Tensorgrip H21 2k Spray Foam Insulation Part B

QUIN GLOBAL ASIA PACIFIC

Chemwatch Hazard Alert Code: **3**

Version No: **3.3**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **15/03/2024**
Print Date: **15/03/2024**
L.GHS.AUS.EN

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

Product Identifier

Product name	Tensorgrip H21 2k Spray Foam Insulation Part B
Synonyms	Not Available
Proper shipping name	CHEMICAL UNDER PRESSURE, N.O.S.
Other means of identification	Not Available

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

Relevant identified uses	Foam Insulation

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**QUIN GLOBAL ASIA PACIFIC**
Address	63 Hincksman Street Queanbeyan, NSW 2620 Australia
Telephone	+61 2 6175 0574
Fax	Not Available
Website	www.quinglobal.com
Email	sales@quinglobal.com.au

Emergency telephone number

Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Gases Under Pressure (Liquefied Gas), Acute Toxicity (Oral) Category 4, Sensitisation (Skin) Category 1, Carcinogenicity Category 2, Reproductive Toxicity Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	
Signal word	**Warning**

Hazard statement(s)

H280	Contains gas under pressure; may explode if heated.
H302	Harmful if swallowed.

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H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.
H361d	Suspected of damaging the unborn child.
AUH044	Risk of explosion if heated under confinement.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves and protective clothing.
P261	Avoid breathing gas.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P272	Contaminated work clothing should 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.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P403	Protect from sunlight. Store in a well-ventilated place.

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
13674-84-5	5-30	tris(2-chloroisopropyl)phosphate
25214-63-5	<10	ethylenediamine, propoxylated
Not Available	Balance	Non-hazardous ingredients
811-97-2	10-20	1,1,1,2-tetrafluoroethane
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	▸ If product comes in contact with eyes remove the patient from gas source or contaminated area. ▸ Take the patient to the nearest eye wash, shower or other source of clean water. ▸ Open the eyelid(s) wide to allow the material to evaporate. ▸ Gently rinse the affected eye(s) with clean, cool water for at least 15 minutes. Have the patient lie or sit down and tilt the head back. Hold the eyelid(s) open and pour water slowly over the eyeball(s) at the inner corners, letting the water run out of the outer corners. ▸ The patient may be in great pain and wish to keep the eyes closed. It is important that the material is rinsed from the eyes to prevent further damage. ▸ Ensure that the patient looks up, and side to side as the eye is rinsed in order to better reach all parts of the eye(s) ▸ Transport to hospital or doctor. ▸ Even when no pain persists and vision is good, a doctor should examine the eye as delayed damage may occur. ▸ If the patient cannot tolerate light, protect the eyes with a clean, loosely tied bandage. ▸ Ensure verbal communication and physical contact with the patient. **DO NOT** allow the patient to rub the eyes **DO NOT** allow the patient to tightly shut the eyes **DO NOT** introduce oil or ointment into the eye(s) without medical advice **DO NOT** use hot or tepid water.
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. In case of cold burns (frost-bite): ▸ Move casualty into warmth before thawing the affected part; if feet are affected carry if possible ▸ Bathe the affected area immediately in luke-warm water (not more than 35 deg C) for 10 to 15 minutes, immersing if possible and without

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	rubbing • **DO NOT** apply hot water or radiant heat. • Apply a clean, dry, light dressing of 'fluffed-up' dry gauze bandage • If a limb is involved, raise and support this to reduce swelling • If an adult is involved and where intense pain occurs provide pain killers such as paracetomol • Transport to hospital, or doctor • Subsequent blackening of the exposed tissue indicates potential of necrosis, which may require amputation.
Inhalation	• Following exposure to gas, remove the patient from the gas source or contaminated area. • NOTE: Personal Protective Equipment (PPE), including positive pressure self-contained breathing apparatus may be required to assure the safety of the rescuer. • Prostheses such as false teeth, which may block the airway, should be removed, where possible, prior to initiating first aid procedures. • If the patient is not breathing spontaneously, administer rescue breathing. • If the patient does not have a pulse, administer CPR. • If medical oxygen and appropriately trained personnel are available, administer 100% oxygen. • Summon an emergency ambulance. If an ambulance is not available, contact a physician, hospital, or Poison Control Centre for further instruction. • Keep the patient warm, comfortable and at rest while awaiting medical care. • **MONITOR THE BREATHING AND PULSE, CONTINUOUSLY.** • Administer rescue breathing (preferably with a demand-valve resuscitator, bag-valve mask-device, or pocket mask as trained) or CPR if necessary.
Ingestion	• Not considered a normal route of entry. • Avoid giving milk or oils. • Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

For frost-bite caused by liquefied petroleum gas:
- If part has not thawed, place in warm water bath (41-46 C) for 15-20 minutes, until the skin turns pink or red.
- Analgesia may be necessary while thawing.
- If there has been a massive exposure, the general body temperature must be depressed, and the patient must be immediately rewarmed by whole-body immersion, in a bath at the above temperature.
- Shock may occur during rewarming.
- Administer tetanus toxoid booster after hospitalization.
- Prophylactic antibiotics may be useful.
- The patient may require anticoagulants and oxygen.

[Shell Australia 22/12/87]
For gas exposures:

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for pulmonary oedema .
- Monitor and treat, where necessary, for shock.
- Anticipate seizures.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

BRONSTEIN, A.C. and CURRANCE, P.L.
EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

All persons handling organic phosphorus ester materials regularly should undergo regular medical examination with special stress on the central nervous systems. Whilst atropine or pyridine-2-aldoxime methiodide (PAM) are beneficial antidotes for acute phosphate ester poisonings, they are of little value in reversing acute or chronic neurological damage due to phosphites and some types of aryl phosphate.
for intoxication due to Freons/ Halons;
A: Emergency and Supportive Measures
- Maintain an open airway and assist ventilation if necessary
- Treat coma and arrhythmias if they occur. Avoid (adrenaline) epinephrine or other sympathomimetic amines that may precipitate ventricular arrhythmias. Tachyarrhythmias caused by increased myocardial sensitisation may be treated with propranolol, 1-2 mg IV or esmolol 25-100 microgm/kg/min IV.
- Monitor the ECG for 4-6 hours

B: Specific drugs and antidotes:
- There is no specific antidote

C: Decontamination
- Inhalation; remove victim from exposure, and give supplemental oxygen if available.
- Ingestion; (a) Prehospital: Administer activated charcoal, if available. **DO NOT** induce vomiting because of rapid absorption and the risk of abrupt onset CNS depression. (b) Hospital: Administer activated charcoal, although the efficacy of charcoal is unknown. Perform gastric lavage only if the ingestion was very large and recent (less than 30 minutes)

D: Enhanced elimination:
- There is no documented efficacy for diuresis, haemodialysis, haemoperfusion, or repeat-dose charcoal.

