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NATIONAL INDUSTRIAL CHEMICALS NOTIFICATION AND ASSESSMENT SCHEME

FULL PUBLIC REPORT

HFC-143a

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For Enquiries please contact Ms Tina Anderson at:

Street Address: 92 Parramatta Rd Camperdown, NSW 2050, AUSTRALIA
Postal Address: GPO Box 58, Sydney 2001, AUSTRALIA
Telephone: (61) (02) 565-9466 **FAX (61) (02) 565-9465**

Director
Chemicals Notification and Assessment

FULL PUBLIC REPORT

HFC-143a

1. APPLICANT

Du Pont (Australia) Ltd, 168 Walker St, North Sydney, NSW, 2060

2. IDENTITY OF THE CHEMICAL

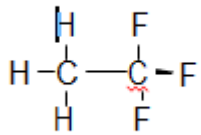
Chemical name: Ethane, 1,1,1-trifluoro-

Chemical Abstracts Service (CAS) Registry No.: 420-46-2

Trade names: HFC-143a (HFC-143a is a component of the refrigerant SUVA HP62)

Molecular formula: CF_3CH_3

Structural formula:



Molecular weight: 84.04

Method of detection and determination:

Infrared Spectroscopy

Spectral data:

The IR spectrum was obtained using a gas cell with an AgCl window. Major peaks were observed at: 960, 1240, 1280 and 1380-1460 cm^{-1}.

3. PHYSICAL AND CHEMICAL PROPERTIES

Appearance at 20°C and 101.3 kPa: colourless gas

Boiling Point: -47.29°C at 101.3 kPa

Freezing Point:	-111.3°C
Specific Gravity/Density:	
liquid density at 25°C:	962 kg/m^3
saturated vapour density:	4.54 kg/m^3
Vapour Pressure:	1262.6 kPa at 25°C
Water Solubility:	0.456 Wt% at 30°C, 1420 kPa
Hydrolytic stability:	No data available. However, hydrolysis is not expected to be a significant degradation pathway as the notified substance is a stable gas
Partition coefficient:	Not measured. The notified substance would be expected to partition to the atmosphere in open systems, rather than between liquid media
Soil adsorption/desorption:	Not measured. Significant sorption to soil is not expected as the notified substance is a gas
Flash Point:	-90°C
Flammability Limits:	
Lower Explosive Limit:	7.7% by volume in air
Upper Explosive Limit:	17.4% by volume in air
Decomposition Products:	hydrogen fluoride and carbonyl halides
Autoignition Temperature:	750°C
Reactivity/Stability:	extremely flammable gas; reacts with oxidising materials

4. PURITY OF THE CHEMICAL

Degree of purity: > 99%

Toxic or hazardous impurities (> 0.1% by weight): None

Non-hazardous impurities (> 1% by weight): None

Additives/Adjuvants: None

5. INDUSTRIAL USE

The notified chemical is to be used as a component (52%) of a refrigerant (SUVA HP62) in low temperature supermarket freezers and chillers. The refrigerant may also be used in other applications where the CFC-containing refrigerant which it replaces is now used. Such applications include refrigerated transport, commercial and retail food distribution and warehousing, groceries, food processing and food service operations, and restaurants. SUVA HP62 will be sold as a non-flammable refrigerant blend to equipment manufacturers and service contractors.

SUVA HP62 containing the notified chemical will be imported in 1 tonne tanks or ISO containers approved for international transportation.

HFC-143a is expected to be imported at a rate of less than 100 tonnes per year for the first 5 years.

6. OCCUPATIONAL EXPOSURE

Following importation, SUVA HP62 will be decanted into nominal 22 kg and 65 kg cylinders for transportation.

It is expected that a very large number of refrigeration service mechanics and personnel in manufacturers plants could be involved in the charging and recharging of refrigerants into refrigeration equipment.

Exposure to HFC-143a is expected to be minimal in view of the methods employed to minimise release of ozone depleting gases to the atmosphere (1). During charging of refrigeration units with refrigerant closed piping is employed. Release of about 0.1 g of refrigerant can normally occur when the flexible hose connectors between the refrigeration system and the cylinder are disconnected at the end of charging. The hoses are fitted with automatic shut off valves which prevent release of the contents of the hose.

