



# PADMANAB MAFATLAL GROUP

## CHLORODIFLUOROMETHANE

Navin Fluorine International Limited

Chemwatch Hazard Alert Code: 1

Chemwatch: 1018  
Version No: 9.1  
Safety Data Sheet

Issue Date: 02/06/2023  
Print Date: 08/04/2024  
L.GHS.IND.EN

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

#### Product Identifier

Product name	CHLORODIFLUOROMETHANE
Chemical Name	Not Available
Synonyms	CHClF <sub>2</sub> ; CH-Cl-F <sub>2</sub> ; difluorochloromethane 22; difluoromonochloromethane; monochlorodifluoromethane; Algofrene Type 6; Amerfrost A-22; Propellant 22; Arcton 4; Refrigerant 22; Ucon 22; Electro-cl 22; Hhalocarbon 22; Eskimon 22; Freon 22 F-22; HCFC22; Fluorocarbon 22 F; Frigen; Genetron; Isceon 22; Isotron 22; Airco; R22
Proper shipping name	CHLORODIFLUOROMETHANE (REFRIGERANT GAS R 22)
Chemical formula	CHClF <sub>2</sub>
Other means of identification	Not Available
CAS number	75-45-6

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

Relevant identified uses	Refrigerant at low temperatures; in airconditioning; as aerosol propellant; low temperature solvent; manufacture of fluorocarbon plastics. HCFCs are used in a variety of applications because of their low toxicity, reactivity and flammability. Every permutation of fluorine, chlorine and hydrogen based on methane and ethane has been examined and most have been commercialized. Furthermore, many examples are known for higher numbers of carbon as well as related compounds containing bromine. Uses include refrigerants, blowing agents, propellants in medicinal applications and degreasing solvents. The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation.
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#### Details of the manufacturer or supplier of the safety data sheet

Registered company name	Navin Fluorine International Limited
Address	Surat-Navsari- Road, PO : Bhestan, near Udhna, SURAT Gujarat 395 023 India
Telephone	+91-261- 6715 340
Fax	+91-22-66509800
Website	<a href="http://www.nfil.in">www.nfil.in</a>
Email	subodh.kumar@nfil.in

#### Emergency telephone number

Association / Organisation	+91-261- 6715 301	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone numbers	+91-261- 6715 301	+918000403230
Other emergency telephone numbers	+91-9714755221	+61 3 9573 3188

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

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

##### Chemwatch Hazard Ratings


	Min	Max
Flammability	0	
Toxicity	1	
Body Contact	1	
Reactivity	1	
Chronic	1	

0 = Minimum  
1 = Low  
2 = Moderate  
3 = High  
4 = Extreme

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<b>Classification</b>	Gases Under Pressure (Compressed Gas), Skin Corrosion/Irritation Category 3, Serious Eye Damage/Eye Irritation Category 2B, Acute Toxicity (Inhalation) Category 5, Hazardous to the Ozone Layer Category 1
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### Label elements

<b>Hazard pictogram(s)</b>	
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<b>Signal word</b>	<b>Warning</b>
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### Hazard statement(s)

<b>H280</b>	Contains gas under pressure; may explode if heated.
<b>H316</b>	Causes mild skin irritation.
<b>H320</b>	Causes eye irritation.
<b>H333</b>	May be harmful if inhaled.
<b>H420</b>	Harms public health and the environment by destroying ozone in the upper atmosphere.

### Precautionary statement(s) Prevention

<b>P264</b>	Wash all exposed external body areas thoroughly after handling.
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### Precautionary statement(s) Response

<b>P304+P312</b>	IF INHALED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
<b>P305+P351+P338</b>	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
<b>P332+P313</b>	If skin irritation occurs: Get medical advice/attention.
<b>P337+P313</b>	If eye irritation persists: Get medical advice/attention.

### Precautionary statement(s) Storage

<b>P410+P403</b>	Protect from sunlight. Store in a well-ventilated place.
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### Precautionary statement(s) Disposal

<b>P502</b>	Refer to manufacturer or supplier for information on recovery or recycling.
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## SECTION 3 Composition / information on ingredients

### Substances

CAS No	%[weight]	Name
75-45-6	>99	<u>chlorodifluoromethane</u>

### Mixtures

See section above for composition of Substances

## SECTION 4 First aid measures

### Description of first aid measures

<b>Eye Contact</b>	<ul style="list-style-type: none"> <li>▶ If product comes in contact with eyes remove the patient from gas source or contaminated area.</li> <li>▶ Take the patient to the nearest eye wash, shower or other source of clean water.</li> <li>▶ Open the eyelid(s) wide to allow the material to evaporate.</li> <li>▶ Gently rinse the affected eye(s) with clean, cool water for at least 15 minutes. Have the patient lie or sit down and tilt the head back. Hold the eyelid(s) open and pour water slowly over the eyeball(s) at the inner corners, letting the water run out of the outer corners.</li> <li>▶ The patient may be in great pain and wish to keep the eyes closed. It is important that the material is rinsed from the eyes to prevent further damage.</li> <li>▶ Ensure that the patient looks up, and side to side as the eye is rinsed in order to better reach all parts of the eye(s)</li> <li>▶ Transport to hospital or doctor.</li> <li>▶ Even when no pain persists and vision is good, a doctor should examine the eye as delayed damage may occur.</li> <li>▶ If the patient cannot tolerate light, protect the eyes with a clean, loosely tied bandage.</li> <li>▶ Ensure verbal communication and physical contact with the patient.</li> </ul> <p><b>DO NOT</b> allow the patient to rub the eyes  <b>DO NOT</b> allow the patient to tightly shut the eyes  <b>DO NOT</b> introduce oil or ointment into the eye(s) without medical advice  <b>DO NOT</b> use hot or tepid water.</p>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ Following exposure to gas, remove the patient from the gas source or contaminated area.</li> <li>▶ NOTE: Personal Protective Equipment (PPE), including positive pressure self-contained breathing apparatus may be required to assure the safety of the rescuer.</li> <li>▶ Prostheses such as false teeth, which may block the airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ If the patient is not breathing spontaneously, administer rescue breathing.</li> <li>▶ If the patient does not have a pulse, administer CPR.</li> <li>▶ If medical oxygen and appropriately trained personnel are available, administer 100% oxygen.</li> <li>▶ Summon an emergency ambulance. If an ambulance is not available, contact a physician, hospital, or Poison Control Centre for further instruction.</li> <li>▶ Keep the patient warm, comfortable and at rest while awaiting medical care.</li> </ul>