POISONING and DRUG OVERDOSE, Californian Poison Control System Ed. Kent R Olson; 3rd Edition
- Do not administer sympathomimetic drugs unless absolutely necessary as material may increase myocardial irritability.
- No specific antidote.
- Because rapid absorption may occur through lungs if aspirated and cause systematic effects, the decision of whether to induce vomiting or not should be made by an attending physician.
- If lavage is performed, suggest endotracheal and/or esophageal control.
- Danger from lung aspiration must be weighed against toxicity when considering emptying the stomach.
- Treatment based on judgment of the physician in response to reactions of the patient

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SECTION 5 Firefighting measures

Extinguishing media

SMALL FIRE: Use extinguishing agent suitable for type of surrounding fire.
LARGE FIRE: Cool cylinder.
DO NOT direct water at source of leak or venting safety devices as icing may occur.

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	--- GENERAL --- ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus and protective gloves. ‣ Fight fire from a safe distance, with adequate cover. ‣ Use water delivered as a fine spray to control fire and cool adjacent area. ‣ **DO NOT** approach cylinders suspected to be hot. ‣ Cool fire exposed cylinders with water spray from a protected location. ‣ If safe to do so, remove cylinders from path of fire. --- SPECIAL REQUIREMENTS: --- ‣ Excessive pressures may develop in a gas cylinder exposed in a fire; this may result in explosion. ‣ Cylinders with pressure relief devices may release their contents as a result of fire and the released gas may constitute a further source of hazard for the fire-fighter. ‣ Cylinders without pressure-relief valves have no provision for controlled release and are therefore more likely to explode if exposed to fire. --- FIRE FIGHTING REQUIREMENTS: --- The need for proximity, entry and special protective clothing should be determined for each incident, by a competent fire-fighting safety professional.
Fire/Explosion Hazard	‣ Containers may explode when heated - Ruptured cylinders may rocket ‣ Fire exposed containers may vent contents through pressure relief devices. ‣ High concentrations of gas may cause asphyxiation without warning. ‣ May decompose explosively when heated or involved in fire. ‣ Contact with gas may cause burns, severe injury and/ or frostbite. Decomposition may produce toxic fumes of: carbon monoxide (CO) Combustion products include: carbon dioxide (CO_2) hydrogen chloride phosgene phosphorus oxides (POx) hydrogen fluoride other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions. ‣ Vented gas is more dense than air and may collect in pits, basements.
HAZCHEM	2ZE

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	‣ Avoid breathing vapour and any contact with liquid or gas. Protective equipment including respirator should be used. ‣ **DO NOT** enter confined spaces where gas may have accumulated. ‣ Increase ventilation. ‣ Clear area of personnel. ‣ Stop leak only if safe to so do. ‣ Remove leaking cylinders to safe place. Release pressure under safe controlled conditions by opening valve. ‣ Do not exert excessive pressure on the valve; do not attempt to operate a damaged valve ‣ Orientate cylinder so that the leak is gas, not liquid, to minimise rate of leakage ‣ Keep area clear of personnel until gas has dispersed.
Major Spills	‣ Clear area of all unprotected personnel and move upwind. ‣ Alert Emergency Authority and advise them of the location and nature of hazard. ‣ Wear breathing apparatus and protective gloves. ‣ Prevent by any means available, spillage from entering drains and water-courses. ‣ Consider evacuation. ‣ Increase ventilation. ‣ No smoking or naked lights within area. ‣ Stop leak only if safe to so do. ‣ Water spray or fog may be used to disperse vapour.

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	▸ **DO NOT** enter confined space where gas may have collected. ▸ Keep area clear until gas has dispersed. ▸ Remove leaking cylinders to a safe place. ▸ Fit vent pipes. Release pressure under safe, controlled conditions ▸ Burn issuing gas at vent pipes. ▸ DO NOT exert excessive pressure on valve; DO NOTattempt to operate damaged valve.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	· Consider use in closed pressurised systems, fitted with temperature, pressure and safety relief valves which are vented for safe dispersal. Use only properly specified equipment which is suitable for this product, its supply pressure and temperature · The tubing network design connecting gas cylinders to the delivery system should include appropriate pressure indicators and vacuum or suction lines. · Fully-welded types of pressure gauges, where the bourdon tube sensing element is welded to the gauge body, are recommended. · Before connecting gas cylinders, ensure manifold is mechanically secure and does not containing another gas. Before disconnecting gas cylinder, isolate supply line segment proximal to cylinder, remove trapped gas in supply line with aid of vacuum pump · When connecting or replacing cylinders take care to avoid airborne particulates violently ejected when system pressurises. · Consider the use of doubly-contained piping; diaphragm or bellows sealed, soft seat valves; backflow prevention devices; flash arrestors; and flow monitoring or limiting devices. Gas cabinets, with appropriate exhaust treatment, are recommended, as is automatic monitoring of the secondary enclosures and work areas for release. · Use a pressure reducing regulator when connecting cylinder to lower pressure (<100 psig) piping or systems · Use a check valve or trap in the discharge line to prevent hazardous back-flow into the cylinder · Check regularly for spills or leaks. Keep valves tightly closed but do not apply extra leverage to hand wheels or cylinder keys. · Open valve slowly. If valve is resistant to opening then contact your supervisor · Valve protection caps must remain in place must remain in place unless container is secured with valve outlet piped to use point. · Never insert a pointed object (e.g hooks) into cylinder cap openings as a means to open cap or move cylinder. Such action can inadvertently turn the valve and gas a gas leak. Use an adjustable strap instead of wrench to free an over-tight or rusted cap. · A bubble of gas may buildup behind the outlet dust cap during transportation, after prolonged storage, due to defective cylinder valve or if a dust cap is inserted without adequate evacuation of gas from the line. When loosening dust cap, preferably stand cylinder in a suitable enclosure and take cap off slowly. Never face the dust cap directly when removing it; point cap away from any personnel or any object that may pose a hazard. under negative pressure (relative to atmospheric gas) · Suck back of water into the container must be prevented. Do not allow backfeed into the container. · Do NOT drag, slide or roll cylinders - use a suitable hand truck for cylinder movement · Test for leakage with brush and detergent - **NEVER** use a naked flame. · Do NOT heat cylinder by any means to increase the discharge rate of product from cylinder. · Leaking gland nuts may be tightened if necessary. · If a cylinder valve will not close completely, remove the cylinder to a well ventilated location (e.g. outside) and, when empty, tag as FAULTY and return to supplier. · Obtain a work permit before attempting any repairs. · DO NOT attempt repair work on lines, vessels under pressure. · Atmospheres must be tested and O.K. before work resumes after leakage. ▸ **DO NOT** transfer gas from one cylinder to another.
Other information	▸ Cylinders should be stored in a purpose-built compound with good ventilation, preferably in the open. ▸ Such compounds should be sited and built in accordance with statutory requirements. ▸ The storage compound should be kept clear and access restricted to authorised personnel only. ▸ Cylinders stored in the open should be protected against rust and extremes of weather. ▸ Cylinders in storage should be properly secured to prevent toppling or rolling. ▸ Cylinder valves should be closed when not in use. ▸ Where cylinders are fitted with valve protection this should be in place and properly secured. ▸ Gas cylinders should be segregated according to the requirements of the Dangerous Goods Act. ▸ Preferably store full and empty cylinders separately. ▸ Check storage areas for hazardous concentrations of gases prior to entry. ▸ Full cylinders should be arranged so that the oldest stock is used first. ▸ Cylinders in storage should be checked periodically for general condition and leakage. ▸ Protect cylinders against physical damage. Move and store cylinders correctly as instructed for their manual handling. **NOTE:** A 'G' size cylinder is usually too heavy for an inexperienced operator to raise or lower.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Cylinder: ▸ Ensure the use of equipment rated for cylinder pressure. ▸ Ensure the use of compatible materials of construction. ▸ Valve protection cap to be in place until cylinder is secured, connected. ▸ Cylinder must be properly secured either in use or in storage. ▸ Cylinder valve must be closed when not in use or when empty. ▸ Segregate full from empty cylinders. **WARNING:** Suckback into cylinder may result in rupture. Use back-flow preventive device in piping.
Storage incompatibility	▸ A number of phosphate and thiophosphate esters are of limited thermal stability and undergo highly exothermic self-accelerating decomposition reactions which may be catalysed by impurities. ▸ The potential hazards can be reduced by appropriate thermal control measures. BRETHERICK L.: Handbook of Reactive Chemical Hazards Thermal decomposition of organophosphate esters, in the presence of trimethylolpropane or its homologues (common components of synthetic lubricants), may produce bicyclic phosphates and phosphites. These may occur be produced in as little as 5 minutes at 650 deg C. These bicyclic compounds are a class of materials with neurotoxic properties which produce convulsive seizures in test animals. The formation of these compounds does not occur, for example, in the presence of a pentaerythritol base (another common component of synthetic lubricants). ▸ Avoid magnesium, aluminium and their alloys, brass and steel. ▸ Avoid reaction with oxidising agents

