COLTACK EVOLUTION

750

ACCESSORY PRODUCTS

APPLICATIONS

ROOFS

TECHNICAL DATA SHEET

ANZ-TDS-01-COLTACK EVOLUTION 750

DESCRIPTION

COLTACK EVOLUTION 750 is a single-component, solvent free, moisture curing polyurethane-based adhesive foam to be used with an application gun.

COLTACK EVOLUTION 750 is used on flat roofs to bond ridged insulation panels with an appropriate finishing (no thermofusible film) to the substrate. It is also suitable for the mutual bonding of panels.

A fast expansion and adhesion of the panels will arise under the influence of air humidity.

The expansion properties will also ensure a good bonding on uneven substrates.

CHARACTERISTICS

	COLTACK EVOLUTION 750
Compostion	polyurethane resins
Open time (1)	±8 min (20 °C/60 % RH)
Curing time (1)	± 40 min (20 °C/60 % RH)
Resistant to loads (1)	±60 min (20 °C/60 % RH)
Consumption	±7 m²/aerosol can (synthetic foam) ±3,5 m²/aerosol can (mineral wool)
Application temperature	+5 / +35 °C
Application temperature product	+10 / +35 °C
Thermal conductivity (W/(m.K))	0,036
Dry Content	>99 %
Viscosity at 20 °C*	about 600 mPas
Dry to touch at 23 °C, 50% HR	about 15min
Walkable at 23 °C, 50% HR	about 2 hours

(1) Depending on conditions (temperature, relative humidity, ...)

SPE PACKAGING AND STORAGE

Canister of 10,4 kg

Aérosol can of 750 ml.

Minimum 18 months in original unopened packaging, stored upright in a dry and cool place at a temperature between +5 °C and +25 °C.

SPECIAL INDICATIONS

Hygiene, Health and Environment

For more information, please refer to the relevant safety data sheet.

Quality-, Environment- and Safety Management

SOPREMA has always attached the highest importance to the quality of the products, the environment and safety.

For this reason, we operate independently monitored Quality and Environment Assurance Systems in line with

EN ISO 9001 and EN ISO 14001.

INSTALLATION

The substrate must be clean, solid and free from dust and grease.

Vigorously shake the aerosol/can/canister for ± 30 seconds before use.

Close the adjusting screw of the application gun.

Screw the aerosol can on the gun (in case of the canister, close the valve of the hose and canister and connect hose. After connecting the hose fully open the valve of the canister and hose).

Adjust the control screw of the gun.

Apply, at a distance of about ±10 mm from the surface uniform beads of ±30 mm diameter.

Apply in case of synthetic insulation panels, in accordance with the wind load calculations, at least 4 parallel beads/m^2 (8 beads for mineral wool) in the length of the panels.

Place the insulation panels immediately after applying the adhesive beads.

Do not shift the panels because it will rupture the adhesive layer/bond.

Always consult the placement guidelines.

Cleaning tools:

- uncured product can be removed with COLTACK CLEANER
- cured material can only be mechanically removed

Coltack Evolution 750 (Aerosol)

Soprema Australia Pty Ltd

Chemwatch Hazard Alert Code: **4**

Chemwatch: **5494-59**
Version No: **2.1.20.12**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **30/09/2021**
Print Date: **30/09/2021**
L.GHS.AUS.EN

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

Product Identifier

Product name	Coltack Evolution 750 (Aerosol)
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	AEROSOLS
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.

Details of the supplier of the safety data sheet

Registered company name	**Soprema Australia Pty Ltd**
Address	L 35/100 Barangaroo Avenue, Sydney NSW 2000
Telephone	+61 3 9221 6230
Fax	Not Available
Website	www.soprema.com.au
Email	info@soprema.com.au

Emergency telephone number

Association / Organisation	**Soprema Australia Pty Ltd**
Emergency telephone numbers	042 595 2526 (Cruz Utanga: Mon-Fri 7.30am to 4pm)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Aerosols Category 1, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 2, Reproductive Toxicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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

Hazard statement(s)

AUH044	Risk of explosion if heated under confinement.
H222+H229	Extremely flammable aerosol. Pressurized container: may burst if heated.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.

