Antas AR15 Acrylic Gap Filler

Antas Sealants and Adhesives

Part Number: **66153**
Version No: **1.2.8.7**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

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

Product Identifier

Product name	Antas AR15 Acrylic Gap Filler
Chemical Name	Not Applicable
Synonyms	ATAR15
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	construction, painting

Details of the supplier of the safety data sheet

Registered company name	**Antas Sealants and Adhesives**
Address	21-23 Pavesi Street Smithfield NSW 2164 Australia
Telephone	+61 2 9157 0368
Fax	Not Available
Website	www.antas.com.au
Email	info@antas.com.au

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone numbers	**+61 2 9157 0368**
Other emergency telephone numbers	**National Poison Information Centre 13 11 26**

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification [1]	Serious Eye Damage/Eye Irritation Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Skin Corrosion/Irritation Category 2, Reproductive Toxicity Category 1B, Skin Sensitizer Category 1, Carcinogenicity Category 2, Acute Aquatic Hazard Category 2
Legend:	*1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	

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Signal word	**Danger**

Hazard statement(s)

H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H315	Causes skin irritation.
H360FD	May damage fertility. May damage the unborn child.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.
H401	Toxic to aquatic life.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
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.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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
6846-50-0	<5	2,2,4-trimethyl-1,3-pentanediol diisobutyrate
471-34-1	30-50	calcium carbonate
117-81-7	<3	di-sec-octyl phthalate

Legend:	*1. Classification by vendor; 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

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Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ‣ Immediately hold eyelids apart and flush the eye continuously with running water. ‣ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ‣ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ‣ Transport to hospital or doctor without delay. ‣ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ‣ Immediately remove all contaminated clothing, including footwear. ‣ Flush skin and hair with running water (and soap if available). ‣ Seek medical attention in event of irritation.
Inhalation	‣ If fumes 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.
Ingestion	‣ Immediately give a glass of water. ‣ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

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	‣ 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. May emit poisonous fumes. May emit corrosive fumes. Heating calcium carbonate at high temperatures(825 C.) causes decomposition, releases carbon dioxide gas and leaves a residue of alkaline lime
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Continued...

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. ▸ 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	Calcium carbonate: ▸ is incompatible with acids, ammonium salts, fluorine, germanium, lead diacetate, magnesium, mercurous chloride, silicon, silver nitrate, titanium. Contact with acid generates carbon dioxide gas, which may pressurise and then rupture closed containers Phthalates: ▸ react with strong acids, strong oxidisers, permanganates and nitrates ▸ attack some form of plastics ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

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	▸ Avoid reaction with oxidising agents

+	x	+	x	+	+	+

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	di-sec-octyl phthalate	Di-sec-octyl phthalate	5 mg/m3	10 mg/m3	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
calcium carbonate	45 mg/m3	210 mg/m3	1,300 mg/m3
di-sec-octyl phthalate	10 mg/m3	1,000 mg/m3	6,100 mg/m3

Ingredient	Original IDLH	Revised IDLH
2,2,4-trimethyl-1,3-pentanediol diisobutyrate	Not Available	Not Available
calcium carbonate	Not Available	Not Available
di-sec-octyl phthalate	5,000 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
2,2,4-trimethyl-1,3-pentanediol diisobutyrate	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

For calcium carbonate:
The TLV-TWA is thought to be protective against the significant risk of physical irritation associated with exposure.

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. 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.

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Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

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.

Body protection

See Other protection below

Other protection

- Overalls.
- P.V.C apron.
- Barrier cream.
- Skin cleansing cream.
- Eye wash unit.

Respiratory protection

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, ANSI Z88 or national equivalent)

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	P1 Air-line*	- -	PAPR-P1 -
up to 50 x ES	Air-line**	P2	PAPR-P2
up to 100 x ES	-	P3	-

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		Air-line*	-
100+ x ES	-	Air-line**	PAPR-P3

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	The phthalates have a clear syrupy liquid consistency and show low water solubility, high oil solubility, and low volatility. The polar carboxyl group contributes little to the physical properties of the phthalates, except when R and R' are very small (such as ethyl or methyl groups). Phthalates are colourless, odourless liquids produced by reacting phthalic anhydride with an appropriate alcohol (usually 6- to 13-carbon). Phthalate esters are the dialkyl or alkyl aryl esters of phthalic acid (also called 1,2-benzenedicarboxylic acid). When added to plastics, phthalates allow the long polyvinyl molecules to slide against one another. White

Physical state	Non Slump Paste	**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**	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)	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 (%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	Product is considered stable and 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	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.
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	The material may accentuate any pre-existing dermatitis condition 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 produces moderate skin irritation; evidence exists, or practical experience predicts, 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	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. 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 than can cuase 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 cuase 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

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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 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 carbohdrate metabolism.

