SAFETY DATA SHEET 2022

SECTION 1: IDENTIFICATION OF MATERIAL AND SUPPLIER

Product Name : ShieldFoam PLF Part A

Other means of Identification : None

Relevant Identified Uses : Part A Liquid component of Polyurethane Foam

Supplier's Information

Name : ShieldCrete® International

Company Name : ShieldCrete® International Sdn Bhd

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SECTION 2: HAZARDS IDENTIFICATION

Classification of the substance or mixture:

Poison Schedule: Not Applicable

Classification: Flammable Liquid Category 3, Acute Toxicity (Oral) Category 4

Legend: 1. Classified by ShieldCrete® International; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label Elements:

Poison Schedule: **Warning**

Hazard Statements:

H226	Flammable liquid and vapor.
H302	Harmful if swallowed.

Precautionary Statement(s) Prevention:

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P233	Keep container tightly closed.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P270	Do not eat, drink, or smoke when using this product.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary Statement(s) Response:

P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P330	Rinse mouth.

Precautionary Statement(s) Storage:

P403+P235	Store in a well-ventilated place. Keep cool.

Precautionary Statement(s) Disposal:

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

SECTION 3: COMPOSITES / INFORMATION ON INGREDIENTS

Substances: See section below for composition of Mixtures

Mixtures:

INGREDIENTS	WEIGHT %	CAS No.
Polyethylene/polypropylene Glycol Glyceryl Ether	>60	9082-00-2
Tris (2-chloroisopropyl) Phosphate	10 - <30	13674-84-5
1,1-dichloro-1-fluoroethane	<10	1717-00-6
Ingredients determined not to be hazardous	<10	Not Available

SECTION 4: FIRST AID MEASURES

Description of first aid measures

Inhalation: If fumes or combustion products are inhaled remove from contaminated area. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor.

Ingestion: If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness, i.e., becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice.

Eye Contact: Immediately hold eyelids apart and flush the eye continuously with running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. Transport to hospital or doctor without delay. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

Skin Contact: Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation.

Indication of any immediate medical attention and special treatment needed: Treat symptomatically.

SECTION 5: FIRE FIGHTING MEASURES

Extinguishing Media:
Water spray or fog.
Alcohol stable foam.
Dry chemical powder.
BCF (where regulations permit).
Carbon dioxide.

Special Hazards arising from the Substrate or Mixture

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

Advice for Firefighters

Fire Fighting:	Alert Fire Brigade and tell them location and nature of hazard. Wear full body protective clothing with breathing apparatus. Prevent, by any means available, spillage from entering drains or water course. Use water delivered as a fine spray to control fire and cool adjacent area. Avoid spraying water onto liquid pools. DO NOT approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard:	Combustible. Slight fire hazard when exposed to heat or flame. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO). May emit acrid smoke. Mists containing combustible materials may be explosive. Combustion products include carbon dioxide (CO2), aldehydes, hydrogen chloride, phosgene, phosphorus oxides (POx), other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
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	Remove all ignition sources.
	Clean up all spills immediately.
	Avoid breathing vapors and contact with skin and eyes.
	Control personal contact with the substance, by using protective equipment.
	Contain and absorb spill with sand, earth, inert material, or vermiculite.
	Wipe up.
	Place in a suitable, labelled container for waste disposal.
Major Spills	Moderate hazard.
	Clear area of personnel and move upwind.
	Alert Fire Brigade and tell them location and nature of hazard.
	Wear breathing apparatus plus protective gloves.
	Prevent, by any means available, spillage from entering drains or water course.
	No smoking, naked lights, or ignition sources.
	Increase ventilation.
	Stop leak if safe to do so.
	Contain spill with sand, earth, or vermiculite.
	Collect recoverable product into labelled containers for recycling.
	Absorb remaining product with sand, earth, or vermiculite.
	Collect solid residues and seal in labelled drums for disposal.
	Wash area and prevent runoff into drains.
	If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7: HANDLING AND STORAGE

Precautions for Safe Handling

Safe Handling:

Avoid all personal contact, including inhalation.
Wear protective clothing when risk of exposure occurs.
Use in a well-ventilated area.
Prevent concentration in hollows and sumps.
DO NOT enter confined spaces until atmosphere has been checked.
Avoid smoking, naked lights or ignition sources.
Avoid contact with incompatible materials.
When handling, DO NOT eat, drink, or smoke.
Keep containers securely sealed when not in use.
Avoid physical damage to containers.
Always wash hands with soap and water after handling.
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.

Other Information:

Store in original containers.
Keep containers securely sealed.
No smoking, naked lights, or ignition sources.
Store in a cool, dry, well-ventilated area.
Store away from incompatible materials and foodstuff containers.
Protect containers against physical damage and check regularly for leaks.
Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for Safe Storage, including any Incompatibilities

Suitable Container:

Metal can or drum.
Packaging as recommended by manufacturer.
Check all containers are clearly labelled and free from leaks.

Storage incompatibility: Avoid reaction with oxidizing agents.
Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8: EXPOSURE CONTROLS / PERSONAL PROTECTION

Control Parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)
INGREDIENT DATA: Not Available

Emergency Limits:

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
Polyethylene/ Polypropylene Glycol Glyceryl Ether	Polyglycol 15-200; (Oxirane, 2-methyl-, polymer with oxirane, ether with 1,2,3-propanetriol (3:1); Calthane NF and ND "B")	30 mg/m3	330 mg/m3	2,000 mg/m3
1,1-dichloro-1-fluoroethane	HCFC-141b; (Dichloro-1-fluoroethane, 1,1-; Freon 141)	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
Polyethylene/ Polypropylene Glycol Glyceryl Ether	Not Available	Not Available
Tris(2-chloroisopropyl) Phosphate	Not Available	Not Available
1,1-dichloro-1-fluoroethane	Not Available	8,245 mg/m3 / 1,700 ppm

Occupational Exposure Banding:

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.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances.