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H351	Suspected of causing cancer.
H361d	Suspected of damaging the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P260	Do not breathe gas.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
9016-87-9	25-<75	polymeric diphenylmethane diisocyanate
13674-84-5	1-<25	tris(2-chloroisopropyl)phosphate
115-10-6	1-<10	dimethyl ether
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 aerosols come in contact with the eyes: ▸ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If solids or aerosol mists are deposited upon the skin: ▸ Flush skin and hair with running water (and soap if available). ▸ Remove any adhering solids with industrial skin cleansing cream. ▸ **DO NOT** use solvents. ▸ Seek medical attention in the event of irritation.
Inhalation	Following uptake by inhalation, move person to an area free from risk of further exposure. Oxygen or artificial respiration should be administered as needed. Asthmatic-type symptoms may develop and may be immediate or delayed up to several hours. Treatment is essentially symptomatic. A physician should be consulted. If aerosols, fumes or combustion products are inhaled:

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	▸ Remove to fresh air. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor.
Ingestion	▸ Not considered a normal route of entry.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
For sub-chronic and chronic exposures to isocyanates:
- This material may be a potent pulmonary sensitiser which causes bronchospasm even in patients without prior airway hyperreactivity.
- Clinical symptoms of exposure involve mucosal irritation of respiratory and gastrointestinal tracts.
- Conjunctival irritation, skin inflammation (erythema, pain vesiculation) and gastrointestinal disturbances occur soon after exposure.
- Pulmonary symptoms include cough, burning, substernal pain and dyspnoea.
- Some cross-sensitivity occurs between different isocyanates.
- Noncardiogenic pulmonary oedema and bronchospasm are the most serious consequences of exposure. Markedly symptomatic patients should receive oxygen, ventilatory support and an intravenous line.
- Treatment for asthma includes inhaled sympathomimetics (epinephrine [adrenalin], terbutaline) and steroids.
- Activated charcoal (1 g/kg) and a cathartic (sorbitol, magnesium citrate) may be useful for ingestion.
- Mydriatics, systemic analgesics and topical antibiotics (Sulamyd) may be used for corneal abrasions.
- There is no effective therapy for sensitised workers.

[Ellenhorn and Barceloux; Medical Toxicology]
NOTE: Isocyanates cause airway restriction in naive individuals with the degree of response dependant on the concentration and duration of exposure. They induce smooth muscle contraction which leads to bronchoconstrictive episodes. Acute changes in lung function, such as decreased FEV1, may not represent sensitivity.
[Karol & Jin, Frontiers in Molecular Toxicology, pp 56-61, 1992]
Personnel who work with isocyanates, isocyanate prepolymers or polyisocyanates should have a pre-placement medical examination and periodic examinations thereafter, including a pulmonary function test. Anyone with a medical history of chronic respiratory disease, asthmatic or bronchial attacks, indications of allergic responses, recurrent eczema or sensitisation conditions of the skin should not handle or work with isocyanates. Anyone who develops chronic respiratory distress when working with isocyanates should be removed from exposure and examined by a physician. Further exposure must be avoided if a sensitivity to isocyanates or polyisocyanates has developed.

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

- Small quantities of water in contact with hot liquid may react violently with generation of a large volume of rapidly expanding hot sticky semi-solid foam.
- Presents additional hazard when fire fighting in a confined space.
- Cooling with flooding quantities of water reduces this risk.
- Water spray or fog may cause frothing and should be used in large quantities.

SMALL FIRE:
- Water spray, dry chemical or CO2

LARGE FIRE:
- Water spray or fog.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ If safe, switch off electrical equipment until vapour fire hazard removed. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ Liquid and vapour are highly flammable. ▸ Severe fire hazard when exposed to heat or flame. ▸ Vapour forms an explosive mixture with air. ▸ Severe explosion hazard, in the form of vapour, when exposed to flame or spark. ▸ Vapour may travel a considerable distance to source of ignition. ▸ Heating may cause expansion or decomposition with violent container rupture. ▸ Aerosol cans may explode on exposure to naked flames. ▸ Rupturing containers may rocket and scatter burning materials. ▸ Hazards may not be restricted to pressure effects. ▸ May emit acrid, poisonous or corrosive fumes. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO2) isocyanates and minor amounts of hydrogen cyanide hydrogen chloride phosgene nitrogen oxides (NOx) phosphorus oxides (POx) other pyrolysis products typical of burning organic material.