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	▸ Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances

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	1,1,1,2-tetrafluoroethane	1,1,1,2-Tetrafluoroethane	1000 ppm / 4240 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
1,1,1,2-tetrafluoroethane	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
tris(2-chloroisopropyl)phosphate	Not Available	Not Available
ethylenediamine, propoxylated	Not Available	Not Available
Non-hazardous ingredients	Not Available	Not Available
1,1,1,2-tetrafluoroethane	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
tris(2-chloroisopropyl)phosphate	E	≤ 0.1 ppm
ethylenediamine, propoxylated	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

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.

May act as a simple asphyxiants; these are gases which, when present in high concentrations, reduce the oxygen content in air below that required to support breathing, consciousness and life; loss of consciousness, with death by suffocation may rapidly occur in an oxygen deficient atmosphere.
CARE: Most simple asphyxiants are odourless or possess low odour and there is no warning on entry into an oxygen deficient atmosphere. If there is any doubt, oxygen content can be checked simply and quickly. It may not be appropriate to only recommend an exposure standard for simple asphyxiants rather it is essential that sufficient oxygen be maintained. Air normally has 21 percent oxygen by volume, with 18 percent regarded as minimum under normal atmospheric pressure to maintain consciousness / life. At pressures significantly higher or lower than normal atmospheric pressure, expert guidance should be sought.

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.

- Areas where cylinders are stored require good ventilation and, if enclosed, need discrete/controlled exhaust ventilation.
- Secondary containment and exhaust gas treatment may be required by certain jurisdictions.
- Local exhaust ventilation may be required in work areas.
- Consideration should be given to the use of diaphragm or bellows-sealed, soft-seat valves; backflow prevention devices and flow-monitoring or limiting devices.
- Automated alerting systems with automatic shutdown of gas-flow may be appropriate and may in fact be mandatory in certain jurisdictions.
- Respiratory protection in the form of air-supplied or self-contained breathing equipment must be worn if the oxygen concentration in the workplace air is less than 19%.
- Cartridge respirators do NOT give protection and may result in rapid suffocation.

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:
gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

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	Within each range the appropriate value depends on: Lower end of the range: 1: Room air currents minimal or favourable to capture; 2: Contaminants of low toxicity or of nuisance value only.; 3: Intermittent, low production.; 4: Large hood or large air mass in motion Upper end of the range: 1: Disturbing room air currents; 2: Contaminants of high toxicity; 3: High production, heavy use; 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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 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.
Individual protection measures, such as personal protective equipment	
Eye and face protection	‣ Chemical goggles. ‣ Full face shield may be required for supplementary but never for primary protection of eyes. ‣ 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	**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. ‣ When handling sealed and suitably insulated cylinders wear cloth or leather gloves. ‣ Insulated gloves: ‣ NOTE: Insulated gloves should be loose fitting so that may be removed quickly if liquid is spilled upon them. Insulated gloves are not made to permit hands to be placed in the liquid; they provide only short-term protection from accidental contact with the liquid.
Body protection	See Other protection below
Other protection	‣ Protective overalls, closely fitted at neck and wrist. ‣ Eye-wash unit. ‣ Ensure availability of lifeline in confined spaces. ‣ Staff should be trained in all aspects of rescue work. ‣ Rescue gear: Two sets of SCBA breathing apparatus Rescue Harness, lines etc.

Respiratory protection

Type KAX-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 5 x ES	Air-line*	KAX-2 P2	KAX-PAPR-2 P2 ^
up to 10 x ES	-	KAX-3 P2	-
10+ x ES	-	Air-line**	-

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

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used
- Positive pressure, full face, air-supplied breathing apparatus should be used for work in enclosed spaces if a leak is suspected or the primary containment is to be opened (e.g. for a cylinder change)
- Air-supplied breathing apparatus is required where release of gas from primary containment is either suspected or demonstrated.

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

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	AX-AUS / Class 1	-
up to 50	1000	-	AX-AUS / Class 1
up to 50	5000	Airline *	-

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up to 100	5000	-	AX-2
up to 100	10000	-	AX-3
100+		-	Airline**

** - Continuous-flow or positive pressure demand.
A(All classes) = Organic vapours, B AUS or B1 = Acid gases, 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 deg C)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Moisture sensitive.

