2,2-Dichloro-1,1,1-trifluoroethane

sc-257551

Material Safety Data Sheet

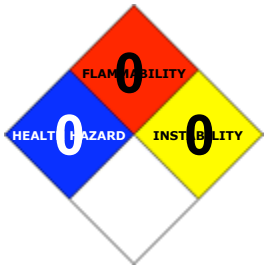
Hazard Alert Code Key:	EXTREME	HIGH	MODERATE	LOW

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
2,2-Dichloro-1,1,1-trifluoroethane

STATEMENT OF HAZARDOUS NATURE
CONSIDERED A HAZARDOUS SUBSTANCE ACCORDING TO OSHA 29 CFR 1910.1200.

NFPA



SUPPLIER
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SYNONYMS
CHCl2-CF3, C2-H-Cl2-F3, "ethane, 2, 2-dichloro-1, 1-trifluoroethane", "1, 1-dichloro-2, 2, 2-trifluoroethane", dichlorotrifluoroethane, dichloro(trifluoromethyl)methane, trifluorodichloroethane, "FC 123", "Freon 123", "R 123", R-123, HCFC-123, "HCFC 123", "hydrochloro fluorocarbon", "1, 1, 1-trifluoro-2, 2-dichloroethane", "hydro chloro fluoro carbon", "halogenated hydrocarbon", fluorocarbon, dichlorotrifluorocarbon

Section 2 - HAZARDS IDENTIFICATION

CHEMWATCH HAZARD RATINGS

		Min	Max
Flammability:	0		
Toxicity:	2		
Body Contact:	2		
Reactivity:	1		
Chronic:	2		

Min/Nil=0
Low=1
Moderate=2
High=3
Extreme=4

CANADIAN WHMIS SYMBOLS

EMERGENCY OVERVIEW

RISK

Limited evidence of a carcinogenic effect.
May cause harm to breastfed babies.
Harmful: danger of serious damage to health by prolonged exposure through inhalation.
Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

■ Although ingestion is not thought to produce harmful effects, the material may still be damaging to the health of the individual following ingestion, especially where pre-existing organ (e.
g.

EYE

■ Although the liquid is not thought to be an irritant, direct contact with the eye may produce transient discomfort characterized by tearing or conjunctival redness (as with windburn).

SKIN

■ Skin contact is not thought to have harmful health effects, however the material may still produce health damage following entry through wounds, lesions or abrasions.
■ Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.
■ There is some evidence to suggest that this material can cause inflammation of the skin on contact in some persons.
■ Fluorocarbons remove natural oils from the skin, causing irritation,dryness and sensitivity.
■ Open cuts, abraded or irritated skin should not be exposed to this material.
■ Material on the skin evaporates rapidly and may cause tingling, chillingand even temporary numbness.
■ Entry into the blood-stream, through, for example, cuts, abrasions 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.

INHALED

■ Inhalation of vapours may cause drowsiness and dizziness.
This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.
■ Inhalation of vapors or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
■ There is some evidence to suggest that the material can cause respiratory irritation in some persons.
The body's response to such irritation can cause further lung damage.
■ 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.
■ Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas.
Vapor is heavier than air and may displace and replace air in breathing zone, acting as a simple asphyxiant.
■ 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.
■ Exposure to fluorocarbons can produce non-specific flu-like symptoms such as chills, fever, weakness, muscle pain, headache, chest discomfort, sore throat and dry cough with rapid recovery.
High concentrations can cause irregular heartbeats and a stepwise reduction in lung capacity.

CHRONIC HEALTH EFFECTS

■ Harmful: danger of serious damage to health by prolonged exposure through inhalation.
Harmful: danger of serious damage to health by prolonged exposure through inhalation.
This material can cause serious damage if one is exposed to it for long periods. It can be assumed that it contains a substance which can produce severe defects.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.
Fluorocarbons can cause an increased risk of cancer, spontaneous abortionand birth defects.
Inhalation of up to 5000 ppm for two years caused benign testicular and benign pancreatic tumours in male rats; benign pancreatic tumours were observed in female rats exposed to 5000 ppm. Both sexes exhibited an increase in benign liver tumours when exposed to 5000 ppm over two years.
It should be concluded * that until further data becomes available about the mechanism of HCFC-123 induced tumours, particularly in respect of cholangiofibroma and pancreatic adenoma induction, that the carcinogenicity findings in rats may have relevance for humans.
* [Commonwealth of Australia Gazette September 1997]
No genotoxic or adverse reproductive effects were apparent in animal studies; one study indicated mammalian cell culture genetic

damage.*
[DuPont]

