



AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY)

Gardner-Gibson, Inc.

Version No: 1.1

Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: 01/09/2023

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L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY)
Synonyms	APOC AP-1 GREEN-SEAL Lifetime Heavy Duty Construction Sealant & Adhesive; AP-1 Green-Seal
Proper shipping name	Environmentally hazardous substance, solid, n.o.s. (contains bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate and dibutyltin bis(acetylacetonate))
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Adhesive and Sealant; All-Purpose, Paintable, Structural Adhesive and Sealant
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Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	Gardner-Gibson, Inc.
Address	4161 East 7th Avenue Tampa FL 33605 United States
Telephone	1-813-248-2101
Fax	1-813-248-6768
Website	www.icpgroup.com
Email	sds@icpgroup.com

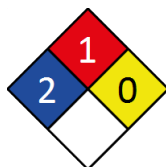
Emergency phone number

Association / Organisation	ChemTel
Emergency telephone numbers	1-800-255-3924
Other emergency telephone numbers	1-813-248-0585

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond



Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Serious Eye Damage/Eye Irritation Category 2A, Reproductive Toxicity Category 1B, Sensitisation (Skin) Category 1
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Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H319	Causes serious eye irritation.
H360	May damage fertility or the unborn child.

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H317	May cause an allergic skin reaction.
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Hazard(s) not otherwise classified

Small amounts of methanol (CAS 67-56-1) are formed by hydrolysis and released upon curing.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing must not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1317-65-3	10-30	<u>limestone</u>
68515-48-0	7-<13	<u>diisononyl phthalate</u>
2768-02-7	1-<5	<u>trimethoxyvinylsilane</u>
1760-24-3	0.1-<1	<u>N-[3-(trimethoxysilyl)propyl]ethylenediamine</u>
22673-19-4	0.1-<1	<u>dibutyltin bis(acetylacetonate)</u>
3069-29-2	0.1-<1	<u>N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine</u>
52829-07-9	0.1-<1	<u>bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate</u>

SECTION 4 First-aid measures**Description of first aid measures**

Eye Contact	If this product comes in contact with the eyes: ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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For acute and short term repeated exposures to methanol:

- Toxicity results from accumulation of formaldehyde/formic acid.
- Clinical signs are usually limited to CNS, eyes and GI tract. Severe metabolic acidosis may produce dyspnea and profound systemic effects which may become intractable. All symptomatic patients should have arterial pH measured. Evaluate airway, breathing and circulation.
- Stabilise obtunded patients by giving naloxone, glucose and thiamine.
- Decontaminate with ipecac or lavage for patients presenting 2 hours post-ingestion. Charcoal does not absorb well; the usefulness of cathartic is not established.
- Forced diuresis is not effective; haemodialysis is recommended where peak methanol levels exceed 50 mg/dL (this correlates with serum bicarbonate levels below 18 mEq/L).
- Ethanol, maintained at levels between 100 and 150 mg/dL, inhibits formation of toxic metabolites and may be indicated when peak methanol levels exceed 20 mg/dL. An intravenous solution of ethanol in D5W is optimal.
- Folate, as leucovorin, may increase the oxidative removal of formic acid. 4-methylpyrazole may be an effective adjunct in the treatment. 8-Phenytoin may be preferable to diazepam for controlling seizure.

[Ellenhorn Barceloux: Medical Toxicology]

Methanol poisoning can be treated with fomepizole, or if unavailable, ethanol. Both drugs act to reduce the action of alcohol dehydrogenase on methanol by means of competitive inhibition. Ethanol, the active ingredient in alcoholic beverages, acts as a competitive inhibitor by more effectively binding and saturating the alcohol dehydrogenase enzyme in the liver, thus blocking the binding of methanol. Methanol is excreted by the kidneys without being converted into the very toxic metabolites formaldehyde and formic acid. Alcohol dehydrogenase instead enzymatically converts ethanol to acetaldehyde, a much less toxic organic molecule. Additional treatment may include sodium bicarbonate for metabolic acidosis, and hemodialysis or hemodiafiltration to remove methanol and formate from the blood. Folinic acid or folic acid is also administered to enhance the metabolism of formate.

BIOLOGICAL EXPOSURE INDEX - BEI

Determinant	Index	Sampling Time	Comment
1. Methanol in urine	15 mg/l	End of shift	B, NS
2. Formic acid in urine	80 mg/gm creatinine	Before the shift at end of workweek	B, NS

B: Background levels occur in specimens collected from subjects **NOT** exposed.
NS: Non-specific determinant - observed following exposure to other materials.

SECTION 5 Fire-fighting 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
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Special protective equipment and precautions for fire-fighters

Fire Fighting	▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use water delivered as a fine spray to control fire and cool adjacent 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	Combustible. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO ₂) other pyrolysis products typical of burning organic material.

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	Environmental hazard - contain spillage. ▶ Clean up all spills immediately. ▶ Avoid contact with skin and eyes. ▶ Wear impervious gloves and safety goggles. ▶ Trowel up/scrape up. ▶ Place spilled material in clean, dry, sealed container. ▶ Flush spill area with water.
Major Spills	Environmental hazard - contain spillage. Minor hazard. ▶ Clear area of personnel. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Control personal contact with the substance, by using protective equipment as required. ▶ Prevent spillage from entering drains or water ways. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▶ Wash area and prevent runoff into drains or waterways.