Physical state	Liquified Gas	**Relative density (Water = 1)**	1.2
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	>743
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	-101	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	-26.2	**Molecular weight (g/mol)**	102
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)**	10
Vapour pressure (kPa)	560.5	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	3.5	**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

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Exposure to high concentrations of fluorocarbons may produce cardiac arrhythmias or cardiac arrest due sensitisation of the heart to adrenalin or noradrenalin. Deaths associated with exposures to fluorocarbons (specifically halogenated aliphatics) have occurred in occupational settings and in inhalation of bronchodilator drugs. Bronchospasm consistently occurs in human subjects inhaling fluorocarbons. At a measured concentration of 1700 ppm of one of the commercially available aerosols there is a biphasic change in ventilatory capacity, the first reduction occurring within a few minutes and the second delayed up to 30 minutes. Most subjects developed bradycardia (reduced pulse rate). Bradycardia is encountered in dogs when administration is limited to upper respiratory tract (oropharyngeal and nasal areas). Cardiac arrhythmias can be experimentally induced in animals (species dependency is pronounced with dogs and monkeys requiring lesser amounts of

Continued...

	fluorocarbon FC-11 than rats or mice). Sensitivity is increased by injection of adrenalin or cardiac ischaemia/necrosis or pulmonary thrombosis/bronchitis. The cardiotoxic effects of the fluorocarbons originate from irritation of the respiratory tract which in turn reflexively influences the heart rate (even prior to absorption of the fluorocarbon) followed by direct depression of the heart after absorption. Exposure to fluorocarbon thermal decomposition products may produce flu-like symptoms including chills, fever, weakness, muscular aches, headache, chest discomfort, sore throat and dry cough. Complete recovery usually occurs within 24 hours of exposure. Inhalation hazard is increased at higher temperatures. Chlorinated phosphate esters are distinguished from their non-halogenated congeners by possessing anaesthetic-like and muscle-relaxant properties. Even at high doses, however, they do not appear to produce pathological side-effects. Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. Symptoms of asphyxia (suffocation) may include headache, dizziness, shortness of breath, muscular weakness, drowsiness and ringing in the ears. If the asphyxia is allowed to progress, there may be nausea and vomiting, further physical weakness and unconsciousness and, finally, convulsions, coma and death. Significant concentrations of the non-toxic gas reduce the oxygen level in the air. As the amount of oxygen is reduced from 21 to 14 volume %, the pulse rate accelerates and the rate and volume of breathing increase. The ability to maintain attention and think clearly is diminished and muscular coordination is somewhat disturbed. As oxygen decreases from 14-10% judgement becomes faulty; severe injuries may cause no pain. Muscular exertion leads to rapid fatigue. Further reduction to 6% may produce nausea and vomiting and the ability to move may be lost. Permanent brain damage may result even after resuscitation at exposures to this lower oxygen level. Below 6% breathing is in gasps and convulsions may occur. Inhalation of a mixture containing no oxygen may result in unconsciousness from the first breath and death will follow in a few minutes. The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments
Skin Contact	Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. In common with other halogenated aliphatics, fluorocarbons may cause dermal problems due to a tendency to remove natural oils from the skin causing irritation and the development of dry, sensitive skin. They do not appear to be appreciably absorbed. 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. Vapourising liquid causes rapid cooling and contact may cause cold burns, frostbite, even through normal gloves. Frozen skin tissues are painless and appear waxy and yellow. Signs and symptoms of frost-bite may include 'pins and needles', paleness followed by numbness, a hardening an stiffening of the skin, a progression of colour changes in the affected area, (first white, then mottled and blue and eventually black; on recovery, red, hot, painful and blistered).
Eye	Although the material is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn). Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..
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. 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 degree of risk and level of surveillance. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Principal route of occupational exposure to the gas is by inhalation. It is generally accepted that the fluorocarbons are less toxic than the corresponding halogenated aliphatic based on chlorine. Repeated inhalation exposure to the fluorocarbon FC-11 does not produce pathologic lesions of the liver and other visceral organs in experimental animals. There has been conjecture in non-scientific publications that fluorocarbons may cause leukemia, cancer, sterility and birth defects; these have not been verified by current research. The high incidence of cancer, spontaneous abortion and congenital anomalies amongst hospital personnel, repeatedly exposed to fluorine-containing general anaesthetics, has caused some scientists to call for a lowering of the fluorocarbon exposure standard to 5 ppm since some are mutagens.

Tensorgrip H21 2k Spray Foam Insulation Part B	**TOXICITY**	**IRRITATION**

Continued...

	Not Available	Not Available

tris(2-chloroisopropyl)phosphate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye (rabbit): non-irritating*
Inhalation (Rat) LC50: >4.6 mg/l4h[2]	Skin (rabbit): mild (24 h): *[Akzo Nobel]
Oral (Rat) LD50: ~632 mg/kg[1]	

ethylenediamine, propoxylated

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

Non-hazardous ingredients

TOXICITY	IRRITATION
Not Available	Not Available

1,1,1,2-tetrafluoroethane

TOXICITY	IRRITATION
Inhalation (Rat) LC50: 359453.102 ppm4h[2]	Not Available

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*

TRIS(2-CHLOROISOPROPYL)PHOSPHATE

For non-polymeric chlorinated trisphosphates (typically (tris(chloroethyl)phosphate (TCEP), tris(chloropropyl)phosphate (TCPP) and tris(dichoropropyl)phosphate (TDCPP)