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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	▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Wear protective clothing, impervious gloves and safety glasses. ▸ Shut off all possible sources of ignition and increase ventilation. ▸ Wipe up. ▸ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▸ Undamaged cans should be gathered and stowed safely.
Major Spills	For isocyanate spills of less than 40 litres (2 m2): ▸ Evacuate area from everybody not dealing with the emergency, keep them upwind and prevent further access, remove ignition sources and, if inside building, ventilate area as well as possible. ▸ Notify supervision and others as necessary. ▸ Put on personal protective equipment (suitable respiratory protection, face and eye protection, protective suit, gloves and impermeable boots). ▸ Control source of leakage (where applicable). ▸ Dike the spill to prevent spreading and to contain additions of decontaminating solution. ▸ Prevent the material from entering drains. ▸ Estimate spill pool volume or area. ▸ Absorb and decontaminate. - Completely cover the spill with wet sand, wet earth, vermiculite or other similar absorbent. - Add neutraliser (for suitable formulations: see below) to the adsorbent materials (equal to that of estimated spill pool volume). Intensify contact between spill, absorbent and neutraliser by carefully mixing with a rake and allow to react for 15 minutes ▸ Shovel absorbent/decontaminant solution mixture into a steel drum. ▸ Decontaminate surface. - Pour an equal amount of neutraliser solution over contaminated surface. - Scrub area with a stiff bristle brush, using moderate pressure. - Completely cover decontaminant with vermiculite or other similar absorbent. - After 5 minutes, shovel absorbent/decontamination solution mixture into the same steel drum used above. ▸ Monitor for residual isocyanate. If surface is decontaminated, proceed to next step. If contamination persists, repeat decontaminate procedure immediately above ▸ Place loosely covered drum (release of carbon dioxide) outside for at least 72 hours. Label waste-containing drum appropriately. Remove waste materials for incineration. ▸ Decontaminate and remove personal protective equipment. ▸ Return to normal operation. ▸ Conduct accident investigation and consider measures to prevent reoccurrence. **Decontamination:** Treat isocyanate spills with sufficient amounts of isocyanate decontaminant preparation ("neutralising fluid"). Isocyanates and polyisocyanates are generally not miscible with water. Liquid surfactants are necessary to allow better dispersion of isocyanate and neutralising fluids/ preparations. Alkaline neutralisers react faster than water/surfactant mixtures alone. Typically, such a preparation may consist of: Sawdust: 20 parts by weight Kieselguhr 40 parts by weight plus a mixture of {ammonia (s.g. 0.880) 8% v/v non-ionic surfactant 2% v/v water 90% v/v}. Let stand for 24 hours Three commonly used neutralising fluids each exhibit advantages in different situations. **Formulation A** : liquid surfactant 0.2-2% sodium carbonate 5-10% water to 100% **Formulation B** liquid surfactant 0.2-2% concentrated ammonia 3-8% water to 100% **Formulation C** ethanol, isopropanol or butanol 50% concentrated ammonia 5% water to 100% After application of any of these formulae, let stand for 24 hours. Formulation B reacts faster than Formulation A. However, ammonia-based neutralisers should be used only under well-ventilated conditions to avoid overexposure to ammonia or if members of the emergency team wear suitable respiratory protection. Formulation C is especially suitable for cleaning of equipment from unreacted isocyanate and neutralizing under freezing conditions. Regard has to be taken to the flammability of the alcoholic solution. ▸ Avoid contamination with water, alkalies and detergent solutions. ▸ Material reacts with water and generates gas, pressurises containers with even drum rupture resulting. ▸ **DO NOT** reseal container if contamination is suspected. ▸ Open all containers with care. ▸ **DO NOT** touch the spill material ▸ 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 NOT** attempt to operate damaged valve. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water courses

Continued...

	▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse / absorb vapour. ▸ Absorb or cover spill with sand, earth, inert materials or vermiculite. ▸ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. ▸ Undamaged cans should be gathered and stowed safely. ▸ Collect residues and seal in labelled drums for disposal.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ **DO NOT** incinerate or puncture aerosol cans. ▸ **DO NOT** spray directly on humans, exposed food or food utensils. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ 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 are maintained.
Other information	Consider storage under inert gas. Rotate all stock to prevent ageing. Use on FIFO (First In-First Out) basis ▸ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can ▸ Store in original containers in approved flammable liquid storage area. ▸ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped. ▸ No smoking, naked lights, heat or ignition sources. ▸ Keep containers securely sealed. Contents under pressure. ▸ Store away from incompatible materials. ▸ Store in a cool, dry, well ventilated area. ▸ Avoid storage at temperatures higher than 40 deg C. ▸ Store in an upright position. ▸ Protect containers against physical damage. ▸ Check regularly for spills and leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Aerosol dispenser. ▸ Check that containers are clearly labelled.
Storage incompatibility	▸ Avoid reaction with oxidising agents, bases and strong reducing agents. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

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	polymeric diphenylmethane diisocyanate	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Not Available
Australia Exposure Standards	dimethyl ether	Dimethyl ether	400 ppm / 760 mg/m3	950 mg/m3 / 500 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
polymeric diphenylmethane diisocyanate	0.15 mg/m3	3.6 mg/m3	22 mg/m3
dimethyl ether	3,000 ppm	3800* ppm	7200* ppm

Ingredient	Original IDLH	Revised IDLH
polymeric diphenylmethane diisocyanate	Not Available	Not Available
tris(2-chloroisopropyl)phosphate	Not Available	Not Available
dimethyl ether	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit

Continued...

