A-Gas R123

A-Gas (Singapore) PTE LTD

Chemwatch Hazard Alert Code: **2**

Chemwatch: **6100-23**
Version No: **6.1.1.1**
Material Safety Data Sheet according to NOHSC and ADG requirements

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Initial Date: **Not Available**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	A-Gas R123
Chemical Name	2,2-DICHLORO-1,1,1-TRIFLUOROETHANE
Synonyms	R 123, HFA-123, HCFC-123, Suva123, Solkane 123
Proper shipping name	Not Applicable
Chemical formula	Not Applicable
Other means of identification	Not Available
CAS number	306-83-2

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

Relevant identified uses	Use according to manufacturer's directions. 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.

Details of the supplier of the safety data sheet

Registered company name	A-Gas (Singapore) PTE LTD		
Address	360 Orchard Road, #10-05, Int'l Building 238869 Singapore		
Telephone	65 6836 0065		
Fax	65 6836 6521		
Website	www.agas.com		
Email	Not Available		

Emergency telephone number

Association / Organisation	Not Available		
Emergency telephone numbers	65 6836 0065		
Other emergency telephone numbers	65 6836 0065		

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	+612 9186 1132	Not Available

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS SUBSTANCE. NON-DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.

CHEMWATCH HAZARD RATINGS

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

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

Poisons Schedule	None
Risk Phrases [1]	**R52/53** Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
	R40(3) Limited evidence of a carcinogenic effect.

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	R48/20	Harmful: danger of serious damage to health by prolonged exposure through inhalation.
	R64	May cause harm to breastfed babies.
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*	
GHS Classification [1]	Carcinogen Category 2, Lactation Effects, STOT - RE Category 2, Chronic Aquatic Hazard Category 3	
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*	

Label elements

GHS label elements	
SIGNAL WORD	**WARNING**

Hazard statement(s)

H351	Suspected of causing cancer
H362	May cause harm to breast-fed children
H373	May cause damage to organs through prolonged or repeated exposure
H412	Harmful to aquatic life with long lasting effects

Precautionary statement(s): Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P263	Avoid contact during pregnancy/while nursing.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.

Precautionary statement(s): Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P314	Get medical advice/attention if you feel unwell.

Precautionary statement(s): Storage

P405	Store locked up.

Precautionary statement(s): Disposal

P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration

Label elements

Relevant risk statements are found in section 2

Indication(s) of danger	T, Xn

SAFETY ADVICE

S01	Keep locked up.
S07	Keep container tightly closed.
S09	Keep container in a well ventilated place.
S13	Keep away from food, drink and animal feeding stuffs.
S20	When using do not eat or drink.
S23	Do not breathe gas/fumes/vapour/spray.
S28	After contact with skin, wash immediately with plenty of water
S29	Do not empty into drains.
S35	This material and its container must be disposed of in a safe way.
S36	Wear suitable protective clothing.
S37	Wear suitable gloves.
S38	In case of insufficient ventilation, wear suitable respiratory equipment.
S40	To clean the floor and all objects contaminated by this material, use water and detergent.

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S45	In case of accident or if you feel unwell IMMEDIATELY contact Doctor or Poisons Information Centre (show label if possible).
S46	If swallowed, seek medical advice immediately and show this container or label.
S51	Use only in well ventilated areas.
S53	Avoid exposure - obtain special instructions before use.
S56	Dispose of this material and its container at hazardous or special waste collection point.
S57	Use appropriate container to avoid environmental contamination.

Other hazards

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
306-83-2	>99.5	2,2-dichloro-1,1,1-trifluoroethane

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	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. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor.
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor. ▸ 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. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

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 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.

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	▸ 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

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

	▸ Foam. ▸ Dry chemical powder. ▸ BCF (where regulations permit). ▸ Carbon dioxide.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO).

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment.
Major Spills	Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe 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
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. |Storage temperature <50 deg.c.>

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents Segregate from: ▸ powdered metals such as aluminium, zinc and ▸ alkali metals such as sodium, potassium and lithium.

PACKAGE MATERIAL INCOMPATIBILITIES

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Continued...

Not Available

EMERGENCY LIMITS

Ingredient	TEEL-0	TEEL-1	TEEL-2	TEEL-3
2,2-dichloro-1,1,1-trifluoroethane	50(ppm)	150(ppm)	1000(ppm)	10000(ppm)

Ingredient	Original IDLH	Revised IDLH
A-Gas R123	Not Available	Not Available

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk.
Personal protection	
Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task.
Skin protection	See Hand protection below
Hand protection	The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C. apron. ▸ Barrier cream.
Thermal hazards	

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the A-Gas R123 Not Available

Material	CPI

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion

Respiratory protection

Type A Filter of sufficient capacity

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P3	-	A-PAPR-AUS / Class 1 P3
up to 50 x ES	-	A-AUS / Class 1 P3	-
up to 100 x ES	-	A-2 P3	A-PAPR-2 P3 ^

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Dark brown viscous, non-volatile, hygroscopic liquid with a slightly ethereal odour; insoluble in water.