Chlorinated trisphosphates do not necessarily have similar chemical, physical, toxicological or environmental properties.
Blooming has been identified as a source of potential exposure (human and environmental) to trisphosphate plasticers/ flame retardants.
Blooming is defined as the migration (or more appropriately, diffusion) of an ingredient in rubber or plastic to the outer surface after curing.
Thus is generally a slow process. Increased temperature may accelerate the rate of migration. For example trisphosphates are know to bloom from car interior plastics, TVs and computer VDUs
Acute toxicity:
In rats, oral doses of TCEP are absorbed and distributed around the body to various organs, particularly the liver and kidney, but also the brain. Metabolites in rats and mice include bis(2-chloroethyl) carboxymethyl phosphate; bis(2-chloroethyl) hydrogen phosphate; and bis(2-chloroethyl)-2-hydroxyethyl phosphate glucuronide. Excretion is rapid, nearly complete and mainly via the urine. TCEP is of low to moderate acute oral toxicity (oral LD50 in the rat = 1150 mg/kg body weight). In repeat dose studies, TCEP caused adverse effects on the brain (hippocampal lesions in rats), liver and kidneys. The NOEL was 22 mg/kg body weight per day and the LOEL 44 mg/kg body weight per day for increased weights of liver and kidneys in rats
TCPP is of low to moderate acute toxicity by the oral (LD50 in rats = 1017-4200 mg/kg body weight), dermal (LD50 in rats and rabbits is > 5000 mg/kg body weight) and inhalation routes (LC50 in rats is > 4.6 mg/litre).
TDCPP is of low to moderate acute toxicity by the oral route (LD50 in rats = 2830 mg/kg body weight) and of low acute toxicity by the dermal route (dermal LD50 in rats is > 2000 mg/kg body weight). In a 3-month study in mice, an exposure of approximately 1800 mg/kg body weight per day caused death within one month. The no-observed-effect level (NOEL) for the study was 15.3 mg/kg body weight per day; the lowest-observed level (LOEL) for increased liver weight was 62 mg/kg body weight per day.
Irritation studies: TCEP is non-irritant to skin and eyes, but has not been tested for sensitization potential.
Rabbit eye and skin irritancy studies have indicated that TCPP is either non-irritant or mildly irritant.
Sensitisation studies: A skin sensitization study showed that TCPP has no sensitizing properties. The sensitization potential of TDCPP has not been investigated
Neurotoxicity: A very high oral dose of TCEP caused some inhibition of plasma cholinesterase and brain neuropathy target esterase in hens, but did not cause delayed neurotoxicity. In rats, a high dose of TCEP caused convulsions, brain lesions and impaired performance in a water maze.
Developmental toxicity: TCEP is not teratogenic
A TDCPP teratology study on rats showed foetotoxicity at an oral dose of 400 mg/kg body weight per day; there was maternal toxicity at doses of 100 and 400 mg/kg body weight per day. No teratogenicity was seen
Reproductive toxicity: TCEP adversely affects the fertility of male rats and mice. Effects on the reproductive system (i.e. effects on testes) were noted in a reproduction study in mice.
The potential for TDCPP to affect human male reproductive ability is unclear in view of testicular toxicity in rats but a lack of effect on male reproductive performance in rabbits. The possible effect on female reproduction has not been investigated.
In a 2-year carcinogenicity study in rats, using tris(dichloroisopropyl)phosphate (TDCiPP), effects were observed on the reproductive system of male rats (i.e. effects on testes). The effects were not confirmed in a fertility study in male rabbits. However, the nature of the reproductive toxicity of TDCiPP has not been sufficiently investigated in a well-designed study.
Histological abnormalities were identified in the testes and seminal vesicles in male rats. A LOAEL of 5 mg/kg is derived from this study. An LOAEL of 5 mg/kg has been proposed
Mutagenicity: No conclusions can be drawn about the mutagenicity of TCEP as *in vitro* test results were inconsistent and an *in vivo* bone marrow micronucleus test gave equivocal results.
The results of *in vitro* and *in vivo* mutagenicity studies investigating an appropriate range of end-points indicate that TCPP is not genotoxic.
TCPP has been investigated for potential delayed neurotoxicity in hens. There was no evidence of delayed neurotoxicity when two oral doses (each of 13 230 mg/kg body weight) were given 3 weeks apart.
Overall, the mutagenicity data show that TDCPP is not genotoxic *in vivo.*
Carcinogenicity: TCEP causes benign and malignant tumours at various organ sites in rats and mice.
The carcinogenicity of TDCPP has been investigated in a single 2-year feeding study. It was carcinogenic (increased occurrence of liver carcinomas) at all exposure levels that were tested (5-80 mg/kg body weight per day) in both male and female rats. Kidney, testicular and brain tumours were also found. In addition, there were non-neoplastic adverse effects in bone marrow, spleen, testis, liver and kidney. The effects in the kidney and testis occurred at all exposure levels. Only animals in the highest dose and control groups were evaluated for effects in the bone marrow and spleen. It was impossible, therefore, to determine whether there was a dose-response relationship for these effects in these organs.
TDCiPP produces liver tumours in rats.
Immunotoxicity: TDCPP exposure produced some indications of immunotoxicity in mice but only at high doses. Limited human studies following occupational exposure are available but they add little to the knowledge of the safety aspects of TDCPP.