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- 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

Safe handling	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ DO NOT enter confined spaces until atmosphere has been checked. ▸ DO NOT allow material to contact humans, exposed food or food utensils. ▸ 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	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Phthalates: ▸ react with strong acids, strong oxidisers, permanganates and nitrates ▸ attack some form of plastics Acetic acid: ▸ vapours forms explosive mixtures with air (above 39 C.) ▸ reacts violently with bases such as carbonates and hydroxides (giving off large quantities of heat), oxidisers, organic amines, acetaldehyde, potassium tert-butoxide ▸ reacts (sometimes violently), with strong acids, aliphatic amines, alkanolamines, alkylene oxides, epichlorohydrin, acetic anhydride, 2-aminoethanol, ammonia, ammonium nitrate, bromine pentafluoride, chlorosulfonic acid, chromic acid, chromium trioxide, ethylenediamine, ethyleneimine, hydrogen peroxide, isocyanates, oleum, perchloric acid, permanganates, phosphorus isocyanate, phosphorus trichloride, sodium peroxide, xylene ▸ attacks cast iron, stainless steel and other metals, forming flammable hydrogen gas ▸ attacks many forms of rubber, plastics and coatings ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	limestone	Marble- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	limestone	Limestone- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	limestone	Calcium Carbonate- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	limestone	Limestone- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	limestone	Marble- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	limestone	Calcium Carbonate- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	limestone	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	limestone	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	limestone	Calcium carbonate - total	10 mg/m3	Not Available	Not Available	Not Available

Continued...

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US NIOSH Recommended Exposure Limits (RELS)	limestone	Calcium carbonate - respirable	5 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELS)	limestone	Calcium carbonate - respirable	5 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELS)	limestone	Marble - respirable	5 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELS)	limestone	Limestone - respirable	5 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELS)	limestone	Calcium carbonate - total	10 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELS)	limestone	Marble - total	10 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELS)	limestone	Limestone - total	10 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	dibutyltin bis(acetylacetonate)	Tin, organic compounds (as Sn)	0.1 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELS)	dibutyltin bis(acetylacetonate)	Tin (organic compounds, as Sn)	0.1 mg/m3	Not Available	Not Available	[skin] [*Note: The REL applies to all organic tin compounds except Cyhexatin.]
US OSHA Permissible Exposure Limits (PELs) Table Z-1	bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELS)	bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
limestone	45 mg/m3	210 mg/m3	1,300 mg/m3
trimethoxyvinylsilane	9.5 ppm	100 ppm	120 ppm
N-[3-(trimethoxysilyl)propyl]ethylenediamine	23 mg/m3	250 mg/m3	1,500 mg/m3

Ingredient	Original IDLH	Revised IDLH
limestone	Not Available	Not Available
diisononyl phthalate	Not Available	Not Available
trimethoxyvinylsilane	Not Available	Not Available
N-[3-(trimethoxysilyl)propyl]ethylenediamine	Not Available	Not Available
dibutyltin bis(acetylacetonate)	25 mg/m3	Not Available
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	Not Available	Not Available
bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
diisononyl phthalate	E	≤ 0.1 ppm
trimethoxyvinylsilane	E	≤ 0.1 ppm
N-[3-(trimethoxysilyl)propyl]ethylenediamine	D	> 0.1 to ≤ 1 ppm
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	D	> 0.1 to ≤ 1 ppm

Notes:

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

MATERIAL DATA

Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.

OSHA (USA) concluded that exposure to sensory irritants can:

- ▶ cause inflammation
- ▶ cause increased susceptibility to other irritants and infectious agents
- ▶ lead to permanent injury or dysfunction
- ▶ permit greater absorption of hazardous substances and
- ▶ acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

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Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class OSF Description

- | | | |
|---|--------|--|
| A | 550 | Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities |
| B | 26-550 | As "A" for 50-90% of persons being distracted |
| C | 1-26 | As "A" for less than 50% of persons being distracted |
| D | 0.18-1 | 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached |
| E | <0.18 | As "D" for less than 10% of persons aware of being tested |

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant. <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="text-align: left;">Type of Contaminant:</th><th style="text-align: left;">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> Within each range the appropriate value depends on: <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="text-align: left;">Lower end of the range</th><th style="text-align: left;">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> 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.	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
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Personal protection	   																				
Eye and face protection	▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]																				
Skin protection	See Hand protection below																				
Hands/feet protection	▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber NOTE: ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.																				

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Body protection	See Other protection below
Other protection	▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Gray		
Physical state	Free-flowing Paste	Relative density (Water = 1)	1.53
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 Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	>100	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Not Available	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▶ 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

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Information on toxicological effects