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	<ul style="list-style-type: none"> <li>▶ <b>MONITOR THE BREATHING AND PULSE, CONTINUOUSLY.</b></li> <li>▶ Administer rescue breathing (preferably with a demand-valve resuscitator, bag-valve mask-device, or pocket mask as trained) or CPR if necessary.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ Not considered a normal route of entry.</li> <li>▶ For advice, contact a Poisons Information Centre or a doctor.</li> <li>▶ Avoid giving milk or oils.</li> <li>▶ Avoid giving alcohol.</li> </ul>

**Indication of any immediate medical attention and special treatment needed**

for intoxication due to Freons/ Halons:

A: Emergency and Supportive Measures

- ▶ Maintain an open airway and assist ventilation if necessary
- ▶ Treat coma and arrhythmias if they occur. Avoid (adrenaline) epinephrine or other sympathomimetic amines that may precipitate ventricular arrhythmias. Tachyarrhythmias caused by increased myocardial sensitisation may be treated with propranolol, 1-2 mg IV or esmolol 25-100 microgm/kg/min IV.
- ▶ Monitor the ECG for 4-6 hours

B: Specific drugs and antidotes:

- ▶ There is no specific antidote

C: Decontamination

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

D: Enhanced elimination:

- ▶ There is no documented efficacy for diuresis, haemodialysis, haemoperfusion, or repeat-dose charcoal.

*POISONING and DRUG OVERDOSE, Californian Poison Control System Ed. Kent R Olson; 3rd Edition*

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

For gas exposures:

**BASIC TREATMENT**

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

**ADVANCED TREATMENT**

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

BRONSTEIN, A.C. and CURRRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

**SECTION 5 Firefighting measures****Extinguishing media**

**SMALL FIRE:** Use extinguishing agent suitable for type of surrounding fire.

**LARGE FIRE:** Cool cylinder.

**DO NOT** direct water at source of leak or venting safety devices as icing may occur.

**Special hazards arising from the substrate or mixture****Fire Incompatibility**

- ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

**Advice for firefighters****Fire Fighting****GENERAL**

- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus and protective gloves.
- ▶ Fight fire from a safe distance, with adequate cover.
- ▶ Use water delivered as a fine spray to control fire and cool adjacent area.
- ▶ **DO NOT** approach cylinders suspected to be hot.
- ▶ Cool fire exposed cylinders with water spray from a protected location.
- ▶ If safe to do so, remove cylinders from path of fire.

**SPECIAL REQUIREMENTS:**

- ▶ Excessive pressures may develop in a gas cylinder exposed in a fire; this may result in explosion.
- ▶ Cylinders with pressure relief devices may release their contents as a result of fire and the released gas may constitute a further source of hazard for the fire-fighter.
- ▶ Cylinders without pressure-relief valves have no provision for controlled release and are therefore more likely to explode if exposed to fire.

**FIRE FIGHTING REQUIREMENTS:**

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	The need for proximity, entry and special protective clothing should be determined for each incident, by a competent fire-fighting safety professional.
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Containers may explode when heated - Ruptured cylinders may rocket</li> <li>▶ Fire exposed containers may vent contents through pressure relief devices.</li> <li>▶ High concentrations of gas may cause asphyxiation without warning.</li> <li>▶ May decompose explosively when heated or involved in fire.</li> <li>▶ Contact with gas may cause burns, severe injury and/ or frostbite.</li> </ul> <p>Decomposition may produce toxic fumes of: carbon monoxide (CO)</p> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) hydrogen chloride phosgene hydrogen fluoride other pyrolysis products typical of burning organic material.</p> <p><b>Contains low boiling substance:</b> Closed containers may rupture due to pressure buildup under fire conditions.</p> <ul style="list-style-type: none"> <li>▶ Non combustible.</li> <li>▶ Not considered to be a significant fire risk.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ May emit corrosive, poisonous fumes.</li> <li>▶ Vented gas is more dense than air and may collect in pits, basements.</li> </ul>