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For tris(2-chloro-1-methylethyl)phosphate (TCPP)
The flame retardant product supplied in the EU, marketed as TCPP, is actually a reaction mixture containing four isomers. The individual isomers in this reaction mixture are not separated or marketed. The individual components are never produced as such. These data are true for TCPP produced by all EU manufacturers. The other isomers in the mixture include bis(1-chloro-2-propyl)-2-chloropropyl phosphate (CAS 76025-08-6); bis(2-chloropropyl)-1-chloro-2-propyl phosphate (CAS 76649-15-5) and tris(2-chloropropyl) phosphate (CAS 6145-73-9). The assumption is made that all isomers have identical properties in respect of risk assessment. The assumption is justified in part by the fact that they exhibit very similar chromatographic properties, even under conditions optimised to separate them. Predicted physicochemical properties differ to only a small extent.
Chlorinated alkyl phosphate esters (particularly TCPP) were identified as possible substitutes for the fire retardant pentabromodiphenyl ether They appear to be relatively persistent substances, and there is some human health concern. Three substances in this group have been characterised to a degree and serve as a read across reference for TCPP. They include tris(2-chloroethyl)phosphate (TCEP, CAS 115-96-8), tris[2-(chloro-1-chloromethyl)ethyl]phosphate (TDCP, CAS 13674-87-8) and 2,2-bis(chloromethyl)trimethylene bis[bis(2-chloroethyl)phosphate] (V6, CAS 38051-10-4). Other flame retardants in this family, which do not appear as EU HPV (High Production Volume) substances, include tetrakis[2-(chloroethyl)ethylene)diphosphate (CAS 33125-86-9), tris (2,3-dichloro-1-propyl)phosphate (CAS 78-43-3, an isomer of TDCP))
Acute toxicity: The inhalation exposure studies in animals were somewhat equivocal and in general lacking in detailed information. One study yielded an LC50 of > 7 mg/L/4 hr. A limit test yielded an acute LC50 value of >4.6 mg/L/4h. No deaths occurred at this concentration. Toxic signs observed in this study, and in 2 further poorly reported studies, included mild lethargy, matted fur, acute bodyweight depression and convulsions. From the studies, it appears that TCPP is more toxic when administered whole body as aerosol than by nose-only exposure. This suggests that some of the systemic toxicity observed when TCPP is administered whole body may result from dermal or oral uptake, rather than inhalation. Therefore, it is concluded that TCPP is of low toxicity via the inhalation route.
Studies in rats indicated that TCPP is of moderate toxicity via the oral route of exposure, with LD50 values from the better quality studies ranging from 632 mg/kg up to 4200 mg/kg, with the majority of values determined to be <2000 mg/kg. Common clinical and macroscopic signs of toxicity observed on nearly all studies included depression, ataxia, hunched posture, lethargy, laboured respiration, increased salivation, partially closed eyelids, body tremors, pilo-erection, ptosis, haemorrhagic lungs and dark liver and/or kidneys. A NOAEL of 200 mg/kg can be identified for acute oral toxicity. This is taken from a 1996 study, in which no clinical signs of toxicity were observed in animals dosed with 200 mg/kg TCPP. Based on the results of the acute oral studies, TCPP should be classified with R22, harmful if swallowed.
In a delayed neurotoxicity study conducted in hens, TCPP showed moderate toxicity. The principle effects were reduced mean body weight and food consumption, feather loss and cessation of laying. There was no evidence of inhibited plasma acetylcholinesterase or brain neurotoxic esterase enzyme levels. Therefore, there is no concern for acute delayed neurotoxicity for TCPP.
Studies in rats and rabbits indicated that TCPP is of low toxicity via the dermal route of exposure with LD50 values of >2000mg/kg.
There is an extensive database in animals, indicating that TCPP is non-irritant in the rabbit eye and skin. The lack of any substantial skin or eye irritation and the lack of irritation observed in the acute inhalation studies suggest that TCPP would be unlikely to produce significant respiratory tract irritation.
Evidence from a guinea pig study as well as from a local lymph node assay, indicates that TCPP does not possess significant skin sensitisation potential. No information is available on the respiratory sensitisation potential of TCPP.
Repeat dose toxicity: A study is available in which male and female rats were fed diets containing TCPP for 13 weeks at concentrations corresponding to mean substance intake values of up to 1349 mg/kg/day and 1745 mg/kg/day for males and females respectively. This study indicated the liver and thyroid to be the main target organs affected by TCPP. Effects observed included statistically significant increases in absolute and relative liver weights in males at all doses and females at the two highest doses, periportal hepatocyte swelling in high dose groups and mild thyroid follicular cell hyperplasia in males at all doses and females at the highest dose.Based on the increase in both absolute and relative liver weights, accompanied by mild thyroid follicular cell hyperplasia observed in males of all dose groups, a LOAEL of 52 mg/kg/day is derived and taken forward to risk characterisation. This LOAEL is taken forward in preference to the NOAEL which was identified in a 4-week study in which rats were dosed with TCPP at concentrations of 0, 10, 100 and 1000 mg/kg/day, as it was derived from a study of longer duration. The 4-week study also showed the liver as the target organ, with increased liver weight changes observed in the high dose groups, accompanied by hepatocyte hypertrophy in all high-dose males and one mid-dose male and changes in ALAT activity in high-dose animals.
A two-week study in which rats were fed diets of TCPP at concentrations corresponding to mean substance intake values of up to 1636 mg/kg/day for males and 1517 mg/kg/day for females showed no major clinical signs of toxicity. There was a significant reduction in weight gain and food consumption in high dose males during week 2, but there were no other significant findings.
In a 2-generation reproductive toxicity study in which rats were fed TCPP in the diet over two successive generations, the low-dose of 99 mg/kg for females is considered to be the LOAEL for parental toxicity. This is based on decreased body weight and food consumption seen in mid and high dose parental animals and the effects on uterus weight seen in all dosed animals. For males, a NOAEL of approximately 85 mg/kg is derived for parental toxicity, based on decreased body weights, food consumption and organ weight changes observed at mid and high dose groups.
No data are available on inhalation and dermal repeated dose toxicity.
Genotoxicity: The mutagenic potential of TCPP has been well investigated *in vitro*. Evidence from several bacterial mutagenicity studies shows that TCPP is not a bacterial cell mutagen. TCPP was also shown to be non-mutagenic in fungi. In mammalian cell studies, TCPP did not induce forward mutations at the TK locus in L5178Y mouse lymphoma cells in one study, but in a second study, the result was considered equivocal (in the presence of rat liver S9 fraction). A confirmatory mouse lymphoma was conducted in accordance with the relevant regulatory guidelines. The results of the assay indicate that TCPP shows clastogenic activity *in vitro* in the presence of metabolic activation.
The main concern for TCPP is clastogenicity, owing to the clearly positive *in vitro* mouse lymphoma study. *In vivo*, TCPP was not clastogenic in a mouse bone marrow micronucleus test. TCPP did not induce an increase in chromosomal aberrations in a rat bone marrow cytogenetics assay. In order to further investigate the potential for TCPP to induce DNA damage, an *in vivo* Comet assay in the rat liver was conducted. The liver was chosen for comet analysis as TCPP caused an increased mutation frequency in the mouse lymphoma assay in the presence of S9 and also induced liver enlargement in repeat dose studies. Under the conditions of this study, TCPP did not induce DNA damage in the liver of rats treated with either 750 or 1500 mg/kg TCPP.
Overall, it is considered that TCPP is not genotoxic *in vivo*.
Carcinogenicity: TCPP is structurally similar to two other chlorinated alkyl phosphate esters, TDCP (tris [2-chloro-1-(chloromethyl)ethyl] phosphate) and TCEP (tris (2-chloroethyl) phosphate). TDCP and TCEP are non-genotoxic carcinogens, in vivo, and have agreed classifications of Carc Cat 3 R40. Based on the available repeat dose toxicity data for TCPP, supported by a qualitative read-across from TDCP and TCEP, there is a potential concern for carcinogenicity for TCPP by a nongenotoxic mechanism. No quantitative read-across can be performed since there are no insights into an underlying mode of action for TCEP and TDCP which would make a prediction on a relatively potency of TCPP possible. Therefore, as a reasonable worst case approach, a risk characterisation will be carried out for this end-point.
It is proposed that the effects observed in the 90-day study for TCPP are taken as a starting point for risk characterisation. If these effects were to progress to cancer, they would do so by a non-genotoxic mechanism. Therefore, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP, should be used as a basis for risk characterisation of the carcinogenicity endpoint.
Reproductive toxicity: In a two-generation reproductive toxicity study with TCPP, there were no treatment related effects in pre-coital time, mating index, female fecundity index, male and female fertility index, duration of gestation and post-implantation loss. There was no effect on sperm parameters at necropsy. In females, the length of the longest oestrus cycle and the mean number of cycles per animal were statistically significantly increased in high dose animals of both generations. A decrease in uterus weight was observed in all dosed females in F0 and in high dose females in F1. Effects were also noted on pituitary weights, significant in high dose females of both generations. A LOAEL of 99 mg/kg is derived for effects on fertility. This is based on effects on the effect on uterus weight seen in all dosed females in F0 and high dose females in F1.
Developmental toxicity: From the same study, a LOAEL of 99 mg/kg is derived for developmental toxicity. This is based on a treatment related effect on the number of runts observed in all TCPP-treated groups of the F0 generation.

