

AP STAB N180 CMP A

Hychem International

Chemwatch: 7972-32

Version No: 2.1

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

Initial Date: 05/08/2025

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

Product Identifier

Product name	AP STAB N180 CMP A
Chemical Name	Not Applicable
Synonyms	Not Available
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	Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	Hychem International
Address	Unit 1, 30 Bluett Drive Smeaton Grange NSW 2567 Australia
Telephone	+61 2 4646 1660
Fax	+61 2 4647 3700
Website	www.hychem.com.au
Email	admin@hychem.com.au

Emergency telephone number


Association / Organisation	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	+61 1800 951 288 (ID#: 7972-32)
Other emergency telephone number(s)	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification ^[1]	Acute Toxicity (Oral) Category 4, 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)

H302	Harmful if swallowed.
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H351	Suspected of causing cancer.
H361d	Suspected of damaging the unborn child.

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P280	Wear protective gloves and protective clothing.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
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.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

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

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
13674-84-5	0-20	<u>tris(2-chloroisopropyl)phosphate</u>
107-21-1	0-9	<u>ethylene glycol</u>

Legend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

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

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prosthesis such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.

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▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

To treat poisoning by the higher aliphatic alcohols (up to C7):

- Gastric lavage with copious amounts of water.
- It may be beneficial to instill 60 ml of mineral oil into the stomach.
- Oxygen and artificial respiration as needed.
- Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5]

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 shock.
- Monitor and treat, where necessary, for pulmonary oedema.
- Anticipate and treat, where necessary, for seizures.
- **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

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.
- If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Acidosis may respond to hyperventilation and bicarbonate therapy.
- Haemodialysis might be considered in patients with severe intoxication.
- Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.

Symptomatic and supportive therapy is advised in managing patients.

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.

SECTION 5 Firefighting measures

Extinguishing media

- Alcohol stable 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

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Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ 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.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) hydrogen chloride phosgene phosphorus oxides (PO_x) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

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	<ul style="list-style-type: none"> ▶ Remove all ignition sources. ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	<p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ 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	<ul style="list-style-type: none"> ▶ 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. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling.
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	<ul style="list-style-type: none"> ▶ Work clothes should be laundered separately. ▶ 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. ▶ DO NOT allow clothing wet with material to stay in contact with skin
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ 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. <p>BREITHERICK 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).</p> <p>Alcohols</p> <ul style="list-style-type: none"> ▶ are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents. ▶ reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen ▶ react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium ▶ should not be heated above 49 deg. C. when in contact with aluminium equipment <p>Ethylene glycol:</p> <ul style="list-style-type: none"> ▶ reacts violently with oxidisers and oxidising acids, sulfuric acid, chlorosulfonic acid, chromyl chloride, perchloric acid ▶ forms explosive mixtures with sodium perchlorate ▶ is incompatible with strong acids, caustics, aliphatic amines, isocyanates, chlorosulfonic acid, oleum, potassium bichromate, phosphorus pentasulfide, sodium chlorite

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	ethylene glycol	Ethylene glycol (vapour)	20 ppm / 52 mg/m ³	104 mg/m ³ / 40 ppm	Not Available	Not Available
Australia Exposure Standards	ethylene glycol	Ethylene glycol (particulate)	10 mg/m ³	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
tris(2-chloroisopropyl)phosphate	Not Available	Not Available
ethylene glycol	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p>
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Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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.

Individual protection measures, such as personal protective equipment



Eye and face protection

- ▶ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.
- ▶ Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. [AS/NZS 1337.1, EN166 or national equivalent]
- ▶ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.
- ▶ Alternatively a gas mask may replace splash goggles and face shields.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

- ▶ Elbow length PVC gloves

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

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

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

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

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

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

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

	<ul style="list-style-type: none"> Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> Excellent when breakthrough time > 480 min Good when breakthrough time > 20 min Fair when breakthrough time < 20 min Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> Overalls. P.V.C apron. Barrier cream. Skin cleansing cream. Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the

computer-generated selection:

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Material	CPI
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A
NEOPRENE/NATURAL	A
NITRILE	A
NITRILE+PVC	A
PE/EVAL/PE	A
PVC	A
TEFLON	A
PVA	B

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Ansell Glove Selection

Glove — In order of recommendation
AlphaTec 02-100
AlphaTec® Solvex® 37-185
AlphaTec® 58-008
TouchNTuff® 83-500
AlphaTec® 15-554

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

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

- 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

MICROFLEX® 93-260
AlphaTec® 38-612
AlphaTec® 53-001
AlphaTec® 58-005
AlphaTec® 58-530B

The suggested gloves for use should be confirmed with the glove supplier.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Colourless liquid with characteristic odour; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	1.085
Odour	Characteristic	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Applicable	Viscosity (cSt)	461
Initial boiling point and boiling range (°C)	197	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	2500 BuAC = 1	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	28.319
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m³)	Not Available	Enclosed Space Ignition Deflagration Density (g/m³)	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	Based on available data, the classification criteria are not met.