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
2,2-dichloro-1,1,1-trifluoroethane	306-83-2	> 99

Section 4 - FIRST AID MEASURES

SWALLOWED
■ If poisoning occurs, contact a doctor or Poisons Information Center. · Avoid giving milk or oils. · Avoid giving alcohol. · If swallowed do NOT induce vomiting. · If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

EYE
■ If this product comes in contact with the eyes: · Wash out immediately with fresh running water. · Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.

SKIN
■ If skin contact occurs: · Immediately remove all contaminated clothing, including footwear · Flush skin and hair with running water (and soap if available).

INHALED
· If fumes or combustion products are inhaled remove from contaminated area. · Lay patient down. Keep warm and rested.

NOTES TO PHYSICIAN
■ Treat symptomatically.
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 sensitization may be treated with propranolol, 1-2 mg IV or esmolol 25-100 microgm/kg/min IV.

Section 5 - FIRE FIGHTING MEASURES

Vapor Pressure (mmHg):	672.055 @ 25 deg C
Upper Explosive Limit (%):	Not Available
Specific Gravity (water=1):	1.46 @ 25 deg C
Lower Explosive Limit (%):	Not Available

EXTINGUISHING MEDIA
· There is no restriction on the type of extinguisher which may be used.
Use extinguishing media suitable for surrounding area.

FIRE FIGHTING
· Alert Emergency Responders and tell them location and nature of hazard.
· Wear breathing apparatus plus protective gloves for fire only.

GENERAL FIRE HAZARDS/HAZARDOUS COMBUSTIBLE PRODUCTS
■ Decomposes on heating and produces acrid and toxic fumes of: carbon dioxide (CO_2), hydrogen chloride, phosgene, hydrogen fluoride, other pyrolysis products typical of burning organic material.
Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.
· Non combustible.
· Not considered to be a significant fire risk.
· Heating may cause expansion or decomposition leading to violent rupture of containers.
· May emit corrosive, poisonous fumes.

FIRE INCOMPATIBILITY
■ Avoid contamination with oxidizing agents i.e. nitrates, oxidizing acids,chlorine bleaches, pool chlorine etc. as ignition may result.

PERSONAL PROTECTION
Glasses:
Chemical goggles.
Gloves:
Respirator:
Type AX Filter of sufficient capacity

Section 6 - ACCIDENTAL RELEASE MEASURES

MINOR SPILLS
· Clean up all spills immediately.
· Avoid breathing vapors and contact with skin and eyes.
MAJOR SPILLS
■ Moderate hazard.
· Clear area of personnel and move upwind.
· Alert Emergency Responders and tell them location and nature of hazard.

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

■ Contains low boiling substance:
Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.
· Check for bulging containers.
· Vent periodically.
· DO NOT allow clothing wet with material to stay in contact with skin.
· Avoid all personal contact, including inhalation.
· Wear protective clothing when risk of exposure occurs.

RECOMMENDED STORAGE METHODS

■ DO NOT use aluminum or galvanized containers.
· Polyethylene or polypropylene container.
· Packing as recommended by manufacturer.

STORAGE REQUIREMENTS

■ Observe manufacturer's storing and handling recommendations.
· Store at 4˚C.

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m³	STEL ppm	STEL mg/m³	Peak ppm	Peak mg/m³	TWA F/CC	Notes
Canada - Alberta Occupational Exposure Limits	2,2-dichloro-1,1,1-trifluoroethane (1,1,1-Trifluoro-2,2dichloroethane (HCFC-123))	50	310						
US AIHA Workplace Environmental Exposure Levels (WEELs)	2,2-dichloro-1,1,1-trifluoroethane (1,1,1-Trifluoro-2,2-Dichloroethane)	50							
US - Hawaii Air Contaminant Limits	2,2-dichloro-1,1,1-trifluoroethane (Fluorides (as F))		2.5						(CAS (Varies with compound))

ENDOELTABLE

PERSONAL PROTECTION

RESPIRATOR

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

EYE
· Safety glasses with side shields.
· Chemical goggles.