Procedures for leak prevention and testing are well established (1). Detection of HFC-143a at 0.1 ppm is possible. It is recommended that leak testing be conducted quarterly on equipment containing in excess of 50 kg of refrigerant.

7. PUBLIC EXPOSURE

Procedures for prevention of release of gas during charging and recharging of refrigeration equipment and testing for leakage are well established and will be practised. As a result, there should be low potential for public exposure during proper work practices.

Disposal practices for this refrigerant will be to recover, reclaim and recycle gas for continued use. It is claimed that any other disoposal recommendations will conform to Australian regulatory guidelines.

8. ENVIRONMENTAL EXPOSURE

. Release

Use

The notified substance will not enter the environment intentionally, but any releases during filling or use of cooling systems, or following disposal of obsolete equipment or recovery of refrigerants therefrom, will rapidly volatilise to the atmosphere. No estimate is provided of likely releases, but commercial systems generally lose less than 10% of working charge per annum. Releases during recharge when hoses are disconnected are estimated at about 0.1 g per operation. The new blends are expensive, providing a financial incentive to minimise losses and install area monitors around large installations.

The Australian Refrigeration and Air Conditioning Code of Good Practice (1) requires that releases of ozone depleting refrigerants to the atmosphere during manufacturing, installation or servicing operations be reduced to the minimum level by re-use of refrigerant recovered. Recovery of refrigerant is required from performance testing during development and production. Refrigerant must be recovered in dedicated cylinders, identified by valving, labelling and colour coding. Where contaminated refrigerants are stored, they must be labelled to indicate the contents. The Code is called up in most State legislation. In

Tasmania, the Code is practically applied, with legislative backing being developed.

Formulation, handling and disposal

As noted above, new blends may be developed following introduction. Blending will be centralised at Lovelocks in Melbourne.

Recovery, reclamation and recycling of refrigerants is preferred to disposal. For disposal, the Code requires that unusable or surplus refrigerant not be discharged to the atmosphere, but be returned to the supplier or stored in a cool shaded place pending disposal. Reprocessing will not occur in Australia as such activities require a production facility. Local disposal will also not occur as acceptable disposal facilities do not exist currently in Australia.

. **Fate**

Given its high volatility, any trifluoroethane released to the environment will partition almost entirely to the atmosphere. The main degradation pathway in the environment is reaction with tropospheric hydroxyl radicals, which abstract hydrogen. The estimated atmospheric lifetime is 72 years (2).

Modelling studies (3) indicate that reaction of HFC-143a with hydroxyl radicals leads via trifluoroacetaldehyde and trifluoromethoxy radical to carbonyl fluoride. Carbonyl fluoride does not react with hydroxyl radicals or undergo photochemical transformation at significant rates in the troposphere. Its fate is somewhat speculative as its Henry's law solubility and reactive sticking coefficient (a measure of the reaction probability on aqueous surfaces) are unknown. However, by using data for phosgene, a tropospheric lifetime of 17 days may be estimated for carbonyl fluoride with respect to incorporation and hydrolysis in clouds (4). Dry deposition may also be important. Tropospheric loss mechanisms may be inferred from the observation of carbonyl fluoride in the stratosphere, but not in the troposphere (5). However, such conclusions are tentative as stratospheric sources of carbonyl fluoride include the common CFCs, CFC-11 and CFC-12.

9. EVALUATION OF TOXICOLOGICAL DATA

9.1 Acute Toxicity

Studies on acute oral toxicity, acute dermal toxicity, skin irritation, eye irritation and skin sensitisation were not conducted. This is acceptable since HFC-143a is a gas.

9.1.1 Inhalation Toxicity (6)

Groups of 6 male Crl:CD BR rats were exposed nose-only for a single 4 hour period to 0, 97,000 or 540,000 ppm HFC-143a. Following termination of treatment, the rats were observed for 14 days.

Rats exposed to the HFC-143a exhibited dry, red ocular and nasal discharges but this was said to be the result of being held in restrainers.

Three rats exposed to 97,000 ppm HFC-143a showed slight weight loss on the day following exposure and 4 rats in the high dose group showed moderate to severe weight loss on the same day.