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	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) Pure calcium carbonate does not produce pneumoconiosis probably being eliminated from the lungs slowly by solution. As mined, unsterilised particulates can carry bacteria into the air passages and lungs, producing infection and bronchitis. For di-sec-octyl phthalate: Oral studies of 90-days to 2-years in rat, 1-year in guinea pig and up to 1-year in dog have shown a no-effect level of about 60 mg/kg/day. Higher doses produced growth retardation and increased weights of livers and kidneys. Rats and mice fed on diets containing 6000-12000 (rats) and 3000-6000 (mice) mg/kg body weight for 103 weeks showed an increased incidence of hepatocellular carcinomas in female rats and male and female mice, and an increased incidence of either hepatocellular carcinomas or neoplastic nodules in male rats. About 35% of the hepatocellular carcinomas in mice had metastasised to the lungs. The substance can cause testicular damage in rats (dietary and gavage studies) with a no-effect level in 0.3% to 0.5% in the diet. Inhalation or dermal exposures did not produce testicular effects. When the substance was fed to pregnant rats (5 ml/kg) it produced slight effects on embryonic and foetal development with skeletal abnormalities more common.

Antas AR15 Acrylic Gap Filler	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
2,2,4-trimethyl-1,3-pentanediol diisobutyrate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye (rabbit): very slight**
	Oral(Rat) LD50; >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin (guinea pig): 5000mg/kg-mild
		Skin: no adverse effect observed (not irritating)[1]
calcium carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 0.75 mg/24h - SEVERE
	Inhalation(Rat) LC50; >3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50; >2000 mg/kg[1]	Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating)[1]
di-sec-octyl phthalate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 250 mg/kg[2]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; >10.62 mg/l4h[2]	Eye: adverse effect observed (irritating)[1]
	Oral(Mouse) LD50; >9860 mg/kg[2]	Skin (rabbit): 500 mg/24h mild
		Skin: adverse effect observed (irritating)[1]
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

2,2,4-TRIMETHYL-1,3-PENTANEDIOL DIISOBUTYRATE	NOAEL oral (rat), 103 days = 1% in diet *** NOEL oral (dog), 90 days = 1% in diet *** Mutagenicity/Genotoxicity Data: *** Chromosomal aberration assay: Negative (+/- activation) CHO/HGPRT assay: Negative (+/- activation) Salmonella-E.coli reverse mutation assay (Ames test): Negative (+/- activation) *,**,*** Various suppliers MSDS Sensitization Species:Guinea pig: Result: sensitizing Effects on foetal development: Species: Rabbit Application Route: Oral Developmental Toxicity: NOAEL: 300 mg/kg body weight Reproductive toxicity;Assessment: Some evidence of adverse effects on development, based on animal experiments. * Eastman Benzoflex 6000 Plasticiser For 2,2,4-trimethyl-1,3-pentanediol diisobutyrate (TXIB) TXIB showed no genotoxic effects in bacteria and chromosomal aberration test *in vitro*. **Reproductive/ developmental toxicity:** In a combined repeat dose and reproductive/developmental toxicity screening test, increase of liver and kidney weights were observed in parental animals from the middle dose level (150 mg/kg/day). In the histopathological examinations, increases in grade of basophilic change of renal tubular epithelium and degeneration of hyaline droplet were observed from the same level. In addition, necrosis and other renal effects were also observed. From the view point of reproductive/developmental end-points, there were no effects observed related to mating, fertility and oestrus cycle and also for dams during the pregnancy and lactation period and for pups after their birth. Therefore, NOEL was 30 mg/kg/day for repeated dose toxicity as well as 750 mg/kg/day for reproductive toxicity. **Genotoxicity:** The chemical showed no genotoxic effects in bacteria and chromosomal aberration tests *in vitro*
CALCIUM CARBONATE	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to