HANDS/FEET
■ Wear chemical protective gloves, eg. PVC.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: such as:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Contaminated gloves should be replaced.
Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
· Protective gloves eg. Leather gloves or gloves with Leather facing.

OTHER
· Overalls.
· P.V.C. apron.
· Barrier cream.
· Skin cleansing cream.
· Eye wash unit.

ENGINEERING CONTROLS
■ Local exhaust ventilation usually required. If risk of overexposure exists, wear an approved respirator.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

PHYSICAL PROPERTIES
Liquid.

State	Liquid	Molecular Weight	152.93
Melting Range (°F)	-161	Viscosity	Not Available
Boiling Range (°F)	82(26-29)	Solubility in water (g/L)	Partly Miscible
Flash Point (°F)	Not Applicable	pH (1% solution)	Not applicable.
Decomposition Temp (°F)	Not available	pH (as supplied)	Not applicable
Autoignition Temp (°F)	Not Available	Vapor Pressure (mmHg)	672.055 @ 25 deg C
Upper Explosive Limit (%)	Not Available	Specific Gravity (water=1)	1.46 @ 25 deg C
Lower Explosive Limit (%)	Not Available	Relative Vapor Density (air=1)	5.3
Volatile Component (%vol)	100	Evaporation Rate	<1 (CCl4=1)

APPEARANCE
Clear colourless highly volatile liquid which boils on a warm day; i.e. at temperatures above 23 C.. Solubility in water = 0.4% @ 25 C. Ethereal odour. Soluble in ether, alcohol and benzene.

Section 10 - CHEMICAL STABILITY

CONDITIONS CONTRIBUTING TO INSTABILITY
· Presence of incompatible materials.
· Product is considered stable.

STORAGE INCOMPATIBILITY
■ Segregate from:
· powdered metals such as aluminium, zinc and
· alkali metals such as sodium, potassium and lithium.
May attack, soften or dissolve rubber, many plastics, paints and coatings.

For incompatible materials - refer to Section 7 - Handling and Storage.

Section 11 - TOXICOLOGICAL INFORMATION

2,2-dichloro-1,1,1-trifluoroethane

TOXICITY AND IRRITATION

2,2-DICHLORO-1,1,1-TRIFLUOROETHANE:

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

TOXICITY	IRRITATION
Inhalation (mouse) LC50: 74000 ppm/1h	Nil Reported
Inhalation (rat) LC50: 32000 ppm/4h*	
Oral (rat) LDLo: 9000 mg/kg*	
Dermal (rabbit) LD50: >2000 mg/kg*	

NOTE: The compound is non-irritating to skin and does not act as a skin sensitiser in experimental animals. [Du Pont]*

No data exist on the oral and dermal toxicity of 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. 12,64 HCFC-123 was a slight eye irritant in rabbits.

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. A NOAEL for CNS (anaesthetic) effects in rats and

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 materno-toxicity 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 30 ppm.As these effects were not consistent between generations, biological significance was considered questionable.

In rabbits, developmental effects (increased resorptions and foetal defects) were seen only at doses which caused maternotoxicity, that is, greater than 10,000 ppm.

In rats, HCFC-123 caused reduced pup growth in the offspring of the F1 generation at and above 30 ppm, and the F0 generation, at and

above 100 ppm. Sexual maturation was also slightly delayed in F1 males (F0 offspring) at and above 300 ppm. However, the group mean body weight at attainment of sexual maturity was similar to controls, suggesting differences in pup growth rates may account for this delay.

Reduced pup growth was not considered to be a developmental effect as significant reduction in pup weight was not seen until seven to 14 days after birth. This effect may however be caused by HCFC-123 in breast milk (a lactational effect) as:

the onset of reduced pup growth occurred during the period when exposure to HCFC-123 was restricted to parent dams;
indicators of the integrity (quantity and quality) of milk, for example, CCK and milk fat, were normal during the suckling period; and
maternal food intake during lactation was only decreased at and above 300 ppm HCFC-123.