No mortality was observed during the observation period and no clinical signs of toxicity were observed in either of the exposure groups.

It can be concluded that the 4 hour acute inhalation LC_{50} is greater than 540,000 ppm.

9.2 Repeated Dose Toxicity

9.2.1 Four-Week Repeated Dose Study (7)

Charles River rats (10/sex/dose) were exposed nose only by inhalation to HFC-143a at concentrations of 0, 2,000, 10,000 or 39,000 ppm for 6 hours per day for 20 days over a 31 day period. There was no recovery period.

Male rats exposed to 2,000, 10,000 or 39,000 ppm HFC-143a had statistically significant decreased body weights and body weight gains compared to controls at various intervals during the exposure period but the decreases were not dose-dependent.

A number of functional observations were made to determine an effect of the nervous system. No compound-related neurotoxic effects were observed.

No compound-related effects on haemotology or clinical chemistry were observed.

One male animal in each of the 2,000, 10,000 ppm dose groups and one female animal in the 39,000 ppm dose group was found dead on

test days 8, 9 and 15 respectively. The cause of death was not determined.

There were no statistically significant changes in final body or organ weights in treated groups relative to controls. However, a trend towards decreased absolute testicular weights was present in male rats.

Regarding gross organ changes, small testes were noted in 1/10 and 2/10 male rats in the 10,000 and 39,000 ppm dose groups respectively.

Significant pathological changes were noted in the testes of exposed male rats. Degenerative changes were present at all exposure concentrations. Microscopically, these changes were characterised by minimal to mild accumulation of eosinophilic debris within the lumen of seminiferous tubules. Tubular architecture was generally intact and germ cell necrosis was not prominent. In the epididymes of affected animals, decreased sperm density and increased exfoliated germ cell debris were correlated to the testicular changes. The changes were minimal to mild in the 39,000 and 10,000 ppm dose groups and less severe in the 2,000 ppm dose group where testicular changes were generally very slight and epididymal sperm density was affected in 3/10 animals.

All other microscopic findings noted were considered incidental occurences of spontaneous lesions common to rats on this strain and age.

A possible explanation of the testicular changes advanced was that the rats were inadvertently exposed to excessive heat leading to increased body temperatures during the nose-only exposures

9.2.2 Four-Week Repeated Dose Study (8)

A second four-week repeated dose study was conducted to discover if the testicular changes observed in a previous study (see section 9.2.2 above) could be confirmed. Toxicity evaluations were limited to body weights, clinical signs and anatomic and/or histopathological evaluations of the testes and epididymes.

Charles River rats (10 males/dose) were exposed whole-body to HFC-143a at concentrations of 0, 2,000, 10,000 or 40,000 ppm for 6 hours per day, for 20 days over a 28-day period. One day following the 20th exposure all rats were killed for pathological examination.

Exposed rats did not exhibit any statistically significant changes in body weights or body weight gains compared to controls.

All rats survived to scheduled termination and no compound-related clinical signs were observed in any of the exposed

groups.

No effect of exposure to HFC-143a was observed on testes weights and there were no compound-related changes in gross or microscopic findings.

9.2.3 90-Day Repeated Dose Study (9)

Charles River rats (20/sex/dose) were exposed whole body to 0, 2,020, 10141 or 40,072 ppm HFC-143a , 6 hours per day, 5 days per week for 90 days.

At the conclusion of the 90 day exposure approximately 10 rats per dose group were allowed to recover for approximately one month.

There were no compound-related effects on body weight or body weight gain or food consumption at any exposure concentration during either the exposure period or the recovery period.

During the exposure and recovery periods there were no compound-related effects on clinical signs.

No compound-related deaths were observed. Three rats died or were killed *in extremis*.

No compound-related effects on ocular tissue were observed.

Isolated statistically significant differences in haemotology and clinical chemistry values were within normal ranges and not considered to be biologically significant.

There were no statistically significant or biologically significant differences in organ weights at any exposure concentration at either 90 days or at the end of the one month recovery period.

There were no compound-related gross or microscopic morphological changes to any organ an any exposure concentration at 90 days. In particular, there was no evidence of pathological changes in the testes.

