not Applicable |
| 14.4. Packing group | II | |
| 14.5. Environmental hazard | Environmentally hazardous | |
| 14.6. Special precautions for user | Special provisions | 274 |
| | Limited quantity | 1 L |

Air transport (ICAO-IATA / DGR)

| | | |
|------------------------------------|--|----------------|
| 14.1. UN number | 1760 | |
| 14.2. UN proper shipping name | Corrosive liquid, n.o.s. * (contains isophorone diamine and tall oil/ tetraethylenepentamine polyamides) | |
| 14.3. Transport hazard class(es) | ICAO/IATA Class | 8 |
| | ICAO / IATA Subsidiary Hazard | 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 | 1760 |
| 14.2. UN proper shipping name | CORROSIVE LIQUID, N.O.S. (contains isophorone diamine and tall oil/ tetraethylenepentamine polyamides) |

| | | |
|------------------------------------|------------------------|----------------|
| 14.3. Transport hazard class(es) | IMDG Class | 8 |
| | IMDG Subsidiary Hazard | 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 |

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

Not Applicable

14.7.2. Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

| Product name | Group |
|---|---------------|
| benzyl alcohol | Not Available |
| isophorone diamine | Not Available |
| formaldehyde/ benzenamine, hydrogenated | Not Available |
| tall oil/ tetraethylenepentamine polyamides | Not Available |
| trimethylhexamethylene diamine | Not Available |
| cycloaliphatic amine | Not Available |
| tetraethylenepentamine | Not Available |

14.7.3. Transport in bulk in accordance with the IGC Code

| Product name | Ship Type |
|---|---------------|
| benzyl alcohol | Not Available |
| isophorone diamine | Not Available |
| formaldehyde/ benzenamine, hydrogenated | Not Available |
| tall oil/ tetraethylenepentamine polyamides | Not Available |
| trimethylhexamethylene diamine | Not Available |
| cycloaliphatic amine | Not Available |
| tetraethylenepentamine | Not Available |

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture****benzyl alcohol is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australian Inventory of Industrial Chemicals (AIIC)

isophorone diamine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
 Australian Inventory of Industrial Chemicals (AIIC)

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

Australian Inventory of Industrial Chemicals (AIIC)

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

Australian Inventory of Industrial Chemicals (AIIC)

trimethylhexamethylene diamine is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

cycloaliphatic amine is found on the following regulatory lists

Not Applicable

tetraethylenepentamine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
 Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

| National Inventory | Status |
|---|---|
| Australia - AIIC / Australia Non-Industrial Use | No (cycloaliphatic amine) |
| Canada - DSL | No (cycloaliphatic amine) |
| Canada - NDSL | No (benzyl alcohol; formaldehyde/ benzenamine, hydrogenated; trimethylhexamethylene diamine; cycloaliphatic amine; tetraethylenepentamine) |
| China - IECSC | No (cycloaliphatic amine) |
| Europe - EINEC / ELINCS / NLP | No (formaldehyde/ benzenamine, hydrogenated; cycloaliphatic amine) |
| Japan - ENCS | No (formaldehyde/ benzenamine, hydrogenated; tall oil/ tetraethylenepentamine polyamides; cycloaliphatic amine) |
| Korea - KECI | No (cycloaliphatic amine) |
| New Zealand - NZIoC | No (cycloaliphatic amine) |
| Philippines - PICCS | No (cycloaliphatic amine) |
| USA - TSCA | No (cycloaliphatic amine) |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (formaldehyde/ benzenamine, hydrogenated; tall oil/ tetraethylenepentamine polyamides; cycloaliphatic amine) |
| Vietnam - NCI | No (cycloaliphatic amine) |
| Russia - FBEPH | No (formaldehyde/ benzenamine, hydrogenated; cycloaliphatic amine) |
| Legend: | <i>Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</i> |

SECTION 16 Other information

| | |
|----------------------|------------|
| Revision Date | 22/11/2023 |
| Initial Date | 22/11/2023 |

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.

Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit,
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration

- ▶ AIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European Inventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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