Correct fit is essential to ensure adequate protection. An approved self-contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Appropriate Engineering Controls

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

Within each range the appropriate value depends on:

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

	Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore, the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Personal Protection	
Eye and Face Protection	▪ Safety glasses with side shields. ▪ Chemical goggles. ▪ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent].
Skin Protection	See Hand Protection below
Hands/Feet Protection	▪ Wear chemical protective gloves, e.g., PVC. ▪ Wear safety footwear or safety gumboots, e.g., Rubber
Body Protection	See Other Protection below
Other Protection	▪ Overalls ▪ P.V.C. apron ▪ Barrier cream ▪ Skin cleansing cream ▪ Eye wash unit

Respiratory Protection

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

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

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	AX-AUS / Class1 P2	-
up to 50	1000	-	AX-AUS / Class 1 P2
up to 50	5000	Airline*	-
up to 100	5000	-	AX-2 P2
up to 100	10000	-	AX-3 P2
100+			Airline**

* Continuous Flow ** Continuous-flow or positive pressure demand
A (All classes) = Organic vapors, 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°C)

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapor concentrations or oxygen content.

The wearer must be warned to leave the contaminated area immediately on detecting any odors through the respirator. The odor may indicate that the mask is not functioning properly, that the vapor 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 hours of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hours. Used cartridges should be discarded daily, regardless of the length of time used.

SECTION 9: PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Clear amber liquid with mild pungent odor, partially mixes with water.
Physical State	Liquid
Odor	Not Available
Odor Threshold	Not Available
pH (as supplied)	Not Applicable
Melting Point / Freezing Point (°C)	Not Applicable
Initial Boiling Point and Boiling Range (°C)	Not Available
Flash Point (°C)	>35 (COC, ASTM D-92)
Evaporation Rate	Not Available
Flammability	Flammable
Upper Explosive Limit (%)	Not Available
Lower Explosive Limit (%)	Not Available
Vapor Pressure (kPa)	<7 @ 25°C
Solubility in water	Partly miscible
Vapor Density (Air = 1)	>1
Relative density (Water = 1)	1.08 @ 25 deg C
Partition coefficient n-octanol / water	Not Available
Auto-ignition temperature (°C)	Not Available
Decomposition temperature	Not Available
Viscosity (cSt)	Not Available
Molecular weight (g/mol)	Not Applicable
Taste	Not Available
Explosive properties	Not Available
Oxidizing Properties	Not Available
Surface Tension (dyn/cm or mN/m)	Not Available
Volatile Component (%vol)	Not Available
Gas Group	Not Available
pH as a solution (1%)	Not Available
VOC g/L	Not Available

SECTION 10: STABILITY AND REACTIVITY

Reactivity: See section 7

Chemical Stability: Unstable in the presence of incompatible materials.
Product is considered stable.
Hazardous polymerization will not occur.

Possibility of hazardous reactions: See section 7

Conditions to avoid: See section 7

Incompatible materials: See section 7

Hazardous decomposition products: See section 5

SECTION 11: TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled: Inhalation hazard is increased at higher temperatures. Inhalation of vapors or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Vapors may cause drowsiness and dizziness.

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.

Skin Contact: Repeated exposure may cause skin cracking, flaking, or drying following normal handling and use. Open cuts abraded or irritated skin should not be exposed to this material. Entry into the bloodstream 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: Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to 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 characterized by temporary redness (like windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Chronic: Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.

ShieldFoam PLF Part A	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
Polyethylene/ Polypropylene Glycol Glyceryl Ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >5000 mg/kg[2]	Not Available
	Oral (rat) LD50: >10000 mg/kg[2]	
Tris (2-chloroisopropyl) Phosphate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >5000 mg/kg[2]	Eye (rabbit): non-irritating*
	Inhalation (rat) LC50: >7 mg/l4 h[1]	Skin (rabbit): mild (24 h)
	Oral (rat) LD50: ~500-2000 mg/kg[2]	
1,1-dichloro-1-fluoroethane	**TOXICITY**	**IRRITATION**
	Dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Inhalation (rat) LC50: 239.9 mg/l/4H[2]	
	Oral (rat) LD50: >5000 mg/kg[2]	
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	

POLYETHYLENE/ POLYPROPYLENE GLYCOL GLYCERYL ETHER	No significant acute toxicological data identified in literature search.
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 known to bloom from car interior plastics, TVs, and computer VDUs. Acute toxicity: In rats, oral doses of TCEP are absorbed and distributed around the body to various organs, particularly the liver and kidney, but also the brain. Metabolites in rats and mice include bis(2-chloroethyl) carboxymethyl phosphate; bis(2-chloroethyl) hydrogen phosphate; and bis (2-chloroethyl)-2-hydroxyethyl phosphate glucuronide. Excretion is rapid, nearly complete, and mainly via the urine. TCEP is of low to moderate acute oral toxicity (oral LD50 in the rat = 1150 mg/kg body weight). In repeat dose studies, TCEP caused adverse effects on the brain (hippocampal lesions in rats), liver and kidneys. The NOEL was 22 mg/kg body weight per day and the LOEL 44 mg/kg body weight per day for increased weights of liver and kidneys in rats TCPP is of low to moderate acute toxicity by the oral (LD50 in rats = 1017-4200 mg/kg body weight), dermal (LD50 in rats and rabbits is >5000 mg/kg body weight) and inhalation routes (LC50 in rats is > 4.6 mg/liter). 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. Sensitization 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. A 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 vivo mutagenicity studies investigating an appropriate range of endpoints 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, th mutagenicity data show that TDCPP is not genotoxic in vivo. Carcinogenicity: TCEP causes benign and malignant tumors 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 tumors were also found. In addition, there were non-neoplastic adverse effects in bone marrow, spleen, testis, liver, and kidney. The effects in the kidney and testis occurred at all exposure levels. Only animals in the highest dose and control groups were evaluated for effects in the bone marrow and spleen. It was impossible, therefore, to determine whether there was a dose-response relationship for these effects in these organs. TDCiPP produces liver tumors 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 optimized 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 characterized 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, labored 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 principal 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 sensitization potential. No information is available on the respiratory sensitization 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 characterization. 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 characterization will be carried out for this endpoint. It is proposed that the effects observed in the 90-day study for TCPP are taken as a starting point for risk characterization. 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 characterization of the carcinogenicity endpoint.