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	irritants may produce conjunctivitis. 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.
DI-SEC-OCTYL PHTHALATE	Di-sec-octyl phthalate (DEHP) is not acutely toxic in small laboratory animals via the oral route. The oral LD50 reported for mice is 26.3 g/kg; for rats, it is 33.8 g/kg . No skin irritation or sensitisation potential has been demonstrated in either animals or humans, and the lethal dermal dose in rabbits is about 25 ml/kg. Deaths in rats and chronic diffuse inflammation of the lung in mice exposed to DEHP at unspecified levels have been reported. Long-term dietary toxicity studies in rats, guinea pigs, and dogs have established a no-effect dose level of about 60 mg/kg/day, and no carcinogenic or histologic abnormalities were observed at this level . Higher doses were associated with growth retardation and increased liver and kidney weights but not histologic abnormalities. Metabolic studies have demonstrated that laboratory animals do not appreciably metabolise DEHP . Teratogenicity studies in pregnant rats indicated that fertility is unaffected at doses of 0.1, 0.2, or 0.33 percent of the acute intraperitoneal LD50 dose for rats, although slight effects on embryonic and foetal development were observed in these animals; skeletal deformities were the most common teratogenic effects observed . Mutagenic effects were observed at intravenous doses of one-third, one-half, and two-thirds of the acute LD50; these effects are consistent with DEHP's ability to produce dominant lethal mutations . A study of workers exposed to a mixture of the vapors of diethyl phthalate, dibutyl phthalate, and di-2-ethylhexyl phthalate reported that exposures to 1 to 6 ppm caused no peripheral polyneuritis However, Russian investigators examined male and female workers exposed to between 1.7 and 66 mg/m3 of various combinations of airborne phthalates (including butyl phthalate, higher aryl phthalates, dioctyl phthalate and others) and noted complaints of pain, numbness, and spasms in the upper and lower extremities after six to seven years of exposure. Polyneuritis was observed in 32 percent of the workers studied, and 78 percent of these workers showed depression of vestibular receptors The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. **Transitional Phthalate Esters:** produced from alcohols with straight-chain carbon backbones of C4 to C6. This subcategory also includes a phthalate produced from benzyl alcohol as one ester group with the second ester composed of an alkyl group with a C5 carbon backbone and butyrate group. Phthalate esters containing >10% C4 to C6 molecules were conservatively included in this subcategory. Branched C7 and C8 isomers (di-iso-heptyl, di-iso-octyl and diethylhexyl phthalates) in contrast to linear dihexyl and dioctyl phthalate are members of this family. Transitional phthalates have varied uses, but are largely used as plasticisers for PVC. Physicochemical properties also vary in that the lower molecular weight transitional phthalates are more water-soluble than higher molecular weight transitional phthalates, but none would be characterised as highly water soluble. Transitional phthalates have lower water solubility than the low molecular weight phthalates and except for butylbenzyl phthalate (BBP), existing data suggest they do not exhibit acute or chronic aquatic toxicity. What distinguishes some of the transitional phthalates from others is their greater mammalian toxicity potential, particularly with regard to reproductive and developmental effects, compared to either the low or high molecular weight phthalate subcategories **Acute Toxicity.** The available data on phthalates spanning the carbon range from C4 to C6 indicate that phthalate esters in the transitional subcategory are minimally toxic by acute oral and dermal administration. The oral LD50 value for BBP exceeds 2 g/kg, and for materials with higher molecular weights, the LD50 values exceed the maximum amounts which can be administered to the animals in a manner consistent with the principles of responsible animal use. One member of this subcategory, diethylhexyl phthalate (DEHP), has been tested for acute inhalation toxicity. It did not cause an effect at the highest concentration tested. Further, considering the low volatility of these substances, inhalation exposure at toxicologically significant levels is not anticipated. **Repeated Dose Toxicity.** Several substances in the C4 to C6 range, including BBP, have been tested for repeated dose toxicity in studies ranging from 3 weeks to 2 years . The principal effects found in these studies were those associated with peroxisome proliferation including liver enlargement and induction of peroxisomal enzymes. As shown in a comparative study of liver effects, the strongest inducers of peroxisome proliferation are diisononyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) with substances of shorter chain length (e.g., BBP) showing much less pronounced effects. Thus it is reasonable to conclude that other members of this subcategory would show effects similar to BBP and less pronounced than DINP or 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 (PPARa) and that levels of PPARa are much higher in rodents than they are in humans. Thus one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence that this is true is provided by studies in primates in which repeated administration of DINP had no effects on liver, kidney or testicular parameters. Several of the substances in the transitional phthalate esters subcategory, however, have been shown to produce testicular atrophy when given to juvenile rats at high levels. Testicular atrophy has been associated with BBP and other substances with C4 to C6 linear carbon chains. However, molecules with fewer than 4 or more than 6 carbons did not produce testicular atrophy in these studies. Although the relevance of these data are uncertain, as the testes is not a target organ for diethylhexyl phthalate (DEHP) in primates, these data do provide one of the distinguishing toxicological characteristics of this subcategory and are one of the underlying reasons supporting the differentiation of phthalate esters on the basis of length of the linear region of the carbon chain. **Genetic Toxicity (Salmonella)**. A number of the substances in this subcategory including the reference substance BBP has been assessed in the Salmonella and mouse lymphoma assays. All of these substances were inactive in these assays. **Chromosomal Aberrations**. BBP and dihexyl phthalate (DHP) were inactive in micronucleus assays in mice. DEHP was inactive in a cytogenetics assay in rat bone marrow. Diisoheptyl phthalate was inactive in CHO cells, in vitro.. **Reproductive toxicity:** A series of studies assessed the structure-activity relationship of the effects of phthalate esters on fertility using a continuous breeding protocol . The test substances included in these studies were diethyl-, dipropyl-, dibutyl-, dipentyl-, d-n-hexyl-, di-2(ethylhexyl)-, and di-n-octyl phthalates. The most profound effects were on fertility (i.e., number of females delivering/ number mated) and number of live births. The substance showing the greatest activity was DEHP which produced effects at dietary levels of 0.1 % with a no effect level of 0.01 %. The next most active compounds were di-n-hexyl- and di-n-pentyl phthalate which showed effects in the range of 0.3 to 0.5 %; no effect levels were not experimentally defined. Dipropyl phthalate had an effect on live birth index at 2.5 % but produced no effects at 1.25 %. Diethyl phthalate and di-n-octyl phthalate