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
tris(2-chloroisopropyl)phosphate	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

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.
Provide adequate ventilation in warehouse or closed storage areas.
Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

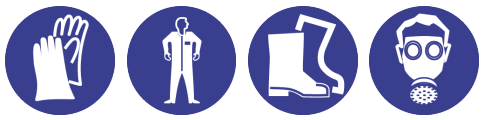
Type of Contaminant:	Speed:
aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s
direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

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

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

Personal protection



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
- Close fitting gas tight goggles

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.
- Isocyanate resistant materials include Teflon, Viton, nitrile rubber and some PVA gloves.
- Protective gloves and overalls should be worn as specified in the appropriate national standard.
- Contaminated garments should be removed promptly and should not be re-used until they have been decontaminated.
- NOTE: Natural rubber, neoprene, PVC can be affected by isocyanates

Avoid contact with moisture.
- No special equipment needed when handling small quantities.
- **OTHERWISE:**
- For potentially moderate exposures:
- Wear general protective gloves, eg. light weight rubber gloves.
- For potentially heavy exposures:
- Wear chemical protective gloves, eg. PVC. and safety footwear.

Body protection

See Other protection below

Other protection

No special equipment needed when handling small quantities.
OTHERWISE:
- Overalls.
- Skin cleansing cream.
- Eyewash unit.
- Do not spray on hot surfaces.
- The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton.

Continued...

	▸ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. BRETHERICK: Handbook of Reactive Chemical Hazards.

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:
Coltack Evolution 750 (Aerosol)

Material	CPI
BUTYL	A
NEOPRENE	A

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Type AX-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	AX-AUS P2	-	AX-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AX-AUS / Class 1 P2	-
up to 100 x ES	-	AX-2 P2	AX-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

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Liquid with a characteristic odour; does not mix with water.

Physical state	Compressed Gas	**Relative density (Water = 1)**	0.95
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Applicable
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	187

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Elevated temperatures. ▸ Presence of open flame. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7

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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	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Common, generalised symptoms associated with toxic gas inhalation include: ▸ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures; ▸ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest; ▸ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest; ▸ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain. The vapour/mist may be highly irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning for several hours after exposure. Sensitized people can react to very low doses, and should not be allowed to work in situations allowing exposure to this material. Continued exposure of sensitised persons may lead to possible long term respiratory impairment. 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. **WARNING**:Intentional misuse by concentrating/inhaling contents may be lethal. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.
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. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments
Skin Contact	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. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material may accentuate any pre-existing dermatitis condition Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Spray mist may produce discomfort 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.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. 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. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. 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

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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.
Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.
Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