d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severe damage to the health of the individual. Relatively small amounts absorbed through the lungs may prove fatal.</p> <p>Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.</p> <p>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.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Overexposure to non-ring alcohols causes nervous system symptoms. These include headache, muscle weakness and inco-ordination, giddiness, confusion, delirium and coma.</p> <p>For ethylene glycol:</p> <p>Symptoms following swallowing ethylene glycol include failure of breathing, central nervous system depression, cardiovascular collapse, lung swelling, acute kidney failure, and even brain damage. Swallowing 100 millilitres has caused death.</p> <p>There are three stages of ethylene glycol poisoning. The severity of each stage depends upon the amount of ethylene glycol swallowed. There is usually minimal damage to the liver. In the first 12 hours, central nervous system depression is seen. A temporary feeling of exhilaration occurs, without the odour of ethanol. There may be gastrointestinal complaints including nausea and vomiting. Acidosis, coma, convulsions and seizures may also occur. Disorders in eye movements may occur, although otherwise eye examination usually remains normal.</p> <p>At 12-24 hours after swallowing, effects on the lung and heart appear. These are characterized by fast heart rate, fast breathing, and mildly high blood pressure. Congestive heart failure and circulatory collapse may occur in severe poisonings. Effects on the kidney are seen 24-72 hours post-ingestion and are characterized by reduced urine output, flank pain, death of kidney tubules, kidney failure, and rarely, failure of the bone marrow. Kidney damage may be permanent.</p> <p>Acid base disturbances (acidosis) in the blood result from the formation of glycolic acid and some lactic acid.</p> <p>Animal testing showed that ethylene glycol, if swallowed during pregnancy, may lead to birth defects.</p>
Skin Contact	<p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Skin contact with the material may produce severe damage to the health of the individual; systemic effects may result following absorption and these may be fatal.</p> <p>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.</p> <p>Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</p>
Eye	Although the liquid 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).
Chronic	<p>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.</p> <p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.</p>

AP STAB N180 CMP A

There is some evidence to provide a presumption that human exposure to the material may result in impaired fertility on the basis of: some 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.

Human volunteers exposed to ethylene glycol, 20 to 22 hours/day at mean daily concentrations ranging from 1.4 to 27 ppm for about 4 weeks complained of throat irritation, mild headache and low backache. Complaints became marked when the concentration in the exposure chamber was raised above 56 mg/m³ for part of the day. The most common complaint was irritation of the upper respiratory tract. Concentrations above 80 ppm were intolerable with a burning sensation along the trachea and a burning cough. Excessively exposed workers have reported drowsiness.

AP STAB N180 CMP A	TOXICITY	IRRITATION
	Dermal (None) LD50: >2000 mg/kg ^[2]	Not Available
Oral (None) LD50: 1724.138 mg/kg ^[2]		
tris(2-chloroisopropyl)phosphate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >4.6 mg/l4h ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
Oral (Rat) LD50: >500 mg/kg ^[1]		
ethylene glycol	TOXICITY	IRRITATION
	dermal (mouse) LD50: >3500 mg/kg ^[1]	Eye (Rodent - rabbit): 0.012ppm/3D
	Oral (Rat) LD50: >2000 mg/kg ^[2]	Eye (Rodent - rabbit): 100mg/1H - Mild
		Eye (Rodent - rabbit): 1440mg/6H - Moderate
		Eye (Rodent - rabbit): 500mg/24H - Mild
		Eye (Rodent - rat): 0.012%/3D
		Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Rodent - rabbit): 555mg - Mild
	Skin: no adverse effect observed (not 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

TRIS(2-CHLOROISOPROPYL)PHOSPHATE	
	For non-polymeric chlorinated trisphosphates (typically tris(chloroethyl)phosphate (TCEP), tris(chloropropyl)phosphate (TCPP) and tris(dichloropropyl)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 plasticizers/ 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 known 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.

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. 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-di-esters 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.

ETHYLENE GLYCOL

[Estimated Lethal Dose (human) 100 ml; RTECS quoted by Orica] Substance is reproductive effector in rats (birth defects). Mutagenic to rat cells.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed throughout the gastrointestinal tract. Limited information suggests that it is also absorbed through the airways; absorption through skin is apparently slow. Following absorption, it is distributed throughout the body. In humans, it is initially metabolized by alcohol dehydrogenase to form glycoaldehyde, which is rapidly converted to glycolic acid and glyoxal. These breakdown products are oxidized to glyoxylate, which may be further metabolized to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate carbon dioxide, which is one of the major elimination products of ethylene glycol. In addition to exhaled carbon dioxide, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination is rapid and occurs within a few hours.

Respiratory effects: Respiratory system involvement occurs 12-24 hours after swallowing sufficient amounts of ethylene glycol. Symptoms include hyperventilation, shallow rapid breathing, and generalized swelling of the lungs with calcium oxalate deposits occasionally appearing in the lungs. Respiratory system involvement appears to be dose-dependent and occurs at the same time as cardiovascular changes. Later, there may be other changes compatible with adult respiratory distress syndrome (ARDS). Swelling of the lung can be a result of heart failure, ARDS, or aspiration of stomach contents. Symptoms related to acidosis such as fast or excessive breathing are frequently observed; however, major symptoms such as swelling of the lung and inflammation of the bronchi and lungs are relatively rare, and are usually seen only in extreme poisoning.