### 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

<b>Minor Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Avoid breathing vapour and any contact with liquid or gas. Protective equipment including respirator should be used.</li> <li>▶ <b>DO NOT enter confined spaces where gas may have accumulated.</b></li> <li>▶ Increase ventilation.</li> <li>▶ Clear area of personnel.</li> <li>▶ Stop leak only if safe to do so.</li> <li>▶ Remove leaking cylinders to safe place. Release pressure under safe controlled conditions by opening valve.</li> <li>▶ Do not exert excessive pressure on the valve; do not attempt to operate a damaged valve</li> <li>▶ Orientate cylinder so that the leak is gas, not liquid, to minimise rate of leakage</li> <li>▶ Keep area clear of personnel until gas has dispersed.</li> </ul>
<b>Major Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Clear area of all unprotected personnel and move upwind.</li> <li>▶ Alert Emergency Authority and advise them of the location and nature of hazard.</li> <li>▶ Wear breathing apparatus and protective gloves.</li> <li>▶ Prevent by any means available, spillage from entering drains and water-courses.</li> <li>▶ Consider evacuation.</li> <li>▶ Increase ventilation.</li> <li>▶ No smoking or naked lights within area.</li> <li>▶ Stop leak only if safe to do so.</li> <li>▶ Water spray or fog may be used to disperse vapour.</li> <li>▶ <b>DO NOT enter confined space where gas may have collected.</b></li> <li>▶ Keep area clear until gas has dispersed.</li> <li>▶ Remove leaking cylinders to a safe place.</li> <li>▶ Fit vent pipes. Release pressure under safe, controlled conditions</li> <li>▶ Burn issuing gas at vent pipes.</li> <li>▶ DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve.</li> </ul>

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

### SECTION 7 Handling and storage

#### Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>· Consider use in closed pressurised systems, fitted with temperature, pressure and safety relief valves which are vented for safe dispersal. Use only properly specified equipment which is suitable for this product, its supply pressure and temperature</li> <li>· The tubing network design connecting gas cylinders to the delivery system should include appropriate pressure indicators and vacuum or suction lines.</li> <li>· Fully-welded types of pressure gauges, where the bourdon tube sensing element is welded to the gauge body, are recommended.</li> <li>· Before connecting gas cylinders, ensure manifold is mechanically secure and does not contain another gas. Before disconnecting gas cylinder, isolate supply line segment proximal to cylinder, remove trapped gas in supply line with aid of vacuum pump</li> <li>· When connecting or replacing cylinders take care to avoid airborne particulates violently ejected when system pressurises.</li> <li>· Consider the use of doubly-contained piping; diaphragm or bellows sealed, soft seat valves; backflow prevention devices; flash arrestors; and flow monitoring or limiting devices. Gas cabinets, with appropriate exhaust treatment, are recommended, as is automatic monitoring of the secondary enclosures and work areas for release.</li> <li>· Use a pressure reducing regulator when connecting cylinder to lower pressure (&lt;100 psig) piping or systems</li> <li>· Use a check valve or trap in the discharge line to prevent hazardous back-flow into the cylinder</li> <li>· Check regularly for spills or leaks. Keep valves tightly closed but do not apply extra leverage to hand wheels or cylinder keys.</li> <li>· Open valve slowly. If valve is resistant to opening then contact your supervisor</li> <li>· Valve protection caps must remain in place unless container is secured with valve outlet piped to use point.</li> <li>· Never insert a pointed object (e.g hooks) into cylinder cap openings as a means to open cap or move cylinder. Such action can inadvertently turn the valve and gas a gas leak. Use an adjustable strap instead of wrench to free an over-tight or rusted cap.</li> <li>· A bubble of gas may buildup behind the outlet dust cap during transportation, after prolonged storage, due to defective cylinder valve or if a dust cap is inserted without adequate evacuation of gas from the line. When loosening dust cap, preferably stand cylinder in a suitable enclosure and take cap off slowly. Never face the dust cap directly when removing it; point cap away from any personnel or any object that may pose a hazard. under negative pressure (relative to atmospheric gas)</li> <li>· Suck back of water into the container must be prevented. Do not allow backfeed into the container.</li> <li>· Do NOT drag, slide or roll cylinders - use a suitable hand truck for cylinder movement</li> <li>· Test for leakage with brush and detergent - <b>NEVER use a naked flame.</b></li> </ul>
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	<ul style="list-style-type: none"> <li>- Do NOT heat cylinder by any means to increase the discharge rate of product from cylinder.</li> <li>- Leaking gland nuts may be tightened if necessary.</li> <li>- If a cylinder valve will not close completely, remove the cylinder to a well ventilated location (e.g. outside) and, when empty, tag as FAULTY and return to supplier.</li> <li>- Obtain a work permit before attempting any repairs.</li> <li>- DO NOT attempt repair work on lines, vessels under pressure.</li> <li>- Atmospheres must be tested and O.K. before work resumes after leakage.             <ul style="list-style-type: none"> <li>▶ DO NOT transfer gas from one cylinder to another.</li> </ul> </li> </ul>
Other information	<ul style="list-style-type: none"> <li>- Do NOT store halogenated aliphatics in areas containing alkali or alkaline earth metals such as powdered aluminum, zinc, or beryllium</li> <li>▶ Cylinders should be stored in a purpose-built compound with good ventilation, preferably in the open.</li> <li>▶ Such compounds should be sited and built in accordance with statutory requirements.</li> <li>▶ The storage compound should be kept clear and access restricted to authorised personnel only.</li> <li>▶ Cylinders stored in the open should be protected against rust and extremes of weather.</li> <li>▶ Cylinders in storage should be properly secured to prevent toppling or rolling.</li> <li>▶ Cylinder valves should be closed when not in use.</li> <li>▶ Where cylinders are fitted with valve protection this should be in place and properly secured.</li> <li>▶ Gas cylinders should be segregated according to the requirements of the Dangerous Goods Act.</li> <li>▶ Preferably store full and empty cylinders separately.</li> <li>▶ Check storage areas for hazardous concentrations of gases prior to entry.</li> <li>▶ Full cylinders should be arranged so that the oldest stock is used first.</li> <li>▶ Cylinders in storage should be checked periodically for general condition and leakage.</li> <li>▶ Protect cylinders against physical damage. Move and store cylinders correctly as instructed for their manual handling.</li> </ul> <p><b>NOTE:</b> A 'G' size cylinder is usually too heavy for an inexperienced operator to raise or lower.</p>

## Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> <li>▶ DO NOT use aluminium or galvanised containers</li> <li>▶ Cylinder:             <ul style="list-style-type: none"> <li>▶ Ensure the use of equipment rated for cylinder pressure.</li> <li>▶ Ensure the use of compatible materials of construction.</li> <li>▶ Valve protection cap to be in place until cylinder is secured, connected.</li> <li>▶ Cylinder must be properly secured either in use or in storage.</li> <li>▶ Cylinder valve must be closed when not in use or when empty.</li> <li>▶ Segregate full from empty cylinders.</li> </ul> </li> </ul> <p><b>WARNING:</b> Suckback into cylinder may result in rupture. Use back-flow preventive device in piping.</p>
Storage incompatibility	<p>Haloalkanes:</p> <ul style="list-style-type: none"> <li>▶ are highly reactive: some of the more lightly substituted lower members are highly flammable; the more highly substituted may be used as fire suppressants, not always with the anticipated results.</li> <li>▶ may react with the lighter divalent metals to produce more reactive compounds analogous to Grignard reagents.</li> <li>▶ may produce explosive compounds following prolonged contact with metallic or other azides</li> <li>▶ may react on contact with potassium or its alloys - although apparently stable on contact with a wide range of halocarbons, reaction products may be shock-sensitive and may explode with great violence on light impact; severity generally increases with the degree of halocarbon substitution and potassium-sodium alloys give extremely sensitive mixtures .</li> </ul> <p>BREITHERICK L.: Handbook of Reactive Chemical Hazards</p> <ul style="list-style-type: none"> <li>▶ react with metal halides and active metals, eg. sodium (Na), potassium (K), lithium (Li), calcium (Ca), zinc (Zn), powdered aluminium (Al) and aluminium alloys, magnesium (Mg) and magnesium alloys.</li> <li>▶ may react with brass and steel.</li> <li>▶ may react explosively with strong oxidisers</li> <li>▶ may degrade rubber, and plastics such as methacrylate polymers, polyethylene and polystyrene, paint and coatings</li> </ul> <p>Chlorodifluoromethane:</p> <ul style="list-style-type: none"> <li>▶ mixtures with 50% air are combustible but difficult to ignite</li> <li>▶ contact with water causes slow decomposition</li> <li>▶ is incompatible with alkalis, alkali earth metals (e.g. aluminium powder, sodium, potassium, zinc), barium, beryllium, decaborane, difluoromethylene, dihypofluorite, fluorine, lithium, magnesium, potassium acetylene 1,2-dioxide, potassium sodium alloy, sodium amide, titanium, uranium hydride</li> <li>▶ may form explosive compounds with divalent light metals and metal azides</li> <li>▶ produces violent, self-sustaining reaction with aluminium, releasing high heat; the violence of the reaction may depend on a combination of two factors; vapour pressure and the degree of fluorination - the size of the aluminium particles may also be a factor</li> <li>▶ undergoes thermal decomposition when exposed to red hot surfaces or fire, forming chlorine, hydrogen fluoride, hydrogen chloride, phosgene, and carbonyl fluoride</li> <li>▶ may slowly decompose in the presence of rust and moisture, forming toxic gases</li> <li>▶ attacks some paints, rubbers, and coatings</li> <li>▶ attacks magnesium and its alloys, aluminium; corrosion can occur when magnesium alloys or aluminium containing more than 2% magnesium is used with fluorocarbon systems in which water may be present</li> <li>▶ CFCs may react with strong oxidising or reducing agents.</li> <li>▶ Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances</li> </ul>

## SECTION 8 Exposure controls / personal protection

## Control parameters

## Occupational Exposure Limits (OEL)

## INGREDIENT DATA

Not Available

## Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
chlorodifluoromethane	1,250 ppm	2,400 ppm	14,000 ppm
Ingredient	Original IDLH		Revised IDLH
chlorodifluoromethane	Not Available		Not Available

## MATERIAL DATA

for chlorodifluoromethane:

The recommended TLV-TWA should provide an ample margin of safety to prevent cardiac sensitisation and systemic injury.


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May act as a simple asphyxiants; these are gases which, when present in high concentrations, reduce the oxygen content in air below that required to support breathing, consciousness and life; loss of consciousness, with death by suffocation may rapidly occur in an oxygen deficient atmosphere.

**CARE:** Most simple asphyxiants are odourless or possess low odour and there is no warning on entry into an oxygen deficient atmosphere. If there is any doubt, oxygen content can be checked simply and quickly. It may not be appropriate to only recommend an exposure standard for simple asphyxiants rather it is essential that sufficient oxygen be maintained. Air normally has 21 percent oxygen by volume, with 18 percent regarded as minimum under normal atmospheric pressure to maintain consciousness / life. At pressures significantly higher or lower than normal atmospheric pressure, expert guidance should be sought.