Inhaled	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. Phthalates (aromatic dicarboxylic acid esters), in general, exhibit low toxicity, partly because of poor absorption but mainly as a result of rapid metabolism in which the esters are saponified to phthalic acid (which is rapidly excreted) and the parent alcohol (which is subsequently metabolised). The pathology of these compounds seems to be related to the released alcohol and its biological effects. The rate of absorption of ingested phthalate esters is influenced by the content of dietary fat. Ingested phthalate esters may to a lesser degree be absorbed as the monoester derivatives or in the case of di(2-ethylhexyl)phthalate, as the diester. Cumulative toxicity of the phthalates has been observed on repeated administration. Both di-n-octyl phthalate and di(2-ethylhexyl)phthalate were found to have 22-28 times greater toxicity (based on LD50s) following repeated administration to animals. The liver has been shown to be the target organ affected by the phthalates. In general phthalates have induced liver enlargement; this increase in liver weight has been attributed to rapid cell division (hyperplasia) along with the detachment of cells (hypertrophy). The increase in liver weight caused by phthalates has been found to reverse to normal or even below normal levels on prolonged exposure. Exposure to phthalates, in general, has been found to be associated with a reduction in circulating cholesterol and serum triglyceride levels which accounted for a reduction in liver steroidogenesis. The phthalates also effect carbohydrate metabolism in the liver producing depleted glycogen electron transport inhibitors following interaction with mitochondria. Testicular atrophy produced in rats during feeding studies depends on the length and structure of the alcohol; in general the lower molecular weight esters produce the more severe effects. The toxicity of phthalic acid isomers decreases in the order o-phthalic acid, isophthalic acid and terephthalic acid. Phthalic acid is not metabolised but is excreted, unchanged, in the urine and faeces. Terephthalic acid appears to potentiate the biological effects of substances such as antibiotics, thiamine and sulfonamides.
Skin Contact	Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either: - produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or - produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Eye	Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	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. 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. 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. 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. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies 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 is not a secondary non-specific consequence of other toxic effects. The various phthalates have different uses, chemical structures and toxicity profiles. It is therefore difficult to generalise about the safety of all phthalates as a group. The main health concern associated with some phthalates is that animal studies have shown that high regular doses can affect the reproductive system in developing young, particularly males. While there is no significant risk to the general population, young children may experience higher exposures than the general population if they chew or suck on phthalate-containing toys, or if they ingest phthalates over a long period from other products containing high levels of phthalates. In animal tests, phthalates have been shown to "feminise" male animals, increasing the likelihood of small or undeveloped testes, undescended testicles, and low sperm counts. A 2005 study also linked higher foetal exposure to phthalates through the mother's blood with increased risk of developmental abnormalities in male infants. Higher phthalate levels are also associated with lower testosterone production and reduced sperm count in men. One study suggested that high levels of phthalates may be connected to the current obesity epidemic in children. It was found that obese children show greater exposure to phthalates than non-obese children. It was reported that the obesity risk increases according to the level of the chemical found in the children's bloodstream. In a national cross-section of U.S. men, concentrations of several prevalent phthalate metabolites showed statistically significant correlations with abnormal obesity and insulin resistance. A further study found that people with elevated phthalate levels had roughly twice the risk of developing diabetes compared with those with lower levels. This study also found that phthalates were associated with disrupted insulin production. Much of the current research on effects of phthalate exposure has been focused towards children and men's health, however, women may be at higher risk for potential adverse health effects of phthalates due to increased cosmetic use. According to in vivo and observational studies there is an association between phthalate exposure and endocrine disruption leading to development of breast cancer. This finding may be associated with the demethylation of the oestrogen receptor complex in breast cancer cells. A Russian study describes exposure by workers to mixed phthalates (and other plasticisers) - pain, numbness and spasms in the upper and

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lower extremities were related to duration of exposures. Symptoms usually developed after the sixth or seventh year of work. Neurological studies revealed the development of polyneuritis in about 30% of the workers involved in this study. About 30% of the workforce showed depression of the vestibular receptors. Because the study described mixed exposures it is difficult to determine what, if any, unique role was played by the phthalates. Increased incidences of anovulatory reproductive cycles and low oestrogen concentrations were reported among Russian women working with phthalate plasticisers; the abnormal cycles were associated with spontaneous abortion. The specific phthalates implicated, dose levels and other data were not reported. It has been alleged that the phthalates mimic or interfere with sex packaging) and are used as ingredients in paints, inks and adhesives. Their potential for entering the human body is marked. They have been added to a list of chemicals (including alkyl phenolics, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and dioxins) which are implicated in reducing sperm counts and fertility in males a phenomenon which has apparently arisen since the mid 1960s. Phthalates are generally considered to be in a class of endocrine disruptors known as "xenoestrogens," for their ability to mimic the effect of oestrogen on the body.

Although the human foetus is "bathed" in naturally occurring oestrogens during pregnancy it is suggested that it has developed a protective mechanism against natural oestrogens but is not safe from synthetic variants. These tend to accumulate in body fats which sets them apart from the natural product. During early pregnancy, fats are broken down and may flood the body with concentrated pollutants. Human phthalate exposure during pregnancy results in decreased anogenital distance among baby boys. Boys born to mothers with the highest levels of phthalates were 7 times more likely to have a shortened anogenital distance.

While anogenital distance is routinely used as a measure of foetal exposure to endocrine disruptors in animals, this parameter is rarely assessed in humans, and its significance is unknown.

One study also found that female animals exposed to higher levels of phthalates experienced increased risk of miscarriage, a common symptom of excessive estrogen levels in human women, and stillbirth. Prematurity may also be linked to phthalate exposure.

Another study found a link between exposure to phthalates and increased rates of childhood obesity.

In adult human men, phthalates have been linked to greater waist circumference and higher insulin resistance, a common precursor to type 2 (adult onset) diabetes. They have been linked to thyroid irregularities, asthma, and skin allergies in both sexes. Though the exact mechanism is unclear, studies have linked higher rates of respiratory infections and other symptoms in children living in houses with vinyl floors. One possible explanation is inhalation of dust tainted by phthalates, which are used in cosmetics such as nail polishes and hand creams precisely because of their ability to bind to human tissues.

Animal studies have shown increased risks of certain birth defects (including the genital abnormalities and, in rats, extra ribs) and low birth rates in rats whose mothers were fed higher levels of phthalates.

These effects on foetal development are of particular concern because young women of childbearing age often have higher than average phthalate levels in the body thanks to their use of cosmetics, many of which contain phthalates.

The EU has applied limitations to the use of several phthalates in general food contact applications (packaging and closures) and medical device applications. The USA has introduced regulation of phthalate esters as components of children's toys and childcare articles for children under the age of 12 that could be 'placed in the mouth'.

Endocrine disruptors such as phthalates can be add to the effects of other endocrine disruptors, so even very small amounts can interact with other chemicals to have cumulative, adverse "cocktail effects"

Large amounts of specific phthalates fed to rodents have been shown to damage their liver and testes, and initial rodent studies also indicated hepatocarcinogenicity. Later studies on primates showed that the mechanism is specific to rodents - humans are resistant to the effect.