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	In a separate study, no treatment-related effects on foetal mortality, implantation number, resorption or foetal weight were observed following treatment of pregnant dams with TCPP. Cervical ribs and missing 13th ribs were noted at a low incidence in all treatment groups, but not in the control group. However, as a specific rib count undertaken in the 2-generation study did not reveal an increase in this effect, it is concluded that this is not toxicologically significant. Weaning rate and rearing condition were unaffected by treatment and there was no evidence of any abnormality for alkyl esters of phosphoric acid: The chemicals in this category exhibit a low to moderate order of acute toxicity. The rat oral LD50 values ranged from 500-1000 mg/kg with 2-ethylhexyl phosphate to >36,800 mg/kg for tris(2-ethylhexyl) phosphate. The dermal LD50 values ranged from 1200 to > 2000 mg/kg (rat) with bis(2-ethylhexyl) hydrogen phosphate to > 20,000 mg/kg (rabbit) with tris(2- ethylhexyl) phosphate. The inhalation LC50 values ranged from > 0.447 mg/l (4 hr. rat) with tris(2-ethylhexyl) phosphate to > 5.14 mg/l **(**4 hr. rat) with triisobutyl phosphate. **Metabolism:** Phosphoric acid esters are metabolized via dealkylation. Metabolism studies conducted on the tributyl phosphate indicate that dealkylation to form the alkyl alcohol is the primary route of metabolism Phosphoric acid tri-esters are rapidly metabolised to di-esters with mono-diesters also being produced. Studies of tributyl phosphate show that 40-64% of the parent compound is metabolised to dibutyl dihydrogen phosphate and that 1.1-2.1 % is metabolised to the monobutyl species. Therefore, tris(2-ethylhexyl) phosphate is expected to be metabolised to bis(2-ethylhexyl) phosphate (CAS RN: 298-07-7) and mono(2-ethylhexyl) phosphate (CAS RN 1070-03-7). Based on the evidence for dealkylation as the primary metabolic pathway, 2-ethylhexanol is the expected metabolite of tris(2-ethylhexyl) phosphate (CAS RN: 78-42-2) and 2-ethylhexyl phosphate (CAS RN: 12645-31-7). Triisobutyl phosphate is expected to be metabolised similarly as tributyl phosphate, with methoxypropanol as the alcohol metabolite Oral repeat dose NOAEL's in rats for dibutyl hydrogen phosphate, tributyl phosphate, ethylhexanol, 2- ethylhexanoic acid, bis(2-ethylhexyl) hydrogen phosphate, tris(2-ethylhexyl) phosphate, and triisobutyl phosphate were 30 mg/kg/day (44 days), 75 mg/kg/day (90 days), 125 mg/kg/day (90 days), 100 mg/kg/day (90 days), 250 mg/kg/day (5 days), and 1000 mg/kg/day (90 days), and 68.4-84.3 mg/kg (90 days), respectively. The weight of the evidence indicates that the members of this category are not genotoxic. Tris(2-ethylhexyl) phosphate, bis(2-ethylhexyl) hydrogen phosphate, 2-ethylhexyl phosphate, dibutyl hydrogen phosphate, tributyl phosphate, triisobutyl phosphate, 2-ethylhexanol, 2-ethylhexanoic acid, and phosphoric acid were negative in the Ames assay. Tris(2-ethylhexyl) phosphate, bis(2-ethylhexyl) phosphate, 2-ethylhexyl phosphate, and 2-ethylhexanol also were negative in the mouse lymphoma assay. Furthermore, tris(2-ethylhexyl) phosphate, dibutyl hydrogen phosphate, tributyl phosphate, and 2-ethylhexanol were negative in the chromosomal aberration assays (in vitro and/ or in vivo). Tris(2-ethylhexyl) phosphate was negative in a sister chromatid exchange assay while 2-ethylhexanoic acid was positive. Triisobutyl phosphate was negative in the in vivo mouse micronucleus assay. **Reproductive toxicity** was evaluated with a number of the members of this category. No effects on reproductive organs were observed in repeat dose studies with tris(2-ethylhexyl) phosphate, dibutyl hydrogen phosphate, tributyl phosphate, 2-ethylhexanol, or 2-ethylhexanoic acid. A two generation reproduction study with tributyl phosphate did not find any reproductive effects in rats at the highest dose tested (225 mg/kg/day). No significant effects on reproduction were seen in rats with an oral OECD 422 combined repeat dose toxicity and reproductive/developmental toxicity screen with dibutyl hydrogen phosphate (NOAEL = 1000 mg/kg). Reproductive effects were reported in rats at 300 mg/kg/day and 600 mg/kg/day in a one generation study with 2-ethylhexanoic acid. **Developmental toxicity**: The developmental toxicity of tributyl phosphate was evaluated in both rats and rabbits. Tributyl phosphate and triisobutyl phosphate were determined not to be teratogenic. 2-Ethylhexanol was found to cause developmental toxicity only at doses that were maternally toxic. Drinking water and gavage developmental toxicity studies have also been conducted with 2-ethylhexanoic acid in rats and rabbits. Developmental effects in rats at concentrations as low as 100 mg/kg administered in drinking water have been reported. Developmental studies with rats and rabbits concluded that 2-ethylhexanoic acid did not produce developmental effects in rats or rabbits under the conditions of these tests. The authors noted that the rat NOAEL was 100 mg/kg/day based on slight foetotoxicity at 250 mg/kg/day and that the rabbit NOAEL was 250 mg/kg/day (highest dose). The maternal NOAEL's for rats and rabbits were 250 mg/kg/day and 25 mg/kg/day, respectively.
ETHYLENEDIAMINE, PROPOXYLATED	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. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
1,1,1,2-TETRAFLUOROETHANE	* with added oxygen - ZhongHao New Chemical Materials MSDS Excessive concentration can have a narcotic effect; inhalation of high concentrations of decomposition products can cause lung oedema.
Tensorgrip H21 2k Spray Foam Insulation Part B & ETHYLENEDIAMINE, PROPOXYLATED	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.
Tensorgrip H21 2k Spray Foam Insulation Part B & 1,1,1,2-TETRAFLUOROETHANE	Disinfection by products (DBPs) re formed when disinfectants such as chlorine, chloramine, and ozone react with organic and inorganic matter in water. The observations that some DBPs such as trihalomethanes (THMs), di-/trichloroacetic acids, and 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) are carcinogenic in animal studies have raised public concern over the possible adverse health effects of DBPs. To date, several hundred DBPs have been identified. Numerous haloalkanes and haloalkenes have been tested for carcinogenic and mutagenic activities. n general, the genotoxic potential is dependent on the nature, number, and position of halogen(s) and the molecular size of the compound. Short-chain monohalogenated (excluding fluorine) alkanes and alkenes are potential direct-acting alkylating agents, particularly if the halogen is at the terminal end of the carbon chain or at an allylic position. Dihalogenated alkanes are also potential alkylating or cross-linking agents (either directly or after GSH conjugation),particularly if they are vicinally substituted (e.g., 1,2-dihaloalkane) or substituted at the two terminal ends of a short to medium-size (e.g., 2-7) alkyl moiety (i.e., alpha, omega-dihaloalkane). Fully halogenated haloalkanes tend to act by free radical or nongenotoxic mechanisms (such as generating peroxisome-proliferative intermediates) or undergo reductive dehalogenation to yield haloalkenes that in turn could be activated to epoxides. Haloalkenes are of concern because of potential to generate genotoxic intermediates after epoxidation. The concern for haloalkenes may be diminished if the double bond is internal or sterically hindered. The cancer concern levels of the 14 haloalkanes and haloalkenes, have been rated based on available screening cancer bioassay

Continued...

	(pulmonary adenoma assay) and genotoxicity data. Five brominated and iodinated methane and ethane derivatives are given a moderate rating. Beyond the fact that bromine and iodine are better leaving groups than chlorine, there is also evidence that brominated THMs may be preferentially activated by a theta-class glutathione S-transferase (GSTT1-1) to mutagens in Salmonella even at low substrate concentrations Furthermore, there are human carcinogenicity implications because of polymorphism in GSTT1-1. Human subpopulations with expressed GSTT1-1 may be at a greater risk to brominate THMs than humans who lack the gene. Six, two, and one haloalkanes/ haloalkene(s) are given low-moderate, marginal, and low concern, respectively.