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	were inactive at the highest levels tested, 2.5 % and 5.0 %, respectively. These data demonstrated that molecules with linear alkyl chains of 4 to **6** carbons profoundly affect fertility in rodents, with DEHP being the most active. Molecules with longer or shorter side chains are essentially inactive in these assays. These data were also a basis for the separation of phthalates into three categories based on length of side chain. In addition to these data there are reproductive toxicity studies on BBP and DEHP . A 2-generation reproductive study was conducted in rats in which BBP was administered via the diet. Parental effects were limited to changes in body weight, weight gain, and increased absolute and relative liver weights. In the F1 parents, treatment with BBP affected mating and fertility indices and sperm number and motility. The F1 male offspring exhibited shortened anogenital distance, delayed acquisition of puberty and retention of nipples and areolae as well as reproductive effects. The NOAEL of the study was reported to be 3750 mg/ kg for reproductive effects. However, for male F1 and F2 offspring, the NOEL for reproductive effects was reported to be 50 mg/ kg based on reductions in anogenital distance. These studies along with previous data provide a good basis to assess the reproductive effects of C4 to C6 phthalate esters. Although several substances (diheptyl, heptyl nonyl, heptyl undecyl) have ester side chain constituents that predominately fall in the high molecular weight subcategory, these substances are conservatively assumed to exhibit reproductive effects similar to other transitional phthalates . **Developmental toxicity:** There have been extensive studies of the developmental toxicity of BBP and DEHP. These substances produce structural malformations and also affect male reproductive development. No effect levels are in the range of 50 to 300 mg/ kg bw/ day. There is also an unpublished developmental toxicity study of di-isoheptyl phthalate (DIHP). The results of these studies are broadly consistent with the structure-activity relationships previously described, i.e., that phthalate esters with linear carbon chains of C4 to C6 carbons produce much more profound effects that either shorter or longer molecules. Phthalate esters with >10% C4 to C6 isomers were conservatively placed in the transitional subcategory. This conclusion is supported by developmental test data on "711P*" (which showed structural malformations in rats at 1000 mg/ kg/ day with a NOAEL of 200 mg/ kg/ day ."711P" is an equal composition mixture of six phthalate esters consisting of linear and methyl-branched C7, C9, and C11 ester side chains. This test substance is considered by EPA under the following CAS Numbers.: 68515-44-6 (di C7), 68515-45-7 (di C9), 3648-20-2 (di C1 I), 111381-89-6 (C7, C9), 111381-90-9 (C7, C11), and 111381-91-0 (C9, C11). The overall content of C4 to C6 isomers in "71 1P" is approximately 10%, based on the contribution from methyl-branched C7 isomers e.g., di C7 (30% C4-C6); C7, C9 (15% C4-C6); and C7, C11 (15 % C4-C6). Test data on 711P were used selectively as read-across data to the C7-containing substances in the mixture, based on the C4 to C6 content of each substance in the mixture. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*] Oral (rat) NOAEL: 28.9-36.1 mg/kg/day Gastrointestinal changes, respiratory system changes, somnolence, haemorrhage, necrotic changes in GI tract, lowered blood pressure, liver, endocrine tumours, foetotoxicity, paternal effects, maternal effects, specific developmental abnormalities (hepatobiliary system, musculoskeletal system, cardiovascular system, urogenital system, central nervous system, eye/ear), foetolethality recorded.
Antas AR15 Acrylic Gap Filler & CALCIUM CARBONATE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
Antas AR15 Acrylic Gap Filler & 2,2,4-TRIMETHYL-1,3-PENTANEDIOL DIISOBUTYRATE	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.
Antas AR15 Acrylic Gap Filler & DI-SEC-OCTYL 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.
2,2,4-TRIMETHYL-1,3-PENTANEDIOL DIISOBUTYRATE & DI-SEC-OCTYL PHTHALATE	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.