	Reproductive toxicity: In a two-generation reproductive toxicity study with TCPP, there were no treatment related effects in pre-coital time, mating index, female fecundity index, male and female fertility index, duration of gestation and post-implantation loss. There was no effect on sperm parameters at necropsy. In females, the length of the longest oestrus cycle and the mean number of cycles per animal were statistically significantly increased in high dose animals of both generations. A decrease in uterus weight was observed in all dosed females in F0 and in high dose females in F1. Effects were also noted on pituitary weights, significant in high dose females of both generations. A LOAEL of 99 mg/kg is derived for effects on fertility. This is based on effects on the effect on uterus weight seen in all dosed females in F0 and high dose females in F1. Developmental toxicity: From the same study, a LOAEL of 99 mg/kg is derived for developmental toxicity. This is based on a treatment related effect on the number of runts observed in all TCPP-treated groups of the F0 generation. In a separate study, no treatment-related effects on foetal mortality, implantation number, resorption or foetal weight were observed following treatment of pregnant dams with TCPP. Cervical ribs and missing 13th ribs were noted at a low incidence in all treatment groups, but not in the control group. However, as a specific rib count undertaken in the 2-generation study did not reveal an increase in this effect, it is concluded that this is not toxicologically significant. Weaning rate and rearing condition were unaffected by treatment and there was no evidence of any abnormality for alkyl esters of phosphoric acid: The chemicals in this category exhibit a low to moderate order of acute toxicity. The rat oral LD50 values ranged from 500-1000 mg/kg with 2-ethylhexyl phosphate to >36,800 mg/kg for tris(2-ethylhexyl) phosphate. The dermal LD50 values ranged from 1200 to > 2000 mg/kg (rat) with bis(2-ethylhexyl) hydrogen phosphate to > 20,000 mg/kg (rabbit) with tris (2-ethylhexyl) phosphate. The inhalation LC50 values ranged from > 0.447 mg/l (4 hr. rat) with tris(2-ethylhexyl) phosphate to > 5.14 mg/l (4 hr. rat) with triisobutyl phosphate. Metabolism: Phosphoric acid esters are metabolized via dealkylation. Metabolism studies conducted on the tributyl phosphate indicate that dealkylation to form the alkyl alcohol is the primary route of metabolism Phosphoric acid tri-esters are rapidly metabolized to di-esters with mono-diesters also being produced. Studies of tributyl phosphate show that 40-64% of the parent compound is metabolized to dibutyl dihydrogen phosphate and that 1.1-2.1 % is metabolized to the monobutyl species. Therefore, tris(2-ethylhexyl) phosphate is expected to be metabolized 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 NOAELs for rats and rabbits were 250 mg/kg/day and 25 mg/kg/day, respectively.
1,1-DICHLORO-1-FLUOROETHANE	Chlorofluorocarbons may enter the human organism by inhalation, ingestion, or dermal contact. Inhalation is the most common and important route of entry, and exhalation is the most significant route of elimination from the body. Controlled studies with volunteer subjects and experimental animals have provided substantial data from exposures to a number of the chlorofluorocarbons. CFCs and HCFCs are known to sensitize the heart to adrenalin-induced arrhythmias. CFCs: ▪ can be absorbed across the alveolar membrane, gastro- intestinal tract, or the skin; ▪ are absorbed rapidly into the blood, following inhalation; ▪ are absorbed into the blood at a decreasing rate as blood concentration increases; ▪ once in the blood, are absorbed by various tissues; ▪ will reach a stable blood level if exposure is sufficiently long, indicating an equilibrium between the air containing the chlorofluorocarbons and the blood; are still absorbed by body tissue, after the initial blood level stabilization, and continue to enter the body.

Studies with animals indicate that chlorofluorocarbons are rapidly absorbed after inhalation and are distributed by blood into practically all tissues of the body. The highest concentrations are usually found in fatty or lipid-containing tissues. However, chlorofluorocarbons are also found in organs with a good blood supply, e.g., heart, lung, kidney, muscle. Results from animal and human metabolic studies have demonstrated the resistance of chlorofluorocarbons to breakdown or metabolic transformation in biological systems. These results suggest that chlorofluorocarbons, in general, are metabolized to a very small degree, if at all, following exposure. Regardless of the route of entry, chlorofluorocarbons are eliminated almost exclusively through the respiratory tract via exhaled air. No significant recovery of chlorofluorocarbons or their metabolites has been reported in studies attempting to identify metabolic transformation products via elimination in urine or feces.

The acute inhalation toxicity of chlorofluorocarbons has been extensively studied. The chlorofluorocarbons generally show low acute inhalation toxicity. The symptomatology of acute intoxication involves CNS effects, secondary effects on the cardiovascular system, and irritation of the respiratory tract. At high concentrations, human subjects experienced a tingling sensation, humming in the ears, and apprehension. EEG changes were noted as well as slurred speech and de- creased performance in psychological tests. An exposure to an 11% (545 g/m3) concentration of CFC-12 for 11 min caused a significant degree of cardiac arrhythmia, followed by a decrease in consciousness with amnesia after 10 min. Significant acute reduction in the ventilatory lung capacity of hairdressers using chlorofluorocarbon-containing hairsprays was observed in several studies. Cases of neurological effects attributed to occupational exposure to chlorofluorocarbons have been reported. Non-occupational exposure and accidental or abusive inhalation of aerosols have also been documented, the main symptoms being CNS depression and cardiovascular reactions. Cardiac arrhythmia, possibly aggravated by elevated levels of catecholamines due to stress or by moderate hypercapnia (a condition where there is too much carbon dioxide (CO2) in the blood), is suggested as the cause of these adverse responses, which may lead to death.