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

Tensorgrip H21 2k Spray Foam Insulation Part B

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

tris(2-chloroisopropyl)phosphate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	4mg/l	1
BCF	1008h	Fish	0.8-2.8	7
ErC50	72h	Algae or other aquatic plants	4mg/l	1
EC50	48h	Crustacea	65335mg/l	1
EC50	72h	Algae or other aquatic plants	82mg/l	Not Available
EC50(ECx)	96h	Algae or other aquatic plants	4mg/l	1
LC50	96h	Fish	56.2mg/l	Not Available

ethylenediamine, propoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	150.67mg/l	2
LC50	96h	Fish	~4600mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	4.25mg/l	2

Non-hazardous ingredients

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

1,1,1,2-tetrafluoroethane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	980mg/l	Not Available
EC50	96h	Algae or other aquatic plants	142mg/l	2
EC50	72h	Algae or other aquatic plants	>114mg/l	2
NOEC(ECx)	96h	Fish	300mg/l	Not Available
LC50	96h	Fish	450mg/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*

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
For haloalkanes and haloalkenes:
Environmental fate:
Certain haloalkane gases in the atmosphere can also contribute to the greenhouse effect by restricting heat loss from the Earth's atmosphere through absorbing infrared emissions from the surface. Generally haloalkanes contributing to the greenhouse effect consist of a fully or partly fluorinated carbon backbone.
Gas-phase reactions with OH radicals are the major tropospheric loss process for the haloalkanes. In addition photooxidation reactions with O3 and NO3 radicals can result in transformation.
Organic substances containing chlorine, if primarily present in the atmospheric compartment and if their lifetime is long enough can reach the stratosphere and decompose through photolysis and other chemical reaction (e.g. with OH radical). Chlorine atoms can then participate in the catalytic ozone destruction cycles. The atmospheric lifetime is too short to enable a significant fraction of the compound emitted to reach the stratosphere
Haloalkanes do not hydrolyse easily. Acids do not catalyse the hydrolysis and base catalysis is only important at higher pHs than are observed in the environment.
The apparent hazard of halo- alkanes and alkenes to human health has prompted investigations concerning their fate in subsurface waters and in soil. Although abiotic transformations may be significant within the time scales commonly associated with groundwater movement, biotic process typically proceed much faster, provided that there are sufficient substrates, nutrients and microbial populations to mediate such transformations. Several bacterial strains including methane-utilising bacteria capable of utilising haloalkanes have been isolated. Microbial dehalogenation by these strains is mediated by enzymes (oxygenase and hydrolase). The biodegradation of haloalkanes can proceed through different

Continued...

pathways. Haloparaffins (C12 to C18) have been reported to be incorporated into fatty acids in bacteria, yeasts, and fungi , resulting in their accumulation in the food chain. Another pathway is the oxygenation at the nonhalogenated end of monohalogenated alkanes by an inherent oxygenase with a tight substrate selectivity In this case fluoroalkanes were defluorinated, but no dehalogenation was observed with chloro-, bromo-, or iodoalkanes. Chain length was reported to have minor effects on this oxygenation reaction. In general, alpha- and alpha,omega-chlorinated haloalkanes with short carbon chains (C1 to C6) are dehalogenated hydrolytically or by a glutathione-dependent mechanism. In contrast, alpha- and alpha,omega-haloalkanes with longer chains, e.g., 1,9-dichlorononane and 1,10-dichlorodecane (1,10-DCD), have been proposed to be dehalogenated by oxidative mechanisms. Studies on the biodegradation of this class of compounds are rare, because haloalkane-degrading microorganisms are not easily found
In water and terrestrial compartments haloalkanes may hydrolyse in the presence of naturally occurring sulfur-containing nucleophiles Bisulfide ion (HS-) is generally the most important nucleophile because it is moderately reactive and is usually present at the highest concentration. When elemental sulfur is present, polysulfides(S4 2- and S5 2-) will be more important than HS- at pH 7 (approximately) because they are 60 times more reactive and their equilibrium concentrations increase with increasing pH. The end products of such reactions include a variety of mercaptans and dialkyl sulfides.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
tris(2-chloroisopropyl)phosphate	HIGH	HIGH
1,1,1,2-tetrafluoroethane	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
tris(2-chloroisopropyl)phosphate	LOW (BCF = 4.6)
1,1,1,2-tetrafluoroethane	LOW (LogKOW = 1.68)

Mobility in soil

Ingredient	Mobility
tris(2-chloroisopropyl)phosphate	LOW (Log KOC = 1278)
1,1,1,2-tetrafluoroethane	LOW (Log KOC = 96.63)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	**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. Evaporate residue at an approved site. Return empty containers to supplier. If containers are marked non-returnable establish means of disposal with manufacturer prior to purchase. Ensure damaged or non-returnable cylinders are gas-free before disposal.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	2ZE

Land transport (ADG)

14.1. **UN number or ID number**	3500
14.2. **UN proper shipping name**	CHEMICAL UNDER PRESSURE, N.O.S.
14.3. **Transport hazard class(es)**	Class: 2.2 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: 274 362 Limited quantity: 0

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3500

Continued...

14.2. **UN proper shipping name**	Chemical under pressure, n.o.s. *
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 2.2 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 2L
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: A187 Cargo Only Packing Instructions: 218 Cargo Only Maximum Qty / Pack: 150 kg Passenger and Cargo Packing Instructions: 218 Passenger and Cargo Maximum Qty / Pack: 75 kg Passenger and Cargo Limited Quantity Packing Instructions: Forbidden Passenger and Cargo Limited Maximum Qty / Pack: Forbidden

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3500
14.2. **UN proper shipping name**	CHEMICAL UNDER PRESSURE, N.O.S.
14.3. **Transport hazard class(es)**	IMDG Class: 2.2 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	Not Applicable
14.5 **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	EMS Number: F-C , S-V Special provisions: 274 362 Limited Quantities: 0

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

Not Applicable

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

Product name	Group
tris(2-chloroisopropyl)phosphate	Not Available
ethylenediamine, propoxylated	Not Available
Non-hazardous ingredients	Not Available
1,1,1,2-tetrafluoroethane	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
tris(2-chloroisopropyl)phosphate	Not Available
ethylenediamine, propoxylated	Not Available
Non-hazardous ingredients	Not Available
1,1,1,2-tetrafluoroethane	Not Available

SECTION 15 Regulatory information

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

tris(2-chloroisopropyl)phosphate is found on the following regulatory lists

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

ethylenediamine, propoxylated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Non-hazardous ingredients is found on the following regulatory lists

Not Applicable

1,1,1,2-tetrafluoroethane is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

Continued...

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (tris(2-chloroisopropyl)phosphate; ethylenediamine, propoxylated; 1,1,1,2-tetrafluoroethane)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (ethylenediamine, propoxylated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (ethylenediamine, propoxylated)
Vietnam - NCI	Yes
Russia - FBEPH	No (ethylenediamine, propoxylated)
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	15/03/2024
Initial Date	12/10/2022

SDS Version Summary

Version	Date of Update	Sections Updated
2.3	14/03/2024	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Toxicological information - Chronic Health, Hazards identification - Classification, Exposure controls / personal protection - Engineering Control, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, Firefighting measures - Fire Fighter (extinguishing media), Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (swallowed), Composition / information on ingredients - Ingredients, Stability and reactivity - Instability Condition, Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Transport information - Transport

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

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

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory

Continued...

- 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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