Physical state	Liquid	**Relative density (Water = 1)**	1.58

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Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	350
pH (as supplied)	Not Applicable	**Decomposition temperature**	>160
Melting point / freezing point (°C)	<-100 (freezing point)	**Viscosity (cSt)**	7000 mPa.s
Initial boiling point and boiling range (°C)	>160	**Molecular weight (g/mol)**	152.9
Flash point (°C)	196 (OC)	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	0.470 @ 20 deg.C	**Gas group**	Not Available
Solubility in water (g/L)	Immiscible	**pH as a solution(1%)**	Not Applicable
Vapour density (Air = 1)	>1	**VOC g/L**	

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	▸ Presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. 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.
Ingestion	The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence.
Skin Contact	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.
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. 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.

A-Gas R123	**TOXICITY**	**IRRITATION**
	Dermal (Rat) LD50: >2000 mg/kg	Guinea Pig, Non sensitising (skin)
	Inhalation (Rat) LC50: 200 mg/l	Rabbit, slightly irritant (eyes)
	Oral (Rat) LD50: >2000 mg/kg	Rabbit, slightly irritant (skin)
2,2-dichloro-1,1,1-trifluoroethane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg*	
	Inhalation (mouse) LC50: 74000 ppm/1h	
	Inhalation (rat) LC50: 32000 ppm/4h*	
	Not Available	Not Available

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** Value obtained from manufacturer's msds*
unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances

A-Gas R123	Chronic toxicity . Dog, > 1 % v/v air , cardiac sensitisation following adrenergic stimulation . Inhalation, after prolonged exposure, rat, Target organ: liver, 30 ppm, observed effect . Inhalation, after repeated exposure, guinea pig, Target organ: liver / metabolism (lipids) / Endocrine system, 0.94 % v/v air , observed effect . Inhalation, after repeated exposure, monkey, Target organ: liver, 0.1 % v/v air , observed effect . Inhalation, after prolonged exposure, rabbit, Target organ: testes / pancreas / liver, Remark: Leydig cells/benign tumours . No mutagenic, teratogenic effects
2,2-DICHLORO-1,1,1-TRIFLUOROETHANE	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. 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. 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. 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

Continued...

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.

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

CMR STATUS

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

DO NOT discharge into sewer or waterways.
|Acute ecotoxicity:|. Fishes, Salmo gairdneri, LC 50, 96 h, 55.5 mg/l|. Crustaceans, Daphnia magna, EC 50, 48 h, 17.3 mg/l|. Algae, Selenastrum capricornutum, EC 50, 96 h, 96.6 mg/l|Mobility:|. Air, Henry's law constant (H) ca. 3,570 Pa.m3/mol|Result: considerable volatility||. Water, evaporation, t 1/2 ca. 23 hour(s)|Conditions: 25 ° C / calculated value|. Soil/sediments, adsorption, log KOC from 1.8 - 2.6|Abiotic degradation:|. Air, indirect photo-oxidation, t 1/2 = 1.18 year(s)|Conditions: sensitiser: OH radicals|Degradation's products: trifluoroacetic acid / carbon dioxide / hydrochloric acid/fluorhydric acid|. Air, photolysis, ODP = 0.02|Result: limited effect on stratospheric ozone|Reference value for CFC 11: ODP = 1.|. Air, greenhouse effect, GWP = 0.022|Reference value for CFC 11: GWP = 1.|. Water/soil|Result: non-significant hydrolysis and photolysis|Biotic degradation:|. Aerobic, test: ready biodegradability/closed bottle, degradation = 24 %, 28 day(s)|Result: non-readily biodegradable|. Aerobic, test: biodegradation by methane oxidation|Result: non-biodegradable|Conditions: inoculum: Methylosinus trichosporium OB3b

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
Not Available	Not Available	Not Available

Bioaccumulative potential

Ingredient	Bioaccumulation
Not Available	Not Available

Mobility in soil

Ingredient	Mobility
Not Available	Not Available

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

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

SECTION 14 TRANSPORT INFORMATION

Continued...

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

SECTION 15 REGULATORY INFORMATION

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

2,2-dichloro-1,1,1-trifluoroethane(306-83-2) is found on the following regulatory lists	"International Council of Chemical Associations (ICCA) - High Production Volume List","OECD List of High Production Volume (HPV) Chemicals","Australia Customs (Prohibited Exports) Regulations 1958 - Schedule 15 Ozone depleting substances - Part 5 Hydrochlorofluorocarbons","Australia National Pollutant Inventory","Australia Hazardous Substances Information System - Consolidated Lists"

SECTION 16 OTHER INFORMATION

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.
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.