The limited information available on the acute oral toxicity of chlorofluorocarbons indicates low toxicity. When applied dermally in high doses, CFCs cause various degrees of irritation but no other significant effects. Limited studies indicate that individuals with a prior history of skin reaction to deodorant sprays containing CFC-11 or CFC-12 may become sensitized to dermal applications of certain chlorofluorocarbons.

The available information indicates that the fully halogenated chlorofluorocarbons have little or no mutagenic or carcinogenic potential. Negative results have been obtained in vitro using bacteria and mammalian cells with or without metabolic activation and in the dominant lethal test.

Long-term carcinogenicity studies (by oral and inhalation routes) with CFC-11 and CFC-12 in rats and mice showed negative results.

Although a tumorigenic response in the nasal cavity was observed in rats upon inhalation of CFC-113, this response was considered equivocal. The tumors were of various morphologies and the incidences were not dose-related it has been also suggested that supersensitive 5-HT(1B/1D) receptors may be involved in the pathophysiology of obsessive-compulsive disorders (OCD). In the 5-HT(1B/1D) agonist field, since the discovery of sumatriptan (26) (a 5-HT(1B/1D) receptor agonist) as an effective treatment for migraine headache, intensive research in this area has led to several second-generation compounds, a few of which have either entered the marketplace or are in late clinical trials. Beside the antimigraine activity of the 5-HT(1B/1D) agonists in clinical evaluation or already on the market, other potential therapeutic evaluations (such as gastric motor effect, bipolar disorder, autism, anti-aggressive effects) with these drugs are being investigated Cerebral hemorrhage, subarachnoid hemorrhage, stroke, and other cerebrovascular events have been reported in patients treated with 5-HT1 agonists; and some have resulted in fatalities. In a number of cases, it appears possible that the cerebrovascular events were primary, the agonist having been administered in the incorrect belief that the symptoms experienced were a consequence of migraine, when they were not. It should be noted that patients with migraine may be at increased risk of certain cerebrovascular events (e.g., stroke, hemorrhage, transient ischemic attack).

An 18% increase in mean pulmonary artery pressure was seen following dosing with one 5-HT1 agonist in a study evaluating subjects undergoing cardiac catheterization. 5-HT1 Agonists may cause vasospastic reactions other than coronary artery vasospasm such as peripheral and gastrointestinal vascular ischaemia. Significant elevations in systemic blood pressure have been reported on rare occasions Very rare gastrointestinal ischaemic events including ischemic colitis and gastrointestinal infarction or necrosis have been reported with 5HT1 agonists; these may present as bloody diarrhea or abdominal pain.

For dichlorotrifloroethane (HCFC -123) and dichloropentafluoropropane (HCFC-225). Prolonged inhalation of high concentrations of HCFC-123 vapor may cause temporary nervous system depression with anesthetic effects such as dizziness, headache, confusion, incoordination, and loss of consciousness. With gross overexposure (greater than 20% concentration), a temporary alteration of the heart's electrical activity with irregular pulse, palpitations, or inadequate circulation may occur. Similar effects are observed in overexposure to CFC-11. Inhalation may cause liver effects with extended high-level exposures. Intentional misuse or deliberate inhalation of HCFC-123 may cause death without warning.

Exposure in the range of 650 to 1,000 ppm with HCFC-225ca produced effects on the liver in rodents, but a minor effect in a primate. Exposures in the range of 1,000 to 5,000 ppm with HCFC-225cb resulted in only marginal effects in rodents or a primate.

If HCFC-123 vapours are inhaled at a concentration of 20,000 ppm or greater, the heart may become sensitized to adrenaline leading to cardiac irregularities and, possibly, to cardiac arrest. Similar effects are observed with CFC-11 at concentrations in air of 3,500 ppm or greater. The likelihood of these cardiac problems increases under physical or emotional stress. Because of possible disturbances of cardiac rhythm, catecholamine drugs, such as epinephrine, should be considered only as a last resort in life-threatening emergencies. As with many other halocarbons and hydrocarbons, inhalation of HCFC-225ca and HCFC-225cb followed by intravenous injection of epinephrine, which simulates human stress reactions, results in a cardiac sensitization response in experimental screening studies with dogs. This cardiac sensitization response is observed at approximately 15,000 ppm for the mixture of HCFC-225ca/ HCFC-225cb (45/55 weight percent) and 20,000 ppm for HCFC-225cb, which are levels well above expected exposures. By comparison, a cardiac sensitization response is observed with CFC-113 at approximately 5,000 ppm HCFC-225ca and HCFC-225cb have low acute oral, dermal and inhalation toxicity. Neither isomer causes eye irritation nor dermal toxicity in standardized tests; skin application of both isomers at high doses (2,000 mg/kg body weight) produces no adverse effects.

HCFC-225ca and HCFC-225cb have low acute oral, dermal and inhalation toxicity. Oral administration of either isomer at high doses (5,000 mg/kg body weight) does not cause any mortality. Therefore, the oral LD50s are greater than 5,000 mg/kg body weight. Both isomers also have very low acute inhalation toxicity as measured by the concentration that causes 50% mortality in experimental animals, the LC50. The 4-hour

exposure LC50s for both isomers are approximately 37,000 ppm in rats. Anesthetic-like effects are observed in rats at high inhalation concentration (greater than 5,000 ppm).

In 28-day inhalation studies with rats, the activity and responsiveness of the animals was reduced at exposures of 5,000 ppm or greater for each isomer. Toxicity was otherwise confined to the liver; liver enlargement and induction of peroxisomes was seen following treatment with either of the isomers. HCFC-225ca was more potent than HCFC-225cb in eliciting these liver effects. To investigate the biological relevance of the liver toxicity to humans, comparative repeated inhalation studies with HCFC-225ca and HCFC-225cb, have been conducted with rats, hamsters, guinea pigs and marmosets. In 14- day exposure studies with rats, hamsters and guinea pigs, the liver effects were also observed in rodents, while no such effects were observed in guinea pigs. In the 28-day study with marmosets, exposure to HCFC-225ca at 1,000 ppm caused effects on the liver, such as slight fat deposition associated with changes in serum biochemical parameters. In the same study, exposure to HCFC-225cb at 5,000 ppm caused somnolence during exposure and an increase of cytochrome P-450, indicative of an adaptive response to HCFC-225cb. However, no liver enlargement was seen and virtually no peroxisomal induction was observed with either isomer. Neither isomer causes eye irritation nor dermal toxicity in standardized tests; skin application of both isomers at high doses (2,000 mg/kg body weight) produces no adverse effects.