**Exposure controls**

<p><b>Appropriate engineering controls</b></p>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. 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.</p> <ul style="list-style-type: none"> <li>▶ Areas where cylinders are stored require good ventilation and, if enclosed, need discrete/controlled exhaust ventilation.</li> <li>▶ Secondary containment and exhaust gas treatment may be required by certain jurisdictions.</li> <li>▶ Local exhaust ventilation may be required in work areas.</li> <li>▶ Consideration should be given to the use of diaphragm or bellows-sealed, soft-seat valves; backflow prevention devices and flow-monitoring or limiting devices.</li> <li>▶ Automated alerting systems with automatic shutdown of gas-flow may be appropriate and may in fact be mandatory in certain jurisdictions.</li> <li>▶ Respiratory protection in the form of air-supplied or self-contained breathing equipment must be worn if the oxygen concentration in the workplace air is less than 19%.</li> <li>▶ Cartridge respirators do NOT give protection and may result in rapid suffocation.</li> </ul> <p>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.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 50%;">Type of Contaminant:</td> <td style="width: 50%;">Air Speed:</td> </tr> <tr> <td>gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 50%;">Lower end of the range</th> <th style="width: 50%;">Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	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
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<p><b>Individual protection measures, such as personal protective equipment</b></p>															
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]</li> <li>▶ 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].</li> </ul>														
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>														
<p><b>Hands/feet protection</b></p>	<ul style="list-style-type: none"> <li>▶ Butyl rubber gloves             <ul style="list-style-type: none"> <li>· Butyl rubber gloves should be used when handling halogenated aliphatics .</li> <li>· Nitrile, PVC-coated nitrile, and PVC protective equipment are not recommended</li> </ul> </li> <li>▶ Neoprene gloves</li> <li>▶ When handling sealed and suitably insulated cylinders wear cloth or leather gloves.</li> </ul>														
<p><b>Body protection</b></p>	<p>See Other protection below</p>														
<p><b>Other protection</b></p>	<ul style="list-style-type: none"> <li>· Halogen-selective detectors use a specialized sensor that allows the monitor to detect compounds containing fluorine, chlorine, bromine, and iodine with-out interference from other species. These detectors are typically easy to use, feature higher sensitivity than the nonselective detectors (detection limits are typically &lt;5 ppm when used as an area monitor and &lt;1.4 gm/yr [&lt;0.05 oz/yr] when used as a leak pinpointer).</li> <li>· Compound-Specific Detectors are typically capable of detecting the presence of a single compound without interference from other compounds.</li> <li>▶ Protective overalls, closely fitted at neck and wrist.</li> <li>▶ Eye-wash unit.</li> <li>▶ Ensure availability of lifeline in confined spaces.</li> <li>▶ Staff should be trained in all aspects of rescue work.</li> <li>▶ Rescue gear: Two sets of SCBA breathing apparatus Rescue Harness, lines etc.</li> </ul>														

**Respiratory protection**

Type AX 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
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## CHLORODIFLUOROMETHANE

up to 10	1000	AX-AUS / Class1	-
up to 50	1000	-	AX-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	AX-2
up to 100	10000	-	AX-3
100+			Airline**

\* - Continuous Flow \*\* - Continuous-flow or positive pressure demand

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(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), 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

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

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

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- ▶ Positive pressure, full face, air-supplied breathing apparatus should be used for work in enclosed spaces if a leak is suspected or the primary containment is to be opened (e.g. for a cylinder change)
- ▶ Air-supplied breathing apparatus is required where release of gas from primary containment is either suspected or demonstrated.

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

<b>Appearance</b>	Colourless, practically odourless gas, which is readily liquefied under pressure and/or reduced temperature. Miscible with various hydrocarbons, chlorinated solvents, alcohols, ketones and esters; low solubility in water. Ozone depletion factor 0.05. The physical properties of HCFCs are tunable by changes in the number and identity of the halogen atoms. In general, they are volatile but less so than their parent alkanes. The decreased volatility is attributed to the molecular polarity induced by the halides, which induces intermolecular interactions.		
<b>Physical state</b>	Compressed Gas	<b>Relative density (Water = 1)</b>	1.49 @ -69 deg.
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	632.2
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature (°C)</b>	Not Available
<b>Melting point / freezing point (°C)</b>	-146	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	-40.8	<b>Molecular weight (g/mol)</b>	86.47
<b>Flash point (°C)</b>	Not Available	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Applicable	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Available	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	100
<b>Vapour pressure (kPa)</b>	922.06 @ 20 deg	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Applicable
<b>Vapour density (Air = 1)</b>	2.98	<b>VOC g/L</b>	1490