Studies conducted on mice exposed to phthalates in utero did not result in metabolic disorder in adults. However, "At least one phthalate, monoethyhexyl phthalate (MEHP) has been found to interact with all three peroxisome proliferator-activated receptors (PPARs). PPARs are members of the nuclear receptor superfamily involved in lipid and carbohydrate metabolism.

Prenatal exposure to phthalates may affect children's mental, motor and behavioral development during the preschool year.

A 2009 study found that prenatal phthalate exposure was related to low birth weight in infants. Low birth weight is the leading cause of death in children under 5 years of age and increases the risk of cardiovascular and metabolic disease in adulthood. Another study found that women who deliver prematurely have, on average, up to three times the phthalate level in their urine compared to women who carry to term.

Several findings point to a statistically significant correlation between urine phthalate concentrations in children and symptoms of attention deficit hyperactivity disorder (ADHD).

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.

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	Not Available	Not Available
limestone	TOXICITY	IRRITATION
	Oral (Rat) LD50: 6450 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating) ^[1]
diisononyl phthalate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3160 mg/kg ^[2]	Not Available
	Inhalation(Rat) LC50: >4.4 mg/l4h ^[1]	
	Oral (Rat) LD50: >10000 mg/kg ^[2]	
trimethoxyvinylsilane	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3423 mg/kg ^[2]	Eye (rabbit): 500 mg/24h - mild
	Inhalation(Rat) LC50: 2773 ppm4h ^[2]	Eye (rabbit): 500 mg/24h mild
	Oral (Rat) LD50: >300<2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h mild
		Skin: no adverse effect observed (not irritating) ^[1]
N-[3-(trimethoxysilyl)propyl]ethylenediamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 15 mg SEVERE
	Inhalation(Rat) LC50: >1.49<2.44 mg/l4h ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
	Oral (Rat) LD50: 1897 mg/kg ^[1]	Skin (rabbit): 500 mg mild

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		Skin: no adverse effect observed (not irritating) ^[1]
dibutyltin bis(acetylacetonate)	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral (Rat) LD50; 1864 mg/kg ^[1]	
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Inhalation(Rat) LC50: >1.49<2.44 mg/L4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50; 200<=2000 mg/kg ^[1]	
bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3100 mg/kg ^[2]	Eye (rabbit): Severe
	Inhalation(Rat) LC50: 0.5 mg/L4h ^[1]	Skin (rabbit): Irritant
	Oral (Rat) LD50; 3700 mg/kg ^[2]	Skin sensitisation: Negative
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	

LIMESTONE	Eye (rabbit) 0.75: mg/24h - No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects. 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.
DIISONONYL PHTHALATE	[Huls] The effects of DINP on fertility-related parameters such as reduced testosterone content and production and altered reproductive organ weights (with or without histopathologies) have been demonstrated in rats. Although quantitatively being less potent, DINP has exhibited adverse effects on the male reproductive system and sexual differentiation during development in a number of rodent studies (e.g. increased nipple retention, testicular pathology and decreased AGD/AGI in male offspring), which are components of the antiandrogenic pattern observed with diethylhexyl phthalate (DEHP) (a known reproductive toxicant). Foetal expression of genes involved in androgen synthesis such as StAR and Cyp11a were also reduced. There was also a report of increased gene expression levels of InsI3 (a foetal Leydig cell product critical for testis descent) that may infer the impaired testicular steroidogenesis following exposure to DINP at high doses (e.g. = 750 mg/kg bw/d). Reduced InsI3 was also reported in numerous studies with DEHP. Considering the chemical composition of DINP, which is represented as mixed phthalates with side-chains made up of 5?10% methylethylhexyl, limited evidence of the toxicological properties of transitional phthalates may be expected at high doses of DINP tested. The reduced pup weight was observed at approximately 100 mg/kg bw/d in both sexes, both in one- and two-generation reproductive studies in rats, in the absence of overt maternal toxicity. The pup weight reduction was also sustained and not considered solely related to low birth weight. In a post-natal toxicity study, reduced pup weight was also reduced at = 250 mg/kg bw/d. Therefore, this adverse effect of DINP is assessed as the most sensitive endpoint on offspring development. Overall, the available human data do not provide sufficient evidence for a causal relationship between exposure to DINP and adverse health effects in humans. There is also insufficient information to examine the mode of action of DINP on male reproductive tract development and sexual function in comparison with transitional phthalates. However, elements of the plausible mode of action for DINP effects on the male reproductive system, offspring growth and sexual differentiation are considered likely to be parallel in rats and humans if the exposure to DINP is high and within a critical window of development. Therefore, the effects observed in animal studies are regarded as relevant to a human risk assessment. High Molecular Weight Phthalate Esters (HMWPEs) Category as defined by the Phthalate Esters Panel HPV Testing Group (2001) and OECD (2004). The HMWPE group includes chemically similar substances produced from alcohols having backbone carbon lengths of >= 7. Due to their similar chemical structure, category members are generally similar with respect to physicochemical, biological and toxicological properties or display an expected trend. Thus, read-across for toxicity endpoints is an appropriate approach to characterise selected endpoints for members of this category. In some cases the substances have ester side group constituents that span two subcategories (i.e., transitional and high molecular weight constituents). If the level of C4 to C6 constituents in the substance exceeded 10%, the substance was conservatively placed in the transitional subcategory. High molecular weight phthalates are used nearly exclusively as plasticisers of PVC. They are very poorly soluble in water, and have very low vapor pressure. The extant database demonstrates that these substances have few biological effects. A notable exception to this generalisation is that hepatocarcinogenicity has been observed for diisononyl phthalate (DINP). The hepatocarcinogenicity effects of DINP are by a mechanism (peroxisomal proliferation) to which rodents are particularly sensitive. However, it does not appear to be relevant to humans. The high molecular weight phthalates all demonstrate minimal acute toxicity, are not genotoxic, exhibit some liver and kidney effects at high doses, and are negative for reproductive and developmental effects. Further, the available data indicate that the toxicological activity of these molecules diminishes with increasing molecular weight. Studies on HMWPEs indicate that they are rapidly metabolised in the gastrointestinal tract to the corresponding monoester, absorbed and excreted primarily in the urine. Acute toxicity: The available data on phthalates spanning the carbon range from C8-C13 indicate that phthalate esters in the high molecular weight subcategory are not toxic by acute oral and dermal administration; LD50 values of all substances tested exceed the maximum amounts which can be administered to the animals. There are fewer data available on inhalation toxicity; only di-iso-nonyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) have been tested. However, the phthalates in the high molecular weight subcategory have extremely low vapor pressures, and exposure by inhalation at potentially hazardous levels is not anticipated. Repeat dose toxicity. Several substances ranging from C8-C11 have been tested for repeated dose toxicity in studies ranging from 21 days to two years. Ditridecyl phthalate (CAS 119-06-2) has been studied by the Japan