Pharmacokinetic studies with rats indicated that either isomer found in blood is rapidly eliminated on termination of exposure. No data exist on the oral and dermal toxicity of dichlorotrifloroethane (HCFC-123) in humans. Studies in animals show that HCFC-123 has low acute oral toxicity (ALD of approximately 9000 mg/kg in rats) and low dermal toxicity (LD50 > 2000 mg/kg in rats and rabbits). In rats and hamsters, the acute inhalation LC50 (four hour) for HCFC-123 is low, 28,000->53,000 ppm (175->330 mg/L).

In a single acute inhalation study carried out in guinea pigs, hepatotoxicity was seen at the lowest test level of 1000 ppm (6.25 mg/L) HCFC-123. Similar lesions were described in the same study with the HCFC-123 analogue, halothane. Such lesions were reported as reversible (by one-week post-exposure) in other studies on halothane exposed guinea pigs. Halothane is associated with both fatal (rare) and non-fatal hepatitis in humans. Similarities in metabolism, immunotoxicology and hepatic lesions between halothane and HCFC-123 in rats and guinea pigs support the possibility that acute exposure to high levels of HCFC-123 may elicit a similar toxicological profile to halothane in humans.

Acute reversible CNS effects have been reported in humans and animals following inhalation of HCFC-123. Exposure levels were not categorized in cases of human poisoning. No CNS effects were seen at 2500 ppm (15.6 mg/L) HCFC-123 in a behavioral study in rats. CFCs and HCFCs are known to sensitize the heart to adrenalin-induced arrhythmias. HCFC-123 caused cardiac sensitization in dogs exposed to levels around 20,000 ppm (125 mg/L), whereas no effects were seen at 10,000 ppm (62.5 mg/L). Although no data were available on cardiac sensitization effects for HCFC-123 in humans, such effects have been documented following exposure to other CFCs, including CFC-12, where sensitization was reported at 10,000 ppm. In humans, liver toxicity, cardiac sensitization and CNS depression are likely to be the critical effects following acute exposure to HCFC-123, although asphyxiation may also occur at very high levels. Tests in rabbits and guinea pigs indicate that HCFC-123 is not a skin irritant. HCFC-123 was a slight to moderate eye irritant in rabbits.

Overexposure by eye contact may include eye irritation with discomfort, tearing, or blurring of vision HCFC-123 is not a skin sensitizer. A study on skin sensitization of HCFC-123, carried out in guinea pigs, was considered negative under the conditions of the study. It is possible that the doses used may not have been sufficiently high to elicit a sensitization response. However, sensitization has not been reported in other structural analogues of HCFC-123. There are no reports of adverse effects in humans following repeated or prolonged exposure to HCFC-123. In humans, repeated exposure to other CFCs and HCFCs have been associated with hematological effects, neurological disorders, liver damage, reproductive effects, and coronary heart disease. Although behavioral effects and CNS effects have been seen in animals repeatedly exposed to HCFC-123, histological examination in rats of brain, spinal cord and nerve fibers indicates no neurotoxicity at the highest exposure (inhalation) level of 5000 ppm.

Human liver toxicity has been well documented for structural analogues of HCFC-123 including halothane, which has a similar metabolic, immunological, and hepatotoxic profile to HCFC-123 in animal studies. Adverse hepatic effects were seen in rats, guineapigs and dogs following repeated exposure (inhalation) to HCFC-123. The types of lesions observed varied between species and with duration of study. Generally, the lesions observed were hepatocyte enlargement and vacuolation (at 300 ppm) with necrosis and fatty change (at and above 1000 ppm). Such lesions were reported as reversible (30 days post-exposure) in a single 90-day study in rats exposed to 500?5000 ppm HCFC-123 and were not significantly increased at 300 ppm after 12 months in the two-year inhalation study. The NOAEL reported for hepatic effects in rats (28 weeks exposure in a two-generation reproductive toxicity study) was 100 ppm (0.63 mg/L).

Adverse testicular effects were seen in sub-acute inhalation studies in rats (NOAEL = 10,000 ppm) but not in guinea pigs. The LOAEL determined from chronic exposure (inhalation) in rats is 300 ppm (1.9 mg/L). A statistically significant decrease in insulin levels was seen in a sub-acute study in rats exposed to approximately 18,000 ppm HCFC-123. This finding was considered to be a physiological response to decreased glucose levels rather than an indicator of diminished pancreatic function, a finding supported by data from a 90-day study indicating a non-statistical/biological change in rat insulin levels.74 No pancreatic effects were seen in sub-acute inhalation studies in rats or guinea pigs, although pathological lesions were seen in rats exposed (oral) to HCFC-123a, the major impurity in HCFC-123. The NOAEL determined from chronic exposure (inhalation) in rats is 300 ppm (1.9 mg/L).

In rats, exposure (inhalation) to HCFC-123 did not influence pre-mating interval, copulation index, pregnancy rate or pup sex ratio of the F0 and F1 generations but was associated with decreased implantation sites among F1 females at 1000 ppm, a level at which overt maternotoxicity was observed. Adverse effects on reproductive tissues, such as testicular Leydig (interstitial) cells have been seen in repeated dose studies at and above 300 ppm HCFC-12350 although no histopathological effects on reproductive tissues were seen at 1000 ppm HCFC-123 after weeks in a two-generation reprotoxicity study.

Perturbations in serum sex hormone levels have also been reported in male rats and guinea pigs. Effects on progesterone (F1 generation only) and luteinizing hormone (F0 generation only) levels were seen in male rats at 100 ppm and 300 ppm respectively, with a NOAEL of 30 ppm. As these effects were not consistent between generations, biological significance was considered questionable.