9.3 Developmental Toxicity

9.3.1 Inhalation Developmental Toxicity Study in Rabbits (10)

Three groups of 24 artificially inseminated New Zealand White rabbits were exposed to 0, 2,000, 10,000 or 40,000 ppm HFC-143a by whole-body inhalation for 6 hours on each on 13 consecutive days (gestational days 6-18). A control group of 24 artificially inseminated rabbits was exposed to air. All surviving females were killed on day 29 of gestation for a

scheduled laparohysterectomy.

One animal in the 2,000 ppm dose group spontaneously aborted on day 17 but was not considered compound-related in view of the fact that spontaneous abortions are not uncommon in this species and strain.

No compound-related clinical signs were noted during the study.

No compound-related changes in mean body weights, body weight gains, gravid uterine weights, net body weights or net body weight changes were observed. No compound-related changes in food consumption were observed.

At the scheduled necropsy at day 29, a number of gross organ changes were noted but these were not attributable to HFC-143a including a white precipitate in the amniotic fluid at one implantation site for one 2,000 ppm animal.

Organ weights (kidney, liver and lung) were comparable in the control and exposed groups.

No adverse effects on intrauterine growth or survival were observed at any exposure level. An increased mean number of implantation sites in the 10,000 ppm dose group compared to the control was statistically significant but within the historical control data. One animal in the 2,000 ppm dose group had a completely resorbed litter.

Regarding the foetal morphology, external, soft tissue and skeletal malformations were observed in 4, 14, 5 and 5 foetuses in the control, 2,000, 10,000 and 40,000 ppm dose groups, respectively. The total malformation rate (expressed as per cent per litter) was 3.1%, 8.2%, 3.4% and 7.1% for these same groups respectively which is well within the historical control range for total malformations (0.0 - 12.9%).

9.3.2 Inhalation Developmental Toxicity Study in Rats (11)

HFC-143a was admininstered by inhalation to groups of 25 female Crl:CD BR rats on days 7-16 of gestation (the day copulation was confirmed was termed day 1 of gestation). The target dose levels chosen were 0, 2,000, 10,000 and 40,000 ppm.

All animals survived to scheduled termination on day 22 of gestation. No adverse effects on body weight or body weight gain were observed. No significant effects on clinical signs were noted and no compound-related effects were observed during gross postmortem examinations.

No significant dose-related effects on reproductive parameters (early deliveries, incidence of dams with total resorptions, litter means for live, dead or resorbed foetuses or mean corpora lutea) were detected.

Regarding effects on the foetus, no significant effects on mean foetal weights were observed. No compound-related effects on the incidence of foetal malformations were detected.

The mean percent of affected foetuses examined for variations due to retarded development during the visceral examination was significantly increased for all test groups. The incidences were 1.6, 10.5, 8.7 and 10.0 percent for the 0, 2,000, 10,000 and 40,000 ppm dose groups, respectively. Retarded renal papillary development was the primary and most frequently recorded observation for this category. However, it was concluded that these effects were not biologically significant because the control value was abnormally low, the increases were not dose-dependent and there was no other evidence of developmental toxicity.

9.4 Genotoxicity

9.4.1 Bacterial Reverse Mutation Assay (12)

The effect of the notified chemical on back mutation to prototrophy was tested in *Salmonella typhimurium* strains TA 98, TA 100, TA 1535, TA 1537 and TA 1538 and in *Escherichia coli* strain WP2 *uvrA* both in the presence and the absence of metabolic activation provided by rat liver S9.

Agar plates seeded with the tester strains were exposed to the notified chemical in the vapour phase at nominal concentrations up to 100% v/v.

Negative controls were within acceptable limits. Positive controls of dichloromethane (in vapour phase), benzo[a]pyrene (BaP), 2-nitrofluorene, 2-aminoanthracene (2AA), 9-aminoacridine (9AA), N-ethyl-N'-nitro-N-nitrosoguanidine (ENNG) and sodium azide gave the expected increases in mutant yields. In the absence of S9, dichloromethane was tested on all strains, sodium azide on TA 1535 and TA 100, 9AA on TA 1537, 2-nitrofluorene on TA 1538 and TA 98 and ENNG on WP2 *uvrA*. In the presence and absence of S9, 2AA was tested on TA 1535 and WP2 *uvrA* and BaP on TA 1537, TA 1538 and TA 100.