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	Ministry of Health and Welfare (unpublished report) and data for this substance is used as read-across data for DTDP*. In addition results from repeat dose studies examining DINP (CAS 685 15-48-0) and DIDP (CAS 68515-49-1) are used as read across for the di C9-C11 phthalates (CAS 68515-43-5). The principal effects found are those associated with peroxisomal proliferation, including liver enlargement and induction of peroxisomal enzymes. As shown for example in a comparative study of liver effects, the strongest inducers of peroxisomal proliferation were DEHP, DINP, and DIDP with substances of shorter and longer ester side chains (e.g., 610P*, 711P*, and diundecyl phthalate - DUP) showing less pronounced effects. Thus, it is reasonable to conclude that other members of this subcategory would show effects similar to but not more pronounced than those associated with DINP and DIDP. It should also be noted that the relevance of these findings to human health is, at best, questionable. It has been shown that these effects are mediated through the peroxisome proliferation-activated receptor alpha (PPARα); and that levels of PPARα are much higher in rodents than humans. Thus, one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence supporting this postulation is provided by studies in primates in which repeated administration of DEHP and DINP had no effects on liver, kidney or testicular parameters. In this regard it should also be noted that kidney enlargement is also commonly observed but normally without any pathological changes. There is a component of the kidney changes which is also PPARα-related. It has also been shown that in male rats, DINP induces an alpha 2u-globulin nephropathy which is male rat- specific but without relevance to humans. Thus, as was true for the liver changes, the relevance of the kidney changes to human health is also questionable Finally, some of the lower molecular weight phthalates can induce testicular atrophy when administered to juvenile rats at high levels. However, the higher molecular weight phthalates including di-n-octyl phthalate (DnOP), DINP, DIDP, 610P, and 71 1P do not induce testicular atrophy. Further, the testis was not a target organ for DINP in either marmosets or cynomolgus monkeys. Thus, testicular atrophy is not an effect associated with phthalates in the high molecular weight subcategory Reproductive toxicity: Reproductive toxicity tests in rats have been carried out with DINP, DIDP a linear C7-C9 phthalate (CAS 68515-41-3), a linear C9-C11 phthalate, and ditiodecyl phthalate (Japan Ministry of Health and Welfare, unpublished report). None of these affected fertility or profoundly affected male reproductive development. A slight decrease in offspring viability was reported for both DIDP and ditiodecyl phthalate at levels associated with maternal effects. DnOP was tested for effects on fertility in a continuous breeding protocol in mice, and, like the other members of this subcategory, did not reduce fertility. Thus, it can be concluded that the subcategory of high molecular weight phthalates do not affect fertility. Developmental toxicity: Developmental toxicity tests in rats have been carried out with DINP; DIDP; C7-9 phthalate (CAS 68515-41-3); C9-11 phthalate (CAS 68515-43-5); and ditiodecyl phthalate (CAS 119-06-2). None of the substances tested affected litter size, foetal survival or bodyweight, and none produced teratogenic effects. Increased frequencies of developmental variants including dilated renal pelvis, and supernumerary lumbar and cervical ribs were found at levels associated with maternal effects. The toxicological significance of these developmental variants is unclear. DnOP was not teratogenic in mice when tested at very high levels. Thus, it can be concluded that this subcategory of high molecular weight phthalates do not produce profound developmental effects in rodents Genotoxicity: The majority of the substances in the subcategory of high molecular weight phthalates have been tested for genetic activity in the Salmonella assay, and all were inactive. One large program covering many of these substances was carried out by the National Institute of Environmental Health Sciences. Similarly, a range of substances covering the majority of the carbon numbers in this subcategory were found to be inactive in mouse lymphoma tests Chromosomal Aberrations. Two representative members of the subcategory of high molecular weight phthalates (DINP and DIDP) have been tested for chromosomal mutation in the mouse micronucleus test, and both were inactive. Ditiodecyl phthalate (CAS 119-06-2) induced neither structural chromosomal aberrations nor polyploidy in CHL cells up to the limit concentration of 4.75 mg/ ml, in the absence or presence of an exogenous metabolic activation system (Japan Ministry of Health and Welfare, unpublished report). Further, all of the low molecular weight and transitional phthalates that have been tested were inactive. *610P - mixed decyl, hexyl and octyl esters (CAS Rn: 68648-93-1) *711P - C7,C11, branched and linear esters (CAS Rn: 111381-90-9) * DTDP - di-C11-14, C13 rich ester (CAS 68515-47-9)
TRIMETHOXYVINYL SILANE	Manufacturers Data: For alkoxysilanes: Low molecular weight alkoxysilanes (including alkyl orthosilicates) are a known concern for lung toxicity, due to inhalation of vapours or aerosols causing irreversible lung damage at low doses. Alkoxysilane groups that rapidly hydrolyse when in contact with water, result in metabolites that may only cause mild skin irritation. Although there appears to be signs of irritation under different test conditions, based on the available information, the alkoxysilanes cannot be readily classified as a skin irritant. The trimethoxysilane group of chemicals have previously been associated with occupational eye irritation in exposed workers who experienced severe inflammation of the cornea. Based on the collective information, these substances are likely to be severe irritants to the eyes. Methoxysilanes are generally reported to possess higher reactivity and toxicity compared to ethoxysilanes; some methoxysilanes appear to be carcinogenic. In the US, alkoxysilanes with alkoxy groups greater than C2 are classified as moderate concern. Based on available information on methoxysilanes, the possibility that this family causes skin sensitisation cannot be ruled out. Amine-functional methoxysilanes have previously been implicated as a cause of occupational contact dermatitis, often as a result of repeated skin exposure with workers involved in the manufacture or use of the resins containing the chemical during fibreglass production.
N-[3-(TRIMETHOXSILYL)PROPYL]ETHYLENEDIAMINE	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.