In rabbits, developmental effects (increased resorptions and foetal defects) were seen only at doses which caused maternotoxicity, that is, greater than 10,000 ppm. In rats, HCFC-123 caused reduced pup growth in the offspring of the F1 generation at and above 30 ppm, and the F0 generation, at and above 100 ppm. Sexual maturation was also slightly delayed in F1 males (F0 offspring) at and above 300 ppm. However, the group mean body weight at attainment of sexual maturity was similar to controls, suggesting differences in pup growth rates may account for this delay. Reduced pup growth was not considered to be a

developmental effect as significant reduction in pup weight was not seen until seven to 14 days after birth. This effect may however be caused by HCFC-123 in breast milk (a lactational effect) as: the onset of reduced pup growth occurred during the period when exposure to HCFC-123 was restricted to parent dams; indicators of the integrity (quantity and quality) of milk, for example, CCK and milk fat, were normal during the suckling period; and maternal food intake during lactation was only decreased at and above 300 ppm HCFC-123. The genotoxic potential of HCFC-123 has been studied in a number of in vitro and in vivo bioassays. Most of these studies were designed to evaluate the genotoxic effects from exposure to HCFC-123 vapor. HCFC-123 showed no evidence of mutagenicity with in vitro bacteria or yeast tests and in vivo mouse micronucleus test and showed no evidence of inducing primary DNA damage by unscheduled DNA synthesis or cell transformation. Evidence for clastogenicity, from in vitro and in vivo lymphocyte studies was conflicting. No data exist for carcinogenicity in humans following exposure to HCFC-123. Although other structural analogues of HCFC-123 have been shown to cause tumors in animal studies, inadequate evidence exists for carcinogenicity in humans from epidemiological studies.

Several genetic studies have also been completed with both isomers of HCFC-225. These studies included an Ames assay, in vitro chromosomal aberration with Chinese Hamster Lung (CHL) and human lymphocyte, and in vivo unscheduled DNA synthesis assay. Based on the weight of evidence from all in vitro and in vivo studies, neither isomer is mutagenic. In only one study, which utilized an in vitro culture of human lymphocyte, did HCFC-225ca cause changes in the genetic materials while HCFC-225cb elicited a marginal response. However, the overall evidence from these studies implies that neither isomer is genotoxic. Chronic exposure to HCFC-123 elicited benign tumors (liver, pancreas, and testes) in rats at and above 300 ppm (1.9 mg/L). As the available data indicate HCFC-123 is non-genotoxic, data relevant to characterizing the mechanism for tumorigenicity in animals was reviewed in order to assess its relevance to humans.

Two types of hepatic tumors were observed in the two-year inhalation study in rats- hepatocellular adenomas and cholangiofibromas. HCFC-123, its major metabolite TFA and main impurity HCFC-123a have all been demonstrated to induce hepatic peroxisome proliferation as such, this mechanism has been proposed as the primary mechanism for hepatocellular tumor induction seen in rats exposed to HCFC-123.

Evidence indicates that this mechanism is species-specific: primates (including humans) and guinea pigs are not susceptible (in terms of peroxisome induction) to peroxisome proliferating substances. As such, it has been proposed that peroxisome proliferators are unlikely to present a hepatocarcinogenic hazard to humans. Despite dose-related increases seen in hepatic peroxisome proliferation in sub-acute, sub-chronic and chronic studies, the existence of anomalies serves to question whether this mechanism per se fully accounts for the observed liver effects elicited by HCFC-123.

Firstly, in the two-year study a significant increase in liver adenomas was seen in female rats exposed to 300 ppm HCFC-123 without a concomitant increase in peroxisome proliferation at this exposure level.50 However, a significant increase in peroxisome proliferation was seen at this concentration in female rats in a 90-day study by the same laboratory and as such this anomaly was considered by the study author to represent a biological variation in beta-oxidation potential. In addition, despite a dose related (significant) increase in peroxisome proliferation in male rats (in the two-year study) at 300 ppm and 1000 ppm, no increase was seen in liver adenomas at these exposure levels.

Secondly, HCFC-123 induced hepatic cell proliferation (CPI*), and decreased serum cholesterol and triglycerides in guinea pigs, despite the lack of peroxisome proliferation potential seen in this species. Of these effects, only triglyceride perturbations were statistically significant. However, increases in CPI were comparable to increases in rats. In addition, hepatocellular lesions (fatty change and necrosis) were also seen in HCFC-123 exposed guinea pigs, although their relevance to potential neoplastic lesions is purely speculative.

Finally, HCFC-123 has a similar metabolic profile to halothane with respect to TFA formation, beta-oxidation potential and effects on serum lipids. However, halothane did not induce tumours98 in either rats or mice. This finding should not be regarded as strong evidence of a non-peroxisomal mechanism for HCFC-123 as some peroxisome proliferators are more potent carcinogens than others, despite inducing similar levels of peroxisome proliferation, and only limited data on carcinogenicity for halothane were available. Although it is considered likely that the benign hepatocellular adenomas seen in rats exposed to HCFC-123 are related to increases in hepatic peroxisome proliferation (a mechanism believed not to present a hepatocarcinogenic hazard to humans), anomalies exist with respect to this proposed mechanism, mainly due to the lack of concordance of tumor incidence with liver beta-oxidation activity at certain exposure levels.

The mechanistic significance of benign hepatocholangiofibromas in female rats is unclear as this tumor type is not usually associated with peroxisome proliferation or hormone perturbation. However, its biological significance is confirmed by pre-neoplastic lesions (cholangiofibrosis) seen at 12 months in the same study. There is limited evidence from animal studies to suggest that this tumor type might only be relevant at high dose/exposure levels and statistical Interpretation of the data support a threshold for effect (1000-5000 ppm). Despite limited epidemiological evidence to suggest that the proposed hormonal mechanism (CCK stimulation of pancreas growth) is of questionable relevance for human pancreatic cancers and despite the fact that acinar cell cancers are not common in humans (by far the greatest number of human pancreatic tumors are of the ductal type), it must be assumed that, until more is known about the mechanism for acinar cell tumor induction in animals and humans (particularly the role of CCK), the pancreatic adenomas found in rats may have some predictive value for human carcinogenicity.