HFC-143a did not increase the level of back mutation in any strain at any dose level.

It can be concluded that HFC-143a is not genotoxic as measured by this assay.

9.4.2 Micronucleus Assay in the Bone Marrow Cells of the Mouse (13)

Groups of 10 male and 10 female CD-1(ICR)BR mice were exposed to target concentrations of 0, 2,000, 10,000 or 40,000 ppm HFC-143a for approximately 6 hours/day on 2 consecutive days. Groups of 5 males and 5 females from each negative control and treated group

were killed approximately 24 and 48 hours after the final exposure. The positive control group, consisting of 5 male and 5 female mice were treated with 20 mg/kg cyclophosphamide (CP) of 2 consecutive days (during which time they were sham-exposed to air) and killed approximately 24 hours after the second exposure.

No significant clinical signs were observed as a result of treatment.

One thousand polychromatic erythrocytes (PCEs) per animal were scored for the presence of micronuclei. No statistically significant increases in micronucleated PCEs were observed in male or female mice at either the 24 or 48 hour time points. As expected, CP-treated males and females showed significant increases in micronucleated PCEs compared to controls. Treatment did not change the ratio of PCEs to normochromatic erythrocytes.

It can be concluded that HFC-143a is not genotoxic as measured by this assay.

9.5 Cardiac Sensitisation in Dogs (14)

The effect of intravenous injection of beagle dogs with adrenaline before and during inhalation of HFC-143a on the electrocardiogram was studied.

Optimal doses of adrenaline were chosen on the basis of the number of ectopic beats and ranged from 2 - 12µg/kg.

The concentration of HFC-143a in the air supply ranged from 5 - 30% (v/v). Postive responses were observed only at 30% HFC-143a in 2/5 dogs.

9.6 Overall Assessment of Toxicological Data

HFC-143a is of low acute inhalation toxicity in rats.

Repeated dose studies suggest that HFC-143a does not exhibit toxic effects in rats exposed by inhalation for up to 90 days. Some effects on the testes of exposed male rats were observed in a 4-week study. However, these results were unrepeatable in 2 further studies and it was suggested that the rats were inadvertently overheated during the exposure period.

Developmental toxicological studies with dosing of females did not reveal any effects on foetal development in either rats or rabbits.

HFC-143a was found not to be genotoxic in the bacterial reverse mutation and mouse micronucleus assays.

HFC-143a was found to induce cardiac sensitisation in dogs at a dose of 30% v/v.

10. ASSESSMENT OF ENVIRONMENTAL EFFECTS

Testing was conducted on rainbow trout (15) and *Daphnia magna* (16). The former used flow-through conditions, with end-points expressed as mean daily measured concentrations. A single fish died in one replicate at 2.5 mg.L-1, and in each replicate at 40 mg.L-1. The daphnid test used static, unaerated conditions, with results expressed as mean measured concentrations at 0 and 48 h. The EC50 was 300 mg.L-1. Results indicate that HFC-143a is practically non-toxic to aquatic fauna.

Chronic effects on aquatic fauna and acute effects on algae are not anticipated in view of the limited persistence and absence of significant toxic effects in the tests described above.

Halocarbon refrigerants can affect the atmosphere. HFC-143a contains neither chlorine nor bromine, and thus will not act as a source of ozone depleting halogen radicals in the stratosphere. Scientists from the US National Oceanic and Atmospheric Administration concluded recently that hydrofluorocarbons have negligible potential to destroy ozone (17).

Like other halocarbons, HFC-143a makes a positive contribution to the global warming potential of the atmosphere. Its atmospheric lifetime of 72 years is less than reported (18) for CFC-115 (400 years) but longer than for HCFC-22 (15.3 years). The estimated global warming potential (GWP) for HFC-143a over a 100 year time horizon (relative to CO2 with GWP 1) is 2900, intermediate between those for HCFC-22 and CFC-115 (1500 and 6900, respectively - reference 19). The other components of SUVA HP62 have GWPs of 2500 (HFC-125) and 1200 (HFC-134a). Since R-502 contains roughly equal amounts by weight of HCFC-22 and CFC-115, its replacement by SUVA HP62 should entail a slight easing of the global warming potential.