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	For N-[3-(trimethoxysilyl)propyl]ethylenediamine (AEAPTMS) and its analogues: Acute toxicity: In rabbits, AEAPTMS is moderately irritating to the skin and severely irritating to the eyes. AEAPTMS showed a skin sensitizing potential in a guinea pig maximisation test. Repeat dose toxicity: AEAPTMS was tested in rats in a combined repeated dose toxicity test with a reproductive/ developmental screening test, following the OECD test guideline 422 (28-39 days). Clinical findings attributed to the test substance included clear perioral soiling in several high dose animals and either increased nasal sounds, labored respiration, or soft vocalizations in approximately half of the high dose females and one high dose male. These signs were not seen in the control animals and infrequently seen in either of the two lower dose groups. Observations recorded at dosing indicated a dose-related resistance to dosing. Evaluating all 30 animals/dose over the entire dosing period, the incidence of resistance was 3, 5, 27 and 62% for the controls, 25, 125 and 500 mg/kg bw/day dose groups, respectively. Similar incidence patterns were noted for salivation just prior to dosing, wetness around the mouth at dosing, and wetness around the mouth 5-30 minutes following dosing. These clinical findings are anticipated based on the amine-functionality of the material and indicative of irritation, rather than systemic effects. There were no test substance-related effects on body weight, organ weights or organ-to-body weight ratios, food consumption, FOB or motor activity parameters, or haematology or serum chemistry parameters, and no macroscopic or microscopic findings were attributed to the test-substance. Based on the results of this study, the NOAEL for the systemic toxicity of this material in the rat via oral dosing for at least 28 consecutive days was considered to be 500 mg/kg bw/day. Genetic toxicity: AEAPTMS has been tested in an Ames test, an <i>in vitro</i> Chinese hamster ovary cell HGPRT assay and sister chromatid exchange assay, and an <i>in vivo</i> mouse micronucleus assay. These <i>in vivo</i> and <i>in vitro</i> screening assays have not revealed any evidence of genotoxic potential of AEAPTMS. Reproductive and developmental toxicity: Rats exposed to AEAPTMS by gavage to doses of 0, 25, 125, and 500 mg/kg bw/day, as part of an OECD guideline 422 study, no test substance-related effects were observed in any of the reproductive parameters evaluated. Based on the results of this reproductive/developmental screening study, the NOAEL for maternal (systemic toxicity) and developmental toxicity of AEAPTMS in the rat via the oral dosing was 500 mg/kg bw/day (the highest dose tested).
DIBUTYLTIN BIS(ACETYLACETONATE)	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
N-[3-(DIMETHOXYMETHYLSILYL)PROPYL]ETHYLENEDIAMINE	The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence). The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.
AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY) & N-[3-(TRIMETHOXSILYL)PROPYL]ETHYLENEDIAMINE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY) & DIISONONYL PHTHALATE	The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens. However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure.
LIMESTONE & N-[3-(TRIMETHOXSILYL)PROPYL]ETHYLENEDIAMINE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
TRIMETHOXYVINYLILANE & N-[3-(TRIMETHOXSILYL)PROPYL]ETHYLENEDIAMINE & DIBUTYLTIN BIS(ACETYLACETONATE) & N-[3-(DIMETHOXYMETHYLSILYL)PROPYL]ETHYLENEDIAMINE & BIS(2,2,6,6-TETRAMETHYL-4-PIPERIDINYL)SEBACATE	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.
TRIMETHOXYVINYLILANE & N-[3-(DIMETHOXYMETHYLSILYL)PROPYL]ETHYLENEDIAMINE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
TRIMETHOXYVINYLILANE & N-[3-(TRIMETHOXSILYL)PROPYL]ETHYLENEDIAMINE & N-[3-(DIMETHOXYMETHYLSILYL)PROPYL]ETHYLENEDIAMINE	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.
DIBUTYLTIN BIS(ACETYLACETONATE) & N-[3-(DIMETHOXYMETHYLSILYL)PROPYL]ETHYLENEDIAMINE	No significant acute toxicological data identified in literature search.

AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY)

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

AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
limestone	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	1h	Fish	4-320mg/l	4
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	LC50	96h	Fish	>165200mg/L	4
diisononyl phthalate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	>0.034mg/l	1
	EC50	72h	Algae or other aquatic plants	>88mg/l	2
	EC50	48h	Crustacea	>0.086mg/l	1
	LC50	96h	Fish	>0.1mg/l	2
trimethoxyvinylsilane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>89mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	48h	Crustacea	1mg/l	2
	LC50	96h	Fish	>92.2mg/l	2
N-[3-(trimethoxysilyl)propyl]ethylenediamine	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	5.5mg/l	2
	EC50	48h	Crustacea	81mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1.6mg/l	2
	LC50	96h	Fish	597mg/l	2
dibutyltin bis(acetylacetonate)	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>2mg/l	2
	EC50	48h	Crustacea	0.004mg/l	2
	EC50(ECx)	48h	Crustacea	0.004mg/l	2
	LC50	96h	Fish	>2mg/l	2
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	5.5mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	597mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1.6mg/l	2
bis(2,2,6,6-tetramethyl-4-piperidinyl)sebacate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	24h	Crustacea	20mg/l	Not Available
	EC50	72h	Algae or other aquatic plants	0.705mg/l	2
	LC50	96h	Fish	7.9mg/l	Not Available

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

Continued...

AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY)

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

for phthalate esters:

Phthalates are easily released into the environment. In general, they do not persist due to rapid biodegradation, photodegradation, and anaerobic degradation. Outdoor air concentrations are higher in urban and suburban areas than in rural and remote areas. They also pose no acute toxicity. In general, children's exposure to phthalates is greater than that of adults.

Environmental fate;

Under aerobic and anaerobic conditions, studies reveal that many phthalate esters are degraded by a wide range of bacteria and actinomycetes. Standardized aerobic biodegradation tests with sewage sludge inocula show that within 28 days approximately 50% ultimate degradation occurs. Biodegradation is, therefore, expected to be the dominant pathway in surface soils and sediments. In the atmosphere, photodegradation via free radical attack is the anticipated dominant pathway. The half-life of many phthalate esters is ca. 1 day in the air, from < 1 day to 2 weeks in surface and marine waters, and from < 1 week to several months in soils.

Phthalates are high molecular weight chemicals, and are not expected to partition significantly to air. However for the minor amount that may partition to air, modelled predictions indicate that they would be rapidly oxidised: with a predicted atmospheric oxidation half-life of around 0.52 days. They are expected to react appreciably with other photo oxidative species in the atmosphere, such as O₃. Therefore, it is expected that reactions with hydroxyl radicals will be the most important fate process in the atmosphere for phthalates.

Bioaccumulation of phthalate esters in the aquatic and terrestrial food chain is limited by biotransformation.

Most phthalates have experimental bioaccumulation factor (BCFs) and bioconcentration factor (BAFs) below 5000 L/kg, as they are readily metabolised by fish.

A study of 18 commercial phthalate esters with alkyl chains ranging from one to 13 carbons found an eight order of magnitude increase in octanol-water coefficients (Kow) and a four order of magnitude decrease in vapor pressure with increasing length. This increase in Kow and decrease in vapor pressure results in increased partitioning of the phthalate esters to suspended solids, soils, sediments, and aerosols.

The phthalate esters are distributed throughout the environment ubiquitously. They are found complexed with fulvic acid components of the humic substances in soil and marine and estuarine waters. Fulvic acid appears to act as a solubiliser for the otherwise insoluble ester and serves to mediate its transport and mobilisation in water or immobilisation in soil.

Phthalate esters have been found in open ocean environments, in deep sea jelly fish, Atlantic herring and in mackerel. Phthalic ester plasticisers are clearly recognised as general contaminants of almost every soil and water ecosystem. In general they have low acute toxicity but the weight of evidence supporting their carcinogenicity is substantial. Other subtle chronic effects have also been reported. As little as 4 µg/ml in culture medium is lethal to chick embryo heart cells. This concentration is similar to that reached in human blood stored in vinyl plastic bags for as little as one day. As phthalates are present in drinking water and food, concerns have been raised about their long term effects on humans.

Ecotoxicity:

Some phthalates (notably di-2-ethylhexyl phthalate and dibutyl phthalate) may be detrimental to the reproduction of the water flea (*Daphnia magna*), zebra fish and guppies.

While phthalates may have very low true water solubilities, they possess the ability to form suspensions which may cause adverse effects through physical contact with *Daphnia* at very low concentrations.

Available toxicity and water solubility information suggest that the high molecular weight phthalates, form these suspensions and are able to elicit chronic toxic effects at concentrations of approximately 0.05 mg/L. Therefore, these substances are considered to have the potential to harm aquatic organisms at relatively low concentrations.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
diisononyl phthalate	HIGH	HIGH
trimethoxyvinylsilane	HIGH	HIGH
N-[3-(trimethoxysilyl)propyl]ethylenediamine	HIGH	HIGH
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	HIGH	HIGH
bis(2,2,6,6-tetramethyl-4-piperidinyl)sebacate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
diisononyl phthalate	LOW (BCF = 183.8)
trimethoxyvinylsilane	LOW (LogKOW = -0.3169)
N-[3-(trimethoxysilyl)propyl]ethylenediamine	LOW (LogKOW = -1.6744)
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	LOW (LogKOW = -0.4178)
bis(2,2,6,6-tetramethyl-4-piperidinyl)sebacate	HIGH (LogKOW = 6.5004)

Mobility in soil

Ingredient	Mobility
diisononyl phthalate	LOW (KOC = 467200)
trimethoxyvinylsilane	LOW (KOC = 757.6)
N-[3-(trimethoxysilyl)propyl]ethylenediamine	LOW (KOC = 6856)
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	LOW (KOC = 3451)
bis(2,2,6,6-tetramethyl-4-piperidinyl)sebacate	LOW (KOC = 609900)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible. Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. - Recycle wherever possible or consult manufacturer for recycling options. - Consult State Land Waste Authority for disposal. - Bury or incinerate residue at an approved site. - Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO

Land transport (DOT)

UN number	3077	
UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. (contains bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate and dibutyltin bis(acetylacetonate))	
Transport hazard class(es)	Class	9
	Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Not Applicable	
Special precautions for user	Hazard Label	9
	Special provisions	8, 146, 335, 384, A112, B54, B120, IB8, IP3, N20, N91, T1, TP33

For Individual Packages of Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 that contain LESS THAN the reportable quantity (5 kg or 5 L) - Not Regulated
For Individual Packages of Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 that contain MORE THAN the reportable quantity (5 kg or 5 L) - Regulated and classified as below:

Air transport (ICAO-IATA / DGR)

UN number	3077	
UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. (contains bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate and dibutyltin bis(acetylacetonate))	
Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	9L
Packing group	III	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions	A97 A158 A179 A197 A215
	Cargo Only Packing Instructions	956
	Cargo Only Maximum Qty / Pack	400 kg
	Passenger and Cargo Packing Instructions	956
	Passenger and Cargo Maximum Qty / Pack	400 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Y956
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3077	
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains bis(2,2,6,6-tetramethyl-4-piperidiny)sebacate and dibutyltin bis(acetylacetonate))	
Transport hazard class(es)	IMDG Class	9
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Not Applicable	
Special precautions for user	EMS Number	F-A, S-F
	Special provisions	274 335 966 967 969
	Limited Quantities	5 kg

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

Not Applicable

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

Product name	Group
limestone	Not Available
diisononyl phthalate	Not Available
trimethoxyvinylsilane	Not Available

AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY)

Product name	Group
N-[3-(trimethoxysilyl)propyl]ethylenediamine	Not Available
dibutyltin bis(acetylacetonate)	Not Available
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	Not Available
bis(2,2,6,6-tetramethyl-4-piperidinyl)sebacate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
limestone	Not Available
diisononyl phthalate	Not Available
trimethoxyvinylsilane	Not Available
N-[3-(trimethoxysilyl)propyl]ethylenediamine	Not Available
dibutyltin bis(acetylacetonate)	Not Available
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine	Not Available
bis(2,2,6,6-tetramethyl-4-piperidinyl)sebacate	Not Available

SECTION 15 Regulatory information

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

limestone is found on the following regulatory lists	
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)	US OSHA Permissible Exposure Limits (PELs) Table Z-1
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5	US OSHA Permissible Exposure Limits (PELs) Table Z-3
US - Massachusetts - Right To Know Listed Chemicals	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US DOE Temporary Emergency Exposure Limits (TEELs)	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US NIOSH Recommended Exposure Limits (RELs)	
diisononyl phthalate is found on the following regulatory lists	
Chemical Footprint Project - Chemicals of High Concern List	US EPA Integrated Risk Information System (IRIS)
US - California Proposition 65 - Carcinogens	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US - California Proposition 65 - No Significant Risk Levels (NSRLs) for Carcinogens	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List	US TSCA Section 4/12 (b) - Sunset Dates/Status
US CWA (Clean Water Act) - Toxic Pollutants	
trimethoxyvinylsilane is found on the following regulatory lists	
US DOE Temporary Emergency Exposure Limits (TEELs)	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory	
N-[3-(trimethoxysilyl)propyl]ethylenediamine is found on the following regulatory lists	
US DOE Temporary Emergency Exposure Limits (TEELs)	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory	
dibutyltin bis(acetylacetonate) is found on the following regulatory lists	
Chemical Footprint Project - Chemicals of High Concern List	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US NIOSH Recommended Exposure Limits (RELs)	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US OSHA Permissible Exposure Limits (PELs) Table Z-1	
N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine is found on the following regulatory lists	
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory	US TSCA Chemical Substance Inventory - Interim List of Active Substances
bis(2,2,6,6-tetramethyl-4-piperidinyl)sebacate is found on the following regulatory lists	
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)	US OSHA Permissible Exposure Limits (PELs) Table Z-3
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US NIOSH Recommended Exposure Limits (RELs)	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US OSHA Permissible Exposure Limits (PELs) Table Z-1	

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories	
Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No

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Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	No
Acute toxicity (any route of exposure)	No
Reproductive toxicity	Yes
Skin Corrosion or Irritation	No
Respiratory or Skin Sensitization	Yes
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	No
Aspiration Hazard	No
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	Yes

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)

None Reported

State Regulations

US. California Proposition 65



WARNING: This product can expose you to chemicals including **diisononyl phthalate**, which is known to the State of California to cause cancer. For more information, go to www.P65Warnings.ca.gov.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (diisononyl phthalate; trimethoxyvinylsilane; N-[3-(trimethoxysilyl)propyl]ethylenediamine; dibutyltin bis(acetylacetonate); N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine; bis(2,2,6,6-tetramethyl-4-piperidinyl)sebacate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (trimethoxyvinylsilane; N-[3-(trimethoxysilyl)propyl]ethylenediamine; dibutyltin bis(acetylacetonate); N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine)
Vietnam - NCI	Yes
Russia - FBEPH	No (dibutyltin bis(acetylacetonate); N-[3-(dimethoxymethylsilyl)propyl]ethylenediamine)
Legend:	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.

SECTION 16 Other information

Revision Date	01/09/2023
Initial Date	01/09/2023

CONTACT POINT

PLEASE NOTE THAT TITANIUM DIOXIDE IS NOT PRESENT IN CLEAR OR NEUTRAL BASES

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.

Continued...

AP-1 GREEN-SEAL® HEAVY DUTY CONSTRUCTION SEALANT AND ADHESIVE (GRAY)

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

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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