Benign Leydig cell (interstitial cell) adenomas are common in aging rats and strongly associated with senile endocrine disturbances. In contrast to the rat, Leydig cell tumors in men are extremely rare, representing less than three per cent of all testicular neoplasms. The rarity of this tumor type in humans as compared to its high spontaneous and chemically induced incidence in rodents, in addition to recent evidence indicating that endocrine disturbances and testicular tumors seen in animals may be linked to hepatic peroxisome proliferation, serves to question the relevance of HCFC-123-induced Leydig cell adenomas in humans. For all three tissues in which tumors occur, the cell type (except cholangiocellular tissue) has been a site of tumorigenic activity for other peroxisome proliferators, including hypolipidaemic drugs. As this triad of tumor types have not been reported in epidemiological data on hypolipidaemic drugs (classic peroxisome proliferating substances), it has been hypothesized that hepatic, testicular and pancreatic tumors seen in rodents are not relevant to humans. However, such a conclusion should be viewed with caution as epidemiological data on hypolipidaemic drugs only exist for clofibrate and fenofibrate, neither of which produce testicular or pancreatic tumors in animal studies. In addition, such studies are considered inconclusive due to the short period of exposure and follow-up.

Overall, indications are that the primary mechanism(s) of tumorigenicity for HCFC-123 is non-genotoxic (epigenic) and that hormonal perturbations and peroxisome proliferation may be involved to some degree. In fact, these mechanisms may be interrelated as recent research indicates a link with hepatic peroxisome proliferation and hormonal perturbations. In further support of such an association is the recent discovery of

an oestrogen-like hormone receptor in peroxisome mediated hepatic carcinogenicity.105 Such a mechanism might account for the sex differences and the lack of target organ specificity? with respect to HCFC-123 elicited tumors.

In summary, until further data become available regarding the mechanism of HCFC-123 induced tumors, particularly with respect to cholangiofibroma and pancreatic adenoma induction, it must be concluded that findings in rats may have some relevance for humans. Disinfection by products (DBPs) reformed when disinfectants such as chlorine, chloramine, and ozone react with organic and inorganic matter in water. The observations that some DBPs such as trihalomethanes (THMs), di-/trichloroacetic acids, and 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) are carcinogenic in animal studies have raised public concern over the possible adverse health effects of DBPs. To date, several hundred DBPs have been identified. Numerous haloalkanes and haloalkenes have been tested for carcinogenic and mutagenic activities. In general, the genotoxic potential is dependent on the nature, number, and position of halogen(s) and the molecular size of the compound. Short chain monohalogenated (excluding fluorine) alkanes and alkenes are potential direct-acting alkylating agents, particularly if the halogen is at the terminal end of the carbon chain or at an allylic position. Dihalogenated alkanes are also potential alkylating or cross-linking agents (either directly or after GSH conjugation), particularly if they are vicinally substituted (e.g., 1,2-dihaloalkane) or substituted at the two terminal ends of a short to medium-size (e.g., 2-7) alkyl moiety (i.e., alpha, omega-dihaloalkane). Fully halogenated haloalkanes tend to act by free radical or nongenotoxic mechanisms (such as generating peroxisome-proliferative intermediates) or undergo reductive dehalogenation to yield haloalkenes that in turn could be activated to epoxides.

Haloalkenes are of concern because of potential to generate genotoxic intermediates after epoxidation. The concern for haloalkenes may be diminished if the double bond is internal or sterically hindered. The cancer concern levels of the 14 haloalkanes and haloalkenes, have been rated based on available screening cancer bioassay (pulmonary adenoma assay) and genotoxicity data. Five brominated and iodinated methane and ethane derivatives are given a moderate rating. Beyond the fact that bromine and iodine are better leaving groups than chlorine, there is also evidence that brominated THMs may be preferentially activated by a theta-class glutathione S-transferase (GSTT1-1) to mutagens in Salmonella even at low substrate concentrations Furthermore, there are human carcinogenicity implications because of polymorphism in GSTT1-1. Human subpopulations with expressed GSTT1-1 may be at a greater risk to brominate THMs than humans who lack the gene. Six, two, and one haloalkanes/ haloalkene(s) are given low-moderate, marginal, and low concern, respectively.

The acute toxicity of dichlorofluoroethane (HCFC 141b) is low. No mortality was observed in rats receiving oral doses of 5,000 mg/kg. Dermal exposure of rats or rabbits to 2,000 mg/kg caused no mortality and no signs of toxicity. Single exposures of mice for 30 minutes indicated that the LC50 was between 296,640 and 494,400 mg/m3 (61,800 ppm to 103,000 ppm) and the 4-hr LC50 in rats was 62,000 ppm (approximately 297,600 mg/m3). Also, a 6-hr exposure of mice at 41,000 ppm (approximately 196,800 mg/m3) caused narcosis but not lethality. In a controlled exposure study, exposure of humans to levels up to 1,000 ppm (4800 mg/m3) for periods of 3 or 4 hours produced no reports of any adverse effects. HCFC 141b is considered nonirritating to rabbits' skin and a mild eye irritant. A skin sensitization test in guinea pigs was negative. In repeat inhalation exposure studies of 6 hr/d, 5d/wk for periods from 2 to 13 weeks, the NOEL was judged to be 8,000 ppm (approximately 38,400 mg/m3). The next highest exposure level, 20,000 ppm (96,000 mg/m3), induced only reduced bodyweight gain and slightly increased levels of cholesterol, triglycerides, and glucose. No treatment-related hematological or histopathological changes were noted in any exposure level group. There was no evidence of teratogenic or embryotoxic effects in pregnant rabbits exposed to 1,400, 4,200 or 12,600 ppm (6720 mg/m3, 20,000 mg/m3, and 60,480 mg/m3, respectively) or in pregnant rats exposed to 3,200 or 7,900 ppm (15,360 or 38,000 mg/m3) of HCFC 141b although signs of maternal toxicity were observed at and above 3,200 ppm (15,360 mg/m3) in rats and 4,200 ppm (20,000 mg/m3) in rabbits. A two- generation inhalation study in rats demonstrated a NOEL of 8,000 ppm (38,400 mg/m3) for reproductive parameters. At a higher concentration, 20,000 ppm (96,000 mg/m3) a non-reproducible decrease in the number of litters, in the number of pups per litter and also some retardation of sexual maturation of male pups, which may have been caused by the slight body weight growth retardation, was observed. In "in vitro" studies, negative results were obtained in bacterial reverse mutation assay and both negative and positive results were obtained in cytogenetic assays. In vivo, negative results were obtained in two mouse micronucleus assays. Consequently, the data indicates that the genotoxicity occasionally observed "in vitro" is not expressed "in vivo." Rats were exposed by inhalation in a lifetime study to concentrations of 1,500, 5,000 and 20,000 ppm (7200; 24,000; and 96,000 mg/m3, respectively). No significant evidence of toxicity was seen, however, at the highest exposure concentration reduced body weight gain was observed.