## SECTION 10 Stability and reactivity

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

**Hazardous decomposition products**

See section 5

**SECTION 11 Toxicological information****Information on toxicological effects**

<b>Inhaled</b>	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant 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.</p> <p>Exposure to high concentrations of fluorocarbons may produce cardiac arrhythmias or cardiac arrest due sensitisation of the heart to adrenalin or noradrenalin. Deaths associated with exposures to fluorocarbons (specifically halogenated aliphatics) have occurred in occupational settings and in inhalation of bronchodilator drugs.</p> <p>Bronchospasm consistently occurs in human subjects inhaling fluorocarbons. At a measured concentration of 1700 ppm of one of the commercially available aerosols there is a biphasic change in ventilatory capacity, the first reduction occurring within a few minutes and the second delayed up to 30 minutes. Most subjects developed bradycardia (reduced pulse rate).</p> <p>Bradycardia is encountered in dogs when administration is limited to upper respiratory tract (oropharyngeal and nasal areas). Cardiac arrhythmias can be experimentally induced in animals (species dependency is pronounced with dogs and monkeys requiring lesser amounts of fluorocarbon FC-11 than rats or mice). Sensitivity is increased by injection of adrenalin or cardiac ischaemia/necrosis or pulmonary thrombosis/bronchitis. The cardiotoxic effects of the fluorocarbons originate from irritation of the respiratory tract which in turn reflexively influences the heart rate (even prior to absorption of the fluorocarbon) followed by direct depression of the heart after absorption.</p> <p>Exposure to fluorocarbon thermal decomposition products may produce flu-like symptoms including chills, fever, weakness, muscular aches, headache, chest discomfort, sore throat and dry cough. Complete recovery usually occurs within 24 hours of exposure.</p> <p>Chlorodifluoromethane is low in toxicity at concentrations as high as 4% (40000 ppm). Symptoms may include lightheadedness, giddiness, shortness of breath and possibly cardiac arrhythmias (irregular heartbeats). Narcotic effects have been seen at 200000 ppm. Published animal studies report this material reduces heart efficiency at concentrations of 25000 ppm or more. In addition, cardiac sensitisation to adrenaline has been observed at concentrations of 50000 ppm. Studies on two human volunteers who inhaled 10000 ppm for 2.5 hours showed a 7% reduction in a standardised psychomotor test score but no other adverse effects</p> <p>Acute intoxication by halogenated aliphatic hydrocarbons appears to take place over two stages. Signs of a reversible narcosis are evident in the first stage and in the second stage signs of injury to organs may become evident, a single organ alone is (almost) never involved. Depression of the central nervous system is the most outstanding effect of most halogenated aliphatic hydrocarbons. Inebriation and excitation, passing into narcosis, is a typical reaction. In severe acute exposures there is always a danger of death from respiratory failure or cardiac arrest due to a tendency to make the heart more susceptible to catecholamines (adrenalin)</p> <p>Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.</p> <p>Symptoms of asphyxia (suffocation) may include headache, dizziness, shortness of breath, muscular weakness, drowsiness and ringing in the ears. If the asphyxia is allowed to progress, there may be nausea and vomiting, further physical weakness and unconsciousness and, finally, convulsions, coma and death. Significant concentrations of the non-toxic gas reduce the oxygen level in the air. As the amount of oxygen is reduced from 21 to 14 volume %, the pulse rate accelerates and the rate and volume of breathing increase. The ability to maintain attention and think clearly is diminished and muscular coordination is somewhat disturbed. As oxygen decreases from 14-10% judgement becomes faulty; severe injuries may cause no pain. Muscular exertion leads to rapid fatigue. Further reduction to 6% may produce nausea and vomiting and the ability to move may be lost. Permanent brain damage may result even after resuscitation at exposures to this lower oxygen level. Below 6% breathing is in gasps and convulsions may occur. Inhalation of a mixture containing no oxygen may result in unconsciousness from the first breath and death will follow in a few minutes.</p> <p>The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation.</p>
<b>Ingestion</b>	<p>Not normally a hazard due to physical form of product.</p> <p>Considered an unlikely route of entry in commercial/industrial environments</p>
<b>Skin Contact</b>	<p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>In common with other halogenated aliphatics, fluorocarbons may cause dermal problems due to a tendency to remove natural oils from the skin causing irritation and the development of dry, sensitive skin. They do not appear to be appreciably absorbed.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Material on the skin evaporates rapidly and may cause tingling, chilling and even temporary numbness</p> <p>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.</p>
<b>Eye</b>	<p>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 characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>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..</p>
<b>Chronic</b>	<p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>It is generally accepted that the fluorocarbons are less toxic than the corresponding halogenated aliphatic based on chlorine. Repeated inhalation exposure to the fluorocarbon FC-11 does not produce pathologic lesions of the liver and other visceral organs in experimental animals. There has been conjecture in non-scientific publications that fluorocarbons may cause leukemia, cancer, sterility and birth defects; these have not been verified by current research. The high incidence of cancer, spontaneous abortion and congenital anomalies amongst hospital personnel, repeatedly exposed to fluorine-containing general anaesthetics, has caused some scientists to call for a lowering of the fluorocarbon exposure standard to 5 ppm since some are mutagens.</p>

## CHLORODIFLUOROMETHANE

Principal route of occupational exposure to the gas is by inhalation.

chlorodifluoromethane	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: 220000 ppm4h <sup>[2]</sup>	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

## CHLORODIFLUOROMETHANE

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 sensitise 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 metabolised 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 faeces.

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 decreased performance in psychological tests. An exposure to an 11% (545 g/m<sup>3</sup>) 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 (CO<sub>2</sub>) 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 sensitised 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 tumourigenic response in the nasal cavity was observed in rats upon inhalation of CFC-113, this response was considered equivocal. The tumours 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 market place 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 haemorrhage, subarachnoid haemorrhage, stroke, and other cerebrovascular events have been reported in patients treated with 5-HT<sub>1</sub> 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, haemorrhage, transient ischemic attack).

An 18% increase in mean pulmonary artery pressure was seen following dosing with one 5-HT<sub>1</sub> agonist in a study evaluating subjects undergoing cardiac catheterisation.

5-HT<sub>1</sub> 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 5HT<sub>1</sub> agonists; these may present as bloody diarrhea or abdominal pain.

For dichlorotrifluoroethane (HCFC -123) and dichloropentafluoropropane (HCFC-225)

Prolonged inhalation of high concentrations of HCFC-123 vapour 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. Anaesthetic-like effects are observed in rats at high inhalation concentration (greater than 5,000 ppm).

## CHLORODIFLUOROMETHANE

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 dichlorotrifluoroethane (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 categorised in cases of human poisoning. No CNS effects were seen at 2500 ppm (15.6 mg/L) HCFC-123 in a behavioural study in rats.