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.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.
Polyisocyanates still contain small amounts of monomeric isocyanate (typically <0.5 parts per weight) and both – the polyisocyanate and the monomer - have toxicological importance. In addition, solvents also contribute to the overall toxicity of these products.
Due to the higher molecular weight and the much lower vapor pressure the polyisocyanates exhibit a significantly reduced health hazard as compared to the corresponding monomers. Nevertheless they should only be handled under controlled conditions. They are not or only slightly irritating to the skin and eyes, but might be irritating to the respiratory tract (nose, throat, lung). Polyisocyanates might act as skin sensitisers On that basis there is clear evidence from sensitive animal models that aliphatic polyisocyanates and prepolymers (HDI-based as well as IPDI-based, for example) may cause skin sensitisation. it is decided to classify all HDI-based and IPDI-based polyisocyanates and prepolymers as skin sensitisers. From animal models, however, there is no evidence that polyisocyanates are sensitising to the respiratory tract. Results from animal tests with repeated aerosol exposures indicate that under these conditions the respiratory tract is the primary target of aliphatic polyisocyanates, other organs are not significantly affected..
Available information does not provide evidence that polyisocyanates might either be mutagenic, carcinogenic or toxic to reproduction.
Polymers based on isocyanate monomers (polyurethanes) are generally of low concern. However, in the majority of cases it is not possible to conclude from the chemical name of the polymer whether an individual polyurethane is, or is not, of low concern.
Finished polyurethane polymers used in the majority of household applications contain no unreacted isocyanate groups. The production of these polymers involves the use of an excess of the hydroxyl group-containing monomer or monomers leading to complete reaction of all of the isocyanate groups.
For certain applications, however, similar polymer chemistry can be used with the isocyanate group-containing monomer in excess. This results in the formation of a polyurethane 'pre-polymer', which is intended to be further reacted in its end use. Where the pre-polymer is identified as being 'blocked', it indicates that there are no free isocyanate groups.
The polymer contained in this product has a reactive group generally considered to be of high concern (US EPA). There are health concerns for isocyanates on the basis of their skin and respiratory sensitisation properties and other lung effects e.g TDI and MDI). Aromatic isocyanates may be potentially carcinogenic (e.g. TDI and DADI). Frequently new chemical isocyanates are manufactured with a significant excess of isocyanate monomer. Whilst it is generally accepted that polymers with a molecular weight exceeding 1000 are unlikely to pass through biological membranes, oligomers with lower molecular weight and specifically, those with a molecular weight below 500, may. Estimations based on a "highly" dispersed polymer population suggest that a polymer of approximate molecular weight 5000 could contain no more than one reactive group of high concern for it to be regulated as a polymer of low concern (a so-called PLC) Polymers with a molecular weight above 10000 are generally considered to be PLCs because these are not expected to be absorbed by biological systems. The choice of 10000 as a cut-off value is thought to provide a safety factor of 100, regarded as reasonable in light of limited data, duration of studies, dose levels at which effects are seen, and extrapolation from animals to humans.
Principal route of occupational exposure to the gas is by inhalation.
Persons with a history of asthma or other respiratory problems or are known to be sensitised, should not be engaged in any work involving the handling of isocyanates.
The chemistry of reaction of isocyanates, as evidenced by MDI, in biological milieu is such that in the event of a true exposure of small MDI doses to the mouth, reactions will commence at once with biological macromolecules in the buccal region and will continue along the digestive tract prior to reaching the stomach. Reaction products will be a variety of polyureas and macromolecular conjugates with for example mucus, proteins and cell components.
This is corroborated by the results from an MDI inhalation study. Following an inhalation exposure of rats to radiolabelled MDI, 79% of the dose was excreted in faeces. The faecal excretion in these animals was considered entirely due to ingestion of radioactivity from grooming and ingestion of deposited material from the nasopharangeal region via the mucociliary escalator, i.e. not following systemic absorption. The faecal radioactivity was tentatively identified as mixed molecular weight polyureas derived from MDI. Diamine was not present. Thus, for MDI and diisocyanates in general the oral gavage dosing route is inappropriate for toxicological studies and risk assessment.
It is expected that oral gavage dosing will result in a similar outcome to that produced by TDI or MDI, that is (1) reaction with stomach contents and (2) polymerization to solid polyureas.

- Reaction with stomach contents is very plausibly described in case reports of accidental ingestion of polymeric MDI based glue in domestic animals. Extensive polymerization and CO2 liberation resulting in an expansion of the gastric content is described in the stomach, without apparent acute chemical toxicity
- Polyurea formation in organic and aqueous phases has been described. In this generally accepted chemistry of hydrolysis of an isocyanate the initially produced carbamate decarboxylates to an amine which. The amine, as a reactive intermediate, then reacts very readily with the present isocyanate to produce a solid and inert polyurea. This urea formation acts as a pH buffer in the stomach, thus promoting transformation of the diisocyanate into polyurea, even under the acidic conditions.

At the resorbtive tissues in the small intestine, these high molecular reaction products are likely to be of very low bioavailability, which is substantiated by the absence of systemic toxicity in acute oral bioassays with rats at the OECD limit dose (LC50>2 g/kg bw).
The respiratory tract may be regarded as the main entry for systemically available isocyanates as evidenced following MDI.exposures.
A detailed summary on urinary, plasma and in vitro metabolite studies is provided below. Taken together, all available studies provide convincing evidence that MDI-protein adduct and MDI-metabolite formation proceeds:

- via formation of a labile isocyanate glutathione (GSH)-adduct,
- then transfer to a more stable adduct with larger proteins, and
- without formation of free MDA. MDA reported as a metabolite is actually formed by analytical workup procedures (strong acid or base hydrolysis) and is not an identified metabolite in urine or blood

A 90-day inhalation study in rats with polymeric MDI (6 hours/day, 5 days/week) produced moderate to severe hyperplastic inflammatory lesions in the nasal cavities and lungs at levels of 8 mg/m3 or greater.
Rats exposed for two years to a respirable aerosol of polymeric MDI exhibited chronic pulmonary irritation at high concentrations. Only at the highest level (6 mg/m3),was there a significant incidence of a benign tumour of the lung (adenoma) and one malignant tumour (adenocarcinoma).There were no lung tumours at 1 mg/m3 and no effects at 0.2 mg/m3. Overall, the tumour incidence, both benign and malignant and the number of animals with the tumours were not different from controls.The increased incidence of lung tumours is associated with prolonged respiratory irritation and the concurrent accumulation of yellow material in the lung, which occurred throughout the study.In the absence of prolonged exposure to high concentrations leading to chronic irritation and lung damage,it is highly unlikely that tumour formation will occur.
Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing

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	difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities. Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages. Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.