11. ASSESSMENT OF ENVIRONMENTAL HAZARD

HFC-143a is not expected to exert a direct effect on living organisms as it has minimal biological activity, as evidenced by results from aquatic toxicity testing. The high volatility should ensure minimal exposure of aquatic and terrestrial compartments, and therefore minimal hazard to organisms inhabiting them.

Hazard to the atmosphere will be reduced when SUVA HP62 replaces R-502, as the replacement refrigerant will not carry chlorine or bromine to the stratosphere and has a slightly lower global warming potential.

12. ASSESSMENT OF PUBLIC AND OCCUPATIONAL HEALTH AND SAFETY EFFECTS

Animal tests suggest that HFC-143a is unlikely to exhibit toxic effects to individuals exposed by inhalation either acutely or to repeated doses. The acute studies described above extend limited data previously published (20) in which no mortality was observed in mice exposed for 2 hours to 500,000 ppm.

Based on developmental toxicity studies, HFC-143a is not expected to have effects on the developing foetus.

The data on genotoxicity described above suggest that HFC-143a is not genotoxic. However, there is evidence in the literature that HFC-143a is mutagenic in *Salmonella typhimurium* strains TA 1535 and TA 100 (21). In the same study HFC-143a was negative for transformation of hamster kidney cells and was not carcinogenic in Wistar rats dosed at 300 mg/kg for 52 weeks by gavage. The genotoxic potential of HFC-143a remains uncertain.

HFC-143a induces cardiac sensitisation in dogs though at higher doses than refrigerants it is designed to replace. For example, the lowest dose at which HFC-143a induces cardiac sensitisation is 60 times higher than for CFC-11.

Exposure to HFC-143a during charging or recharging refrigeration equipment is expected to be minimal in view of the well established procedures to minimise release of ozone-depleting gases to the atmosphere (1).

From the above considerations, the risk of adverse health effects resulting from the use of HFC-143a is low.

Although pure HFC-143a is highly flammable, the mixture (SUVA HP62) that is used to charge refrigeration equipment is not. Nevertheless, contact of the refrigerant with hot surfaces or open flames should be avoided because of the potential for release of hydrogen fluoride and carbonyl halides.

A possible hazard from spills of liquid SUVA HP62 contained in gas cylinders is its potential to cause frostbite.

13. RECOMMENDATIONS

To minimise occupational exposure to HFC-143a the following guidelines and precautions should be observed:

. those taking sympathomimetics, bronchodilators or cough and cold medications should have their medication evaluated by their medical adviser, if exposure to the notified chemical is likely;

. the code of practice (1) governing reduction of emissions of ozone depleting refrigerants should be adhered to;

. charging and recharging of refrigeration equipment should be conducted in well ventilated areas;

. if engineering controls and work practices are insufficient to reduce exposure to HFC-143a to a safe level, then personal protective devices which conform to and are used in accordance with Australian Standards (AS) for eye protection (in this case a face shield) (AS 1336, AS 1337) (22,23), respiratory protection (24), thermal gloves (AS 2161) (25) and protective clothing (AS 3765.1, 3765.2) (26,27) should be worn;

. a copy of the Material Safety Data Sheet should be easily accessible to employees.

14. MATERIAL SAFETY DATA SHEET

The attached Material Safety Data Sheets (MSDS) for HFC-143a and SUVA HP62 were provided in Worksafe Australia format (28).

These MSDS were provided by Du Pont (Australia) Ltd as part of their notification statement. The accuracy of this information remains the responsibility of Du Pont (Australia) Ltd.

15. REQUIREMENTS FOR SECONDARY NOTIFICATION

Under the *Industrial Chemicals (Notification and Assessment) Act 1989*, as amended (the Act), secondary notification of HFC-143a shall be required if any of the circumstances stipulated under subsection 64(2) of the Act arise. No other specific conditions are prescribed.

16. REFERENCES

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