CFCs and HCFCs are known to sensitise the heart to adrenalin-induced arrhythmias. HCFC-123 caused cardiac sensitisation 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 sensitisation effects for HCFC-123 in humans, such effects have been documented following exposure to other CFCs, including CFC-12, where sensitisation was reported at 10,000 ppm.

In humans, liver toxicity, cardiac sensitisation 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 sensitisation 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 sensitisation response. However, sensitisation 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 haematological effects, neurological disorders, liver damage, reproductive effects and coronary heart disease.

Although behavioural effects and CNS effects have been seen in animals repeatedly exposed to HCFC-123, histological examination in rats of brain, spinal cord and nerve fibres 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, guinea-pigs 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 luteinising hormone (F0 generation only) levels were seen in male rats at 100 ppm and 300 ppm respectively, with a NOAEL of 300 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 vapour.

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 tumours 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 tumours (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 characterising the mechanism for tumourigenicity in animals was reviewed in order to assess its relevance to humans.

Two types of hepatic tumours 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 tumour 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

## CHLORODIFLUOROMETHANE

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 serve 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.<sup>50</sup> 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 tumours<sup>98</sup> 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 tumour incidence with liver beta-oxidation activity at certain exposure levels.

The mechanistic significance of benign hepatocholangiofibromas in female rats is unclear as this tumour 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 tumour 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 tumours are of the ductal type), it must be assumed that, until more is known about the mechanism for acinar cell tumour 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 tumours in men are extremely rare, representing less than three per cent of all testicular neoplasms. The rarity of this tumour 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 tumours 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 tumours occur, the cell type (except cholangiocellular tissue) has been a site of tumourigenic activity for other peroxisome proliferators, including hypolipidaemic drugs. As this triad of tumour types have not been reported in epidemiological data on hypolipidaemic drugs (classic peroxisome proliferating substances), it has been hypothesised that hepatic, testicular and pancreatic tumours 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 tumours 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 tumourigenicity 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.<sup>105</sup> Such a mechanism might account for the sex differences and the lack of target organ specificity<sup>?</sup>with respect to HCFC-123 elicited tumours.

In summary, until further data become available regarding the mechanism of HCFC-123 induced tumours, 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) re formed when disinfectants such as chlorine, chloramine, and ozone react with organic and inorganic matter in water. The observations that some DBPs such as trihalomethanes (THMs), di-/trichloroacetic acids, and 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) are carcinogenic in animal studies have raised public concern over the possible adverse health effects of DBPs. To date, several hundred DBPs have been identified.

Numerous haloalkanes and haloalkenes have been tested for carcinogenic and mutagenic activities. 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 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.

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

chlorodifluoromethane	Endpoint	Test Duration (hr)	Species	Value	Source
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Continued...

## CHLORODIFLUOROMETHANE

	EC50	48h	Crustacea	433mg/l	2
	EC50	96h	Algae or other aquatic plants	250mg/l	2
	EC50(ECx)	96h	Algae or other aquatic plants	250mg/l	2

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

Koc : 57.5 Half-life (hr) air : 97236-151548 Half-life (hr) H2O surface water : 2.7 Henry's atm m3 /mol: 0.0294 BCF : 3.9 controlled ozone depleting gases: phase out by 1996 (C)

Bioaccumulation : not sig processes Abiotic: RxnOH\*,hydrol

On the basis of the available evidence concerning properties and predicted or observed environmental fate and behavior, the material may present a danger to the structure and/or functioning of the stratospheric ozone layer.

For haloalkanes and haloalkenes:

**Environmental fate:**

Certain haloalkane gases in the atmosphere can also contribute to the greenhouse effect by restricting heat loss from the Earth's atmosphere through absorbing infrared emissions from the surface. Generally haloalkanes contributing to the greenhouse effect consist of a fully or partly fluorinated carbon backbone.

Gas-phase reactions with OH radicals are the major tropospheric loss process for the haloalkanes. In addition photooxidation reactions with O3 and NO3 radicals can result in transformation.

Organic substances containing chlorine, if primarily present in the atmospheric compartment and if their lifetime is long enough can reach the stratosphere and decompose through photolysis and other chemical reaction (e.g. with OH radical). Chlorine atoms can then participate in the catalytic ozone destruction cycles. The atmospheric lifetime is too short to enable a significant fraction of the compound emitted to reach the stratosphere

Haloalkanes do not hydrolyse easily. Acids do not catalyse the hydrolysis and base catalysis is only important at higher pHs than are observed in the environment.

The apparent hazard of halo-alkanes and alkenes to human health has prompted investigations concerning their fate in subsurface waters and in soil. Although abiotic transformations may be significant within the time scales commonly associated with groundwater movement, biotic process typically proceed much faster, provided that there are sufficient substrates, nutrients and microbial populations to mediate such transformations. Several bacterial strains including methane-utilising bacteria capable of utilising haloalkanes have been isolated. Microbial dehalogenation by these strains is mediated by enzymes (oxygenase and hydrolase). The biodegradation of haloalkanes can proceed through different pathways. Haloparaffins (C12 to C18) have been reported to be incorporated into fatty acids in bacteria, yeasts, and fungi, resulting in their accumulation in the food chain. Another pathway is the oxygenation at the nonhalogenated end of monohalogenated alkanes by an inherent oxygenase with a tight substrate selectivity. In this case fluoroalkanes were defluorinated, but no dehalogenation was observed with chloro-, bromo-, or iodoalkanes. Chain length was reported to have minor effects on this oxygenation reaction. In general, alpha- and alpha,omega-chlorinated haloalkanes with short carbon chains (C1 to C6) are dehalogenated hydrolytically or by a glutathione-dependent mechanism. In contrast, alpha- and alpha,omega-haloalkanes with longer chains, e.g., 1,9-dichlorononane and 1,10-dichlorodecane (1,10-DCD), have been proposed to be dehalogenated by oxidative mechanisms. Studies on the biodegradation of this class of compounds are rare, because haloalkane-degrading microorganisms are not easily found. In water and terrestrial compartments haloalkanes may hydrolyse in the presence of naturally occurring sulfur-containing nucleophiles. Bisulfide ion (HS-) is generally the most important nucleophile because it is moderately reactive and is usually present at the highest concentration. When elemental sulfur is present, polysulfides (S4 2- and S5 2-) will be more important than HS- at pH 7 (approximately) because they are 60 times more reactive and their equilibrium concentrations increase with increasing pH. The end products of such reactions include a variety of mercaptans and dialkyl sulfides.