Coltack Evolution 750 (Aerosol)	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
polymeric diphenylmethane diisocyanate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >9400 mg/kg[2]	Eye (rabbit): 100 mg - mild
	Inhalation(Rat) LC50; 0.49 mg/L4h[2]	
	Oral(Rat) LD50; 43000 mg/kg[2]	
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):
	Oral(Rat) LD50; >500 mg/kg[1]	
dimethyl ether	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >20000 ppm4h[1]	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*	

POLYMERIC DIPHENYLMETHANE DIISOCYANATE	product 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. Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. 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. Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities. Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages. Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. for diisocyanates: In general, there appears to be little or no difference between aromatic and aliphatic diisocyanates as toxicants. In addition, there are insufficient data available to make any major distinctions between polymeric (<1000 MW) and monomeric diisocyanates. Based on repeated dose studies in animals by the inhalation route, both aromatic and aliphatic diisocyanates appear to be of high concern for pulmonary toxicity at low exposure levels. Based upon a very limited data set, it appears that diisocyanate prepolymers exhibit the same respiratory tract effects as the monomers in repeated dose studies. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route. Most members of the diisocyanate category have not been tested for carcinogenic potential. Though the aromatic diisocyanates tested positive and the one aliphatic diisocyanate tested negative in one species, it is premature to make any generalizations about the carcinogenic potential of aromatic versus aliphatic diisocyanates. In the absence of more human data, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Diisocyanates are moderate to strong dermal

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	sensitisers in animal studies. Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. For monomers, effects on the respiratory tract (lungs and nasal cavities) were observed in animal studies at exposure concentrations of less than 0.005 mg/L. The experimental animal data available on prepolymeric diisocyanates show similar adverse effects at levels that range from 0.002 mg/L to 0.026 mg/L. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route **Oncogenicity:** Most members of the diisocyanate category have not been tested for carcinogenic potential. Commercially available Poly-MDI was tested in a 2-year inhalation study in rats. The tested material contained 47% aromatic 4,4'-methylenediphenyl diisocyanate (MDI) and 53% higher molecular weight oligomers. Interim sacrifices at one year showed that males and females in the highest dose group (6 mg/m3) had treatment related histological changes in the nasal cavity, lungs and mediastinal lymph nodes. The incidence and severity of degeneration and basal cell hyperplasia of the olfactory epithelium and Bowman's gland hyperplasia were increased in males at the mid and high doses and in females at the high dose following the two year exposure period. Pulmonary adenomas were found in 6 males and 2 females, and pulmonary adenocarcinoma in one male in the high dose group. However, aliphatic hexamethylene diisocyanate (HDI) was found not to be carcinogenic in a two year repeated dose study in rats by the inhalation route. HDI has not been tested in mice by the inhalation route. Though the oral route is not an expected route of exposure to humans, it should be noted that in two year repeated dose studies by the oral route, aromatic toluene diisocyanate (TDI) and 3,3'-dimethoxy-benzidine-4,4'-diisocyanate (dianisidine diisocyanate, DADI) were found to be carcinogenic in rodents. TDI induced a statistically significant increase in the incidence of liver tumors in rats and mice as well as dose-related hemangiosarcomas of the circulatory system and has been classified by the Agency as a B2 carcinogen. DADI was found to be carcinogenic in rats, but not in mice, with a statistically increase in the incidence of pancreatic tumors observed. **Respiratory and Dermal Sensitization:** Based on the available toxicity data in animals and epidemiologic studies of humans, aromatic diisocyanates such as TDI and MDI are strong respiratory sensitisers. Aliphatic diisocyanates are generally not active in animal models for respiratory sensitization. However, HDI and possibly isophorone diisocyanate (IPDI), are reported to be associated with respiratory sensitization in humans. Symptoms resulting from occupational exposure to HDI include shortness of breath, increased bronchoconstriction reaction to histamine challenges, asthmatic reactions, wheezing and coughing. Two case reports of human exposure to IPDI by inhalation suggest IPDI is a respiratory sensitiser in humans. In view of the information from case reports in humans, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Studies in both human and mice using TDI, HDI, MDI and dicyclohexylmethane-4,4'-diisocyanate (HMDI) suggest cross-reactivity with the other diisocyanates, irrespective of whether the challenge compound was an aliphatic or aromatic diisocyanate. Diisocyanates are moderate to strong dermal sensitisers in animal studies. There seems to be little or no difference in the level of reactivity between aromatic and aliphatic diisocyanates. **Dermal Irritation:** Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. The level of irritation ranged from slightly to severely irritating to the skin. One chemical, hydrogenated MDI (1,1-methylenebis-4-isocyanatocyclohexane), was found to be corrosive to the skin in guinea pigs. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
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

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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

Continued...