Cardiovascular effects: Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of ethylene glycol poisoning by swallowing, which is 12-24 hours after acute exposure. The symptoms of poisoning involving the heart include increased heart rate, heart enlargement and ventricular gallop. There may also be high or low blood pressure, which may progress to cardiogenic shock. In lethal cases, inflammation of the heart muscle has been observed at autopsy. Cardiovascular involvement appears to be rare and usually seen after swallowing higher doses of ethylene glycol. In summary, acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal effects: Common early acute effects of swallowing ethylene glycol include nausea, vomiting with or without blood, heartburn and abdominal cramping and pain. One patient showed intermittent diarrhea and pain, and after surgery, deposition of oxalate crystals was shown to have occurred.

Musculoskeletal effects: Reported musculoskeletal effects in cases of acute ethylene glycol poisoning include diffuse muscle tenderness and pain, associated with high levels of creatinine in the blood, and jerks and contractions associated with low calcium.

Liver effects: Autopsies carried out on people who died following acute ethylene glycol poisoning showed deposition of calcium oxalate in the liver as well as hydropic and fatty degeneration and cell death (necrosis) of the liver.

Kidney effects: Adverse kidney effects are seen during the third stage of ethylene glycol poisoning, 2-3 days after acute exposure. Calcium oxalate crystals are deposited in the tubules and are seen in the urine. There may also be degeneration and death of tubule cells, and inflammation of the tubule interstitium. If untreated, the degree of kidney damage progresses and leads to blood and protein in the urine, decreased kidney function, reduction in urine output and ultimately, kidney failure. With adequate supportive therapy, kidney function can return to normal or near normal.

Metabolic effects: Metabolic changes can occur within 12 hours of exposure to ethylene glycol. There may be metabolic acidosis, caused by accumulation of glycolic acid in the blood and therefore a reduction in blood pH. The anion gap is increased, due to increased unmeasured anions (mainly glycolate).

Effects on the nervous system: Adverse reactions involving the nervous system are among the first symptoms to appear in humans after ethylene glycol is swallowed. These early effects are also the only symptoms caused by unmetabolised ethylene glycol. Together with metabolic effects (see above), they occur from 0.5-12 hours after exposure and are considered to be part of the first stage in ethylene glycol poisoning. Inco-ordination, slurred speech, confusion and sleepiness are common in the early stages, as are irritation, restlessness and disorientation. Later, there may be effects on cranial nerves (which may be reversible over many months). Swelling of the brain (cerebrum) and crystal deposits of calcium oxalate in the walls of the small blood vessels of the brain were found at autopsy in people who died after acute ethylene glycol poisoning.

Reproductive effects: Animal testing showed that ethylene glycol may affect fertility, survival of fetuses and the male reproductive organs.

Effects on development: Animal studies indicate that birth defects may occur after exposure in pregnancy; there may also be reduction in foetal weight.

Cancer: No studies are known regarding cancer effects in humans or animal, after skin exposure to ethylene glycol.

Genetic toxicity: No human studies available, but animal testing results are consistently negative.

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

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

SECTION 12 Ecological information

Toxicity

AP STAB N180 CMP A	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
	BCF	1008h	Fish	0.8-2.8	7
	EC50	48h	Crustacea	65335mg/l	1
	EC50	72h	Algae or other aquatic plants	82mg/l	Not Available
	ErC50	72h	Algae or other aquatic plants	4mg/l	1
	EC50	96h	Algae or other aquatic plants	4mg/l	1
	EC50(ECx)	96h	Algae or other aquatic plants	4mg/l	1
	LC50	96h	Fish	56.2mg/l	Not Available
ethylene glycol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	EC50(ECx)	Not Available	Algae or other aquatic plants	6500-7500mg/l	1
	EC50	96h	Algae or other aquatic plants	6500-13000mg/l	1
	LC50	96h	Fish	8050mg/L	4

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
tris(2-chloroisopropyl)phosphate	HIGH	HIGH
ethylene glycol	LOW (Half-life = 24 days)	LOW (Half-life = 3.46 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
tris(2-chloroisopropyl)phosphate	LOW (BCF = 4.6)
ethylene glycol	LOW (BCF = 200)

Mobility in soil

Ingredient	Mobility
tris(2-chloroisopropyl)phosphate	LOW (Log KOC = 1278)
ethylene glycol	HIGH (Log KOC = 1)

SECTION 13 Disposal considerations**Waste treatment methods**

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▶ 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

14.7. Maritime transport in bulk according to IMO instruments**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
ethylene glycol	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
tris(2-chloroisopropyl)phosphate	Not Available
ethylene glycol	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

Continued...

Australian Inventory of Industrial Chemicals (AIIC)

ethylene glycol 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

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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; ethylene glycol)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
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. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	05/08/2025
Initial Date	05/08/2025

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
- ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
- ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
- ▶ IGC: International Gas Carrier Code
- ▶ IBC: International Bulk Chemical Code

- ▶ 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
- ▶ KECl: 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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