HCFC 141b did not produce neoplastic changes in female rats at any test concentration. In male rats no neoplastic changes were noted at 1,500 ppm but increased incidences of testicular interstitial cell (Leydig cells) hyperplasia and adenoma were observed at 5,000 ppm (24,000 mg/m3) and 20,000 ppm (96,000 mg/m3). These changes appeared late in life and were not correlated with increased mortality. Because of the genotoxicity profile of HCFC 141b these effects on the rat Leydig cells are considered as to be of epigenetic origin and associated with senile endocrine disturbances, and therefore of no relevance to tumourigenic hazard for man.

For HCFC 141b: The low octanol/water partition coefficient (log Pow = 2.3) indicates a low potential for bioaccumulation. HCFC 141b is not readily biodegradable. The predominant degradation of HCFC 141b will occur in the air, but at a very slow rate. Acute ecotoxicity studies are available for algae, daphnia, and fish. The 96-hr LC50 for zebra fish was 126 mg/L and the 48-hr EC50 for daphnia was 31.2 mg/L. The 72-hr NOEC for both growth rate and biomass for algae was > 44 mg/L. Applying an uncertainty factor of 100 to the 48-hr EC50 value of 31.2 mg/L for daphnia, a PNEC of 0.31 mg/L was derived.

Acute Toxicity	✓	Carcinogenicity	✗
Skin Irritation/Corrosion	✗	Reproductivity	✗
Serious Eye Damage/Irritation	✗	STOT - Single Exposure	✗
Respiratory or Skin Sensitization	✗	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

ShieldFoam PLF Part A	**ENDPOINT**	**TEST DURATION (HR)**	**SPECIES**	**VALUE**	**SOURCE**
	Not Available	Not Available	Not Available	Not Available	Not Available
Polyethylene/ Polypropylene Glycol Glyceryl Ether	**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**
	LC50	96	Fish	8.900mg/L	3
	EC50	48	Crustacea	63mg/L	2
	EC50	96	Algae or other aquatic plants	1.363mg/L	3
	NOEC	96	Algae or other aquatic plants	6mg/L	1
1,1-dichloro-1-fluoroethane	**ENDPOINT**	**TEST DURATION (HR)**	**SPECIES**	**VALUE**	**SOURCE**
	LC50	96	Fish	10.664mg/L	3
	EC50	48	Crustacea	31mg/L	5
	EC50	72	Algae or other aquatic plants	ca.24mg/L	2
	NOEC	72	Algae or other aquatic plants	ca.7mg/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

DO NOT discharge into sewer or waterways.

Persistence and Degradability:

Ingredient	Persistence: Water/Soil	Persistence: Air
tris(2-chloroisopropyl) phosphate	High	High
1,1-dichloro-1-fluoroethane	High	High

Bioaccumulative Potential:

Ingredient	Bioaccumulation
tris(2-chloroisopropyl) phosphate	LOW (BCF = 4.6)
1,1-dichloro-1-fluoroethane	LOW (LogKOW = 2.3659)

Mobility in Soil:

Ingredient	Mobility
tris(2-chloroisopropyl) phosphate	LOW (KOC = 1278)
1,1-dichloro-1-fluoroethane	LOW (KOC = 48.64)

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.
Recycle wherever possible or consult manufacturer for recycling options.
Consult State Land Waste Authority for disposal.
Bury or incinerate residue at an approved site.
Recycle containers if possible or dispose of in an authorized landfill.

SECTION 14: TRANSPORT INFORMATION

Labels Required

Marine Pollutant: No
HAZCHEM: Not Applicable

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

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

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

Transport in bulk according to Annex II of MARPOL and the IBC code: Not Applicable

SECTION 15: REGULATORY INFORMATION

Safety, Health, and Environmental regulations / legislation specific for the Substance or Mixture

Polyethylene/ Polypropylene Glycol Glyceryl Ether is found on the following regulatory lists:

- Australia Inventory of Chemical Substances (AICS)
- GESAMP/EHS Composite List - GESAMP Hazard Profiles
- IMO IBC Code Chapter 17: Summary of minimum requirements

Tris(2-chloroisopropyl) Phosphate is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Inventory of Chemical Substances (AICS)

1,1-dichloro-1-fluoroethane is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Inventory of Chemical Substances (AICS)
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
- International Air Transport Association (IATA) Dangerous Goods Regulations

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (1,1-dichloro-1-fluoroethane; polyethylene/ polypropylene glycol glyceryl ether; tris(2-chloroisopropyl) phosphate)
China – IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (polyethylene/ polypropylene glycol glyceryl ether)
Japan – ENCS	No (polyethylene/ polypropylene glycol glyceryl ether)
Korea – KECI	Yes
New Zealand – NZIoC	Yes
Philippines – PICCS	Yes

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

SECTION 16: OTHER INFORMATION

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the ShieldCrete® International 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
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