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-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.

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

Coltack Evolution 750 (Aerosol)

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

polymeric diphenylmethane diisocyanate

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
ErC50	72h	Algae or other aquatic plants	4mg/l	1
EC50	72h	Algae or other aquatic plants	33mg/l	2
BCF	1008h	Fish	0.8-2.8	7
EC50	48h	Crustacea	65335mg/l	1
LC50	96h	Fish	11mg/l	2
EC50(ECx)	96h	Algae or other aquatic plants	4mg/l	1

Continued...

	Endpoint	Test Duration (hr)	Species	Value	Source
dimethyl ether	EC50	48h	Crustacea	>4400mg/L	2
	LC50	96h	Fish	1783.04mg/l	2
	NOEC(ECx)	48h	Crustacea	>4000mg/l	1
	EC50	96h	Algae or other aquatic plants	154.917mg/l	2

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

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

Chlorinated trisphosphates are clear liquids or low-melting solids, with little or no odour. They:
- are denser than water, the density increases with the number of chlorines in the molecule
- have low vapour pressure (typically >1 Pa at ambient temperatures and as a consequence have high boiling
- points (>200 C); they are considered to exhibit some volatility (the literature sometimes describes "appreciable" volatility but this is a description relative to other fire retardants)
- cannot be distilled at atmospheric pressures with decomposition
- have high flash points
- are soluble to a limited extent in water (but readily soluble in ketones, alcohols and chlorinated hydrocarbons); solubilities range from 0.053 g/l (TDCPP) to 8 g/l TCEP)
- have moderate log Kow values, increasing with increasing levels of chlorination (1.7 - 3.8)
- are hydrolysed to produce phosphoric acid and a chlorinated alcohol - there may also be hydrolytic attack on aliphatic chlorines to produce ethylene glycol, propylene glycol or glycerol

Environmental fate:
Trisphosphates are somewhat volatile and are likely to be slowly released to the atmosphere from the surfaces of solid articles containing these compounds during normal use. Some may be released to waste water during washing of fabrics containing these substances.
Atmospheric fate: Once in the atmospheric compartment the compounds are destroyed through reaction with atmospheric hydroxyl radicals. Generally th primary pathway for degradation in the atmosphere will be through hydrogen abstraction by OH radicals. Assuming the accepted global average atmospheric concentrations of hydroxyl radicals to be 5 x 10+5 moles/cm3, the atmospheric half-lives range from 35 hrs (TCPP) to 102 hours (TCEP). Degradation of the compounds in the atmosphere is expected to lead to ultimate destruction with formation of HCl, water and carbon dioxde. The phosphorus component is likely to be converted to phosphoric acid and precipitated (along with HCl) to the surface with rain
Aquatic fate: Trisphosphates are normally not readily biodegradable under aerobic conditions and due to their relatively high water solubility are not expected to bioaccumulate
Chlorinated trisphosphates may be released to the water compartment primarily in landfill leachate resulting from degradation of polyurethane foam and polyester.
Generally these compounds show some water solubility and modest log Kows. Even where the log Kow is relatively high (as with TDCPP, log Kow 3.4) environmental models indicate that only around 25% of the compound becomes associated with sludge while 5% may be released into the air - the rest dissolves.
Terrestrial fate: It is estimated that these compounds have an affinity for the organic components of soils and sediments (log Koc >2) and are expected to have low mobility in these media. The relatively high water solubility indicates that partitioning from soil to water phase is possible and that mobility in and from soil media may be quite high.
TDCPP is most likely to become associated with soil and sediments and given its apparent resistance to aerobic bacterial degradation is also likely to be persistant in aerobic soils and sediments.
Abiotic degradation: Hydrolysis of the compounds in aqueous solution is likely to be slow in the normal environmental range 4
Biodegradation: Chlorinated trisphosphates are generally not readily biodegradable according to OECD criteria but the lower chlorinated species may be "ultimately" or inherently biodegradable Species with higher chlorination seem resistant to biodegradation.
While refractory to aerobic bacterial biodegradation these compounds may be metabolised by higher organisms. TDCPP had a half-life of 31 hours in water containing killifish (Oryzias latipes) and 42 hours in water containing goldfish (Carassius auratus)
Bioaccumulation : Relatively high water solubilities and modest values of log Kow indicate a low potential for bioaccumulation. Measured bioconcentration factors (BCF) are low. The largest measured BCF is 107 found for killifish (Oryzias latipes) exposed to 0.3-1.2 mg/l TDCPP for 96 hours. Similar low values were found for TCEP. On transfer of fish to clean water elimination of TDCPP from fish tissues was rapid with a half-life for elimination of 1.65 hours
Ecotoxicity
TCEP and TCPP are slightly toxic to aquatic organisms at all trophic levels and TDCPP is moderately toxic to fish. These compounds are slightly toxic to terrestrial species,, aquatic green algae but are non-toxic to sewage bacteria