for chlorodifluoromethane (HCFC-22):

Atmospheric lifetime approximately 12 years

For HCFCs:

Hydrochlorofluorocarbons (HCFCs) are Class II Ozone Depletion Substances (ODSs). They deplete stratospheric ozone, but to a lesser extent than most Class I ODSs. HCFCs generally have Ozone depleting Potentials (ODPs) of 0.1 or less.

The stratospheric ozone layer shields the Earth from the sun's harmful ultraviolet radiation. Emissions of certain synthetic chemicals - including CFCs, halons, and HCFCs - destroy the ozone layer, and have created an "ozone hole" over the South

Pole. Through the Montreal Protocol on Substances that Deplete the Ozone Layer, the international community committed to a collaborative, international effort to regulate and phase out ozone-depleting substances. While CFCs and halons were phased out in the mid 90s, the use of HCFC consumption must be phased out in a step-wise fashion.

All HCFCs (except for use as refrigerants in equipment manufactured before 1/1/2020) must be phased out by 1/1/2015

All HCFCs (no exceptions) must be phased out by 1/1/2030

**DO NOT discharge into sewer or waterways.**

**Persistence and degradability**

Ingredient	Persistence: Water/Soil	Persistence: Air
chlorodifluoromethane	LOW	LOW

**Bioaccumulative potential**

Ingredient	Bioaccumulation
chlorodifluoromethane	LOW (LogKOW = 1.08)

**Mobility in soil**

Ingredient	Mobility
chlorodifluoromethane	LOW (Log KOC = 23.74)

**SECTION 13 Disposal considerations****Waste treatment methods**

Product / Packaging disposal	
	<ul style="list-style-type: none"> <li>▶ Evaporate residue at an approved site.</li> <li>▶ Return empty containers to supplier. If containers are marked non-returnable establish means of disposal with manufacturer prior to purchase.</li> <li>▶ Ensure damaged or non-returnable cylinders are gas-free before disposal.</li> </ul>

**SECTION 14 Transport information****Labels Required**

	
Marine Pollutant	NO

**Land transport (UN)**

Continued...

## CHLORODIFLUOROMETHANE

14.1. UN number or ID number	1018	
14.2. UN proper shipping name	CHLORODIFLUOROMETHANE (REFRIGERANT GAS R 22)	
14.3. Transport hazard class(es)	Class	2.2
	Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	Not Applicable
	Limited quantity	120 ml

## Air transport (ICAO-IATA / DGR)

14.1. UN number	1018	
14.2. UN proper shipping name	Refrigerant gas R 22; Chlorodifluoromethane	
14.3. Transport hazard class(es)	ICAO/IATA Class	2.2
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	2L
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	Not Applicable
	Cargo Only Packing Instructions	200
	Cargo Only Maximum Qty / Pack	150 kg
	Passenger and Cargo Packing Instructions	200
	Passenger and Cargo Maximum Qty / Pack	75 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Forbidden
	Passenger and Cargo Limited Maximum Qty / Pack	Forbidden

## Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1018	
14.2. UN proper shipping name	REFRIGERANT GAS R 22; CHLORODIFLUOROMETHANE	
14.3. Transport hazard class(es)	IMDG Class	2.2
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	F-C , S-V
	Special provisions	Not Applicable
	Limited Quantities	120 mL

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

Not Applicable

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

Product name	Group
chlorodifluoromethane	Not Available

## 14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
chlorodifluoromethane	Not Available

## SECTION 15 Regulatory information

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

chlorodifluoromethane is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic  
 UNEP (United Nations Environment Programme) Montreal Protocol Ozone Depletors - Annex C

## Additional Regulatory Information

Not Applicable

## National Inventory Status

Continued...

## CHLORODIFLUOROMETHANE

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (chlorodifluoromethane)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
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
<b>Legend:</b>	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

<b>Revision Date</b>	02/06/2023
<b>Initial Date</b>	16/06/2005

## CONTACT POINT

The above information is believed to be correct but does not purport to be all inclusive and shall be used only as a guide. The information in this document is based on the present state of our knowledge and is applicable to the product with regard to appropriate safety precautions. It does not represent any guarantee of the properties of the product. NFIL shall not be held liable for any damage resulting from handling or from contact with the above product.

## SDS Version Summary

Version	Date of Update	Sections Updated
8.1	20/09/2018	Firefighting measures - Fire Fighter (fire/explosion hazard)
9.1	02/06/2023	Expiration. Review and Update

## Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

## Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit,
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration
  
- ▶ AIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European Inventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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TEL (+61 3) 9572 4700.