The genotoxic potential of HCFC-123 has been studied in a number of in vitro and in vivo bioassays. Most of these studies were designed to evaluate the genotoxic effects from exposure to HCFC-123 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.

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 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.50 However, a significant increase in peroxisome proliferation was seen at this concentration in female rats in a 90 day study by the same laboratory and as such this anomaly was considered by the study author to represent a biological variation in beta-oxidation potential. In addition, despite a dose related (significant) increase in peroxisome proliferation in male rats (in the two-year study) at 300 ppm and 1000 ppm, no increase was seen in liver adenomas at these exposure levels.

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

Finally, HCFC-123 has a similar metabolic profile to halothane with respect to TFA formation, beta-oxidation potential and effects on serum lipids. However, halothane did not induce tumours98 in either rats or mice. This finding should not be regarded as strong evidence of a non-peroxisomal mechanism for HCFC-123 as some peroxisome proliferators are more
potent carcinogens than others, despite inducing similar levels of peroxisome proliferation, and only limited data on carcinogenicity for halothane were available.

Although it is considered likely that the benign hepatocellular adenomas seen in rats exposed to HCFC-123 are related to increases in hepatic peroxisome proliferation (a mechanism believed not to present a hepatocarcinogenic hazard to humans), anomalies exist with respect to this proposed mechanism, mainly due to the lack of concordance of 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.105 Such a mechanism might account for the sex differences and the lack of target organ specificity?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.

Section 12 - ECOLOGICAL INFORMATION

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
2,2-dichloro-1,1,1-trifluoroethane	HIGH	No Data Available	LOW	MED

Section 13 - DISPOSAL CONSIDERATIONS

Disposal Instructions
All waste must be handled in accordance with local, state and federal regulations.
¦ Puncture containers to prevent re-use and bury at an authorized landfill.
Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.
A Hierarchy of Controls seems to be common - the user should investigate:
· Reduction
· Reuse
· Recycling
· Disposal (if all else fails)
This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.
DO NOT allow wash water from cleaning equipment to enter drains. Collect all wash water for treatment before disposal.
· Recycle wherever possible.
· Consult manufacturer for recycling options or consult Waste Management Authority for disposal if no suitable treatment or disposal facility can be identified.

Section 14 - TRANSPORTATION INFORMATION

NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS: DOT, IATA, IMDG

Section 15 - REGULATORY INFORMATION

2,2-dichloro-1,1,1-trifluoroethane (CAS: 306-83-2) is found on the following regulatory lists;
"Canada - Alberta Occupational Exposure Limits","Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS (English)","International Council of Chemical Associations (ICCA) - High Production Volume List","US AIHA Workplace Environmental Exposure Levels (WEELs)","US DOE Temporary Emergency Exposure Limits (TEELs)","US EPA High Production Volume Program Chemical List","US EPA Master Testing List - Index I Chemicals Listed","US EPCRA Section 313 Chemical List","US List of Lists - Consolidated List of Chemicals Subject to EPCRA, CERCLA and Section 112(r) of the Clean Air Act","US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory","US TSCA Section 5(a)(2) - Significant New Use Rules (SNURs)","US TSCA Section 8 (d) - Health and Safety Data Reporting"

Section 16 - OTHER INFORMATION

LIMITED EVIDENCE
■ Inhalation may produce health damage*.
■ Cumulative effects may result following exposure*.
■ May produce discomfort of the respiratory system and skin*.
■ Repeated exposure potentially causes skin dryness and cracking*.
■ Vapours potentially cause drowsiness and dizziness*.
* (limited evidence).

Denmark Advisory list for selfclassification of dangerous substances
Substance CAS Suggested codes 2, 2- dichloro- 1, 1, 1- trifluoroethane 306- 83- 2 R52/53

Reasonable care has been taken in the preparation of this information, but the author makes no warranty of merchantability or any other warranty, expressed or implied, with respect to this information. The author makes no representations and assumes no liability for any direct, incidental or consequential damages resulting from its use. For additional technical information please call our toxicology department on +800 CHEMCALL.

■ 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.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net/references.

■ The (M)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.

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