for polyisocyanates:
Polyisocyanates are not readily biodegradable. However, due to other elimination mechanisms (hydrolysis, adsorption), long retention times in water are not to be expected. The resulting polyurea is more or less inert and, due to its molecular size, not bioavailable. Within the limits of water solubility, polyisocyanates have a low to moderate toxicity for aquatic organisms.
Hydrolysis would represents the primary fate mechanism for the majority of the commercial isocyanate monomers, but, is tempered somewhat by the lack of water solubility. In the absence of hydrolysis, sorption to solids (e.g., sludge and sediments) will be the primary mechanism of removal. Hydrolysis products are predominantly insoluble stable polyureas.
Biodegradation is minimal for most compounds and volatilisation is negligible. Atmospheric degradation is not expected with removal from air occurring by washout or dry deposition.
Volatilisation from surface waters (e.g., lakes and rivers) is expected to take years. In wastewater treatment this process is not expected to be significant.
Review of the estimated properties of the isocyanates suggest that sorption is the primary removal mechanism in the ambient environment and in wastewater treatment in the absence of significant hydrolysis. Sorption to solids in wastewater treatment is considered strong to very strong for most compounds. Sorption to sediments and soils in the ambient environment is very strong in most instances. Migration to groundwater and surface waters is not expected due to sorption or hydrolysis.
Hydrolysis of the N=C=O will occur in less than hours in most instances and within minutes for more than 90% of the commercial isocyanates. However, the low to very low solubility of these substances will generally lessen the effectiveness of hydrolysis as a fate pathway. But hydrolysis should be considered one of the two major fate processes for the isocyanates.
Aerobic and/or anaerobic biodegradation of the isocyanates is not expected to occur at significant levels. Most of the substances take several months to degrade.
Degradation of the hydrolysis products will occur at varying rates depending on the moiety formed.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
tris(2-chloroisopropyl)phosphate	HIGH	HIGH
dimethyl ether	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
tris(2-chloroisopropyl)phosphate	LOW (BCF = 4.6)
dimethyl ether	LOW (LogKOW = 0.1)

Mobility in soil

Ingredient	Mobility

Continued...

Ingredient	Mobility
tris(2-chloroisopropyl)phosphate	LOW (KOC = 1278)
dimethyl ether	HIGH (KOC = 1.292)

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. ▸ Consult State Land Waste Management Authority for disposal. ▸ Discharge contents of damaged aerosol cans at an approved site. ▸ Allow small quantities to evaporate. ▸ **DO NOT** incinerate or puncture aerosol cans. ▸ Bury residues and emptied aerosol cans at an approved site.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	Class: 2.1 Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 63 190 277 327 344 381 Limited quantity: 1000ml

Air transport (ICAO-IATA / DGR)

UN number	1950
UN proper shipping name	Aerosols, flammable
Transport hazard class(es)	ICAO/IATA Class: 2.1 ICAO / IATA Subrisk: Not Applicable ERG Code: 10L
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A145 A167 A802 Cargo Only Packing Instructions: 203 Cargo Only Maximum Qty / Pack: 150 kg Passenger and Cargo Packing Instructions: 203 Passenger and Cargo Maximum Qty / Pack: 75 kg Passenger and Cargo Limited Quantity Packing Instructions: Y203 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	IMDG Class: 2.1 IMDG Subrisk: Not Applicable

Continued...

Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-D , S-U Special provisions: 63 190 277 327 344 381 959 Limited Quantities: 1000 ml

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

Not Applicable

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

Product name	Group
polymeric diphenylmethane diisocyanate	Not Available
tris(2-chloroisopropyl)phosphate	Not Available
dimethyl ether	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
polymeric diphenylmethane diisocyanate	Not Available
tris(2-chloroisopropyl)phosphate	Not Available
dimethyl ether	Not Available

SECTION 15 Regulatory information

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

polymeric diphenylmethane diisocyanate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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)

dimethyl ether is found on the following regulatory lists

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (polymeric diphenylmethane diisocyanate; tris(2-chloroisopropyl)phosphate; dimethyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (polymeric diphenylmethane diisocyanate)
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	30/09/2021
Initial Date	30/09/2021

Continued...

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