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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

Antas AR15 Acrylic Gap Filler

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

2,2,4-trimethyl-1,3-pentanediol diisobutyrate

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	0.6-0.8	7
NOEC(ECx)	504h	Crustacea	0.7mg/l	2
EC50	72h	Algae or other aquatic plants	>7.49mg/l	2
LC50	96h	Fish	>1.55mg/l	2
EC50	48h	Crustacea	>1.46mg/l	1

calcium carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	6h	Fish	4-320mg/l	4
EC50	72h	Algae or other aquatic plants	>14mg/l	2
LC50	96h	Fish	>165200mg/L	4

di-sec-octyl phthalate

Endpoint	Test Duration (hr)	Species	Value	Source
ErC50	72h	Algae or other aquatic plants	>130mg/l	1
EC50	72h	Algae or other aquatic plants	>130mg/l	1
EC50	48h	Crustacea	>0.16mg/l	1
LC50	96h	Fish	>0.16mg/l	2
BCF	1344h	Fish	<0.7-29.7	7
NOEC(ECx)	1680h	Fish	0.007mg/l	1
EC50	96h	Algae or other aquatic plants	>0.1mg/l	1

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

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
Toxic to aquatic organisms.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
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,

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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 ug/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
2,2,4-trimethyl-1,3-pentanediol diisobutyrate	HIGH	HIGH
di-sec-octyl phthalate	HIGH (Half-life = 389 days)	LOW (Half-life = 1.21 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
2,2,4-trimethyl-1,3-pentanediol diisobutyrate	LOW (BCF = 1)
di-sec-octyl phthalate	HIGH (BCF = 24500)

Mobility in soil

Ingredient	Mobility
2,2,4-trimethyl-1,3-pentanediol diisobutyrate	LOW (KOC = 607.5)
di-sec-octyl phthalate	LOW (KOC = 165400)

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. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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
2,2,4-trimethyl-1,3-pentanediol diisobutyrate	Not Available
calcium carbonate	Not Available
di-sec-octyl phthalate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
2,2,4-trimethyl-1,3-pentanediol diisobutyrate	Not Available
calcium carbonate	Not Available
di-sec-octyl phthalate	Not Available

SECTION 15 Regulatory information

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

2,2,4-trimethyl-1,3-pentanediol diisobutyrate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

calcium carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

di-sec-octyl phthalate 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 10 / Appendix C
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (2,2,4-trimethyl-1,3-pentanediol diisobutyrate; di-sec-octyl phthalate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes

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National Inventory	Status
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	30/06/2021
Initial Date	30/06/2021

SDS Version Summary

Version	Date of Update	Sections Updated
0.0.2.1	26/04/2021	Regulation Change
0.0.3.1	03/05/2021	Regulation Change
0.0.4.1	06/05/2021	Regulation Change
0.0.5.1	10/05/2021	Regulation Change
0.0.5.2	30/05/2021	Template Change
0.0.5.3	04/06/2021	Template Change
0.0.5.4	05/06/2021	Template Change
0.0.6.4	07/06/2021	Regulation Change
0.0.6.5	09/06/2021	Template Change
0.0.6.6	11/06/2021	Template Change
0.0.6.7	15/06/2021	Template Change
0.0.7.7	17/06/2021	Regulation Change
0.0.8.7	21/06/2021	Regulation Change

Other information

Ingredients with multiple cas numbers

Name	CAS No
calcium carbonate	471-34-1, 13397-26-7, 15634-14-7, 1317-65-3, 72608-12-9, 878759-26-3, 63660-97-9, 459411-10-0, 198352-33-9, 146358-95-4, 1357-85-3

Classification of the preparation and its individual components has drawn on official and authoritative sources 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
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