SIDS Initial Assessment Report For SIAM

1. Chemical Name	1,1,1-trifluoroethane
2. CAS No	420-46-2
3. Sponsor Country	Oscar Hernandez Director, Risk Assessment Division (7403M) U.S. Environmental Protection Agency 1200 Pennsylvania Ave., NW Washington, DC 20460 Phone: 202-564-7641 e-mail: **hernandez.oscar@epa.gov**
4. Shared Partnership With	Honeywell
5. Roles/Responsibilities of the Partners	
- Name of industry sponsor/consortium	George Rusch Honeywell International, Inc. 973-455-3672 george.rusch@honeywell.com
- Process used	Honeywell produced these documents; EPA reviewed the documents.
6. Sponsorship History	The chemical was brought forward through the ICCA.
7. Review Process Prior to the SIAM	The U.S. EPA reviewed this case.

SIDS INITIAL ASSESSMENT REPORT
1,1,1-TRIFLUOROETHANE
CAS No. 420-46-2

1. IDENTITY

1.1 **Identity** (6, 8, 9, 11, 23, 24, 25)

Chemical: 1,1,1-Trifluoroethane
CAS No. 420-46-2
Molecular Weight: 84
Molecular Formula: $C_2F_3H_3$
Structural Formula: CH_3CF_3
Synonyms: HFC-143a
Ethane, trifluoro-
Methyl fluoroform
FC-143a
R-143a

Physical Properties
Physical State: Gas
Vapor Pressure: 12620-12720 kPa @ 25˚C
Boiling Point: -47.4 °C @101.3 kPa
Melting Point: -111.3 °C
Log K_{ow}: 1.74 @ 20 °C
Water Solubility: 761 mg/L @ 25 °C
Henry's Law Constant: No Data

2. GENERAL INFORMATION ON EXPOSURE

Worldwide production is estimated at 10,000 to 50,000 tons in the year 2006.

The major application is in air conditioning systems and commercial refrigeration. (19)

2.1 Environmental Exposure and Fate

The low octanol/water partition coefficient (log K_{ow} = 1.74) indicates a low potential for bioaccumulation. The low water solubility (761 mg/L at 20°C) and high vapor pressure (1262-1272 kPa at 25°C) suggest it should migrate to the air. Modeling, using the Level III Fugacity model, indicates that when the majority of the chemical is released to air, over 99.9 percent will remain in the atmosphere. Photodegradation occurs primarily with hydroxyl radicals, resulting in a half-life of approximately 9,600 days. (15, 21, 25)

A photochemical trajectory model has calculated a **photochemical ozone creation potential (POCP)** of 0.0 for HFC-143a, thus HFC-143a should make a negligible contribution to photochemical ozone production. (29)

A two-dimensional chemical-radiative-transport model of the global atmosphere determined the atmospheric lifetime of HFC-143a to be 47.2, which is appreciably lower than 53.5 value previously reported by WMO (1999). The difference in values appears to be the result from the slow, but not trivial loss in the stratosphere. The direct **global warming potential (GWP)** of HFC-143a at 20-, 100- and 500 year time horizons was calculated to be 5,695, 4,352, and 1,537, respectively. The model took into account evaluated atmospheric lifetimes and radiative forcings. The percent difference in GWP for HFC-143a for the 100-year time horizon in this study (4,352) was 19% less than that reported (5,400) by the WMO (1999). (30,31))

2.2 Exposure to Humans

HFC-143a is a gas, and therefore, exposure will be mainly by inhalation. Exposure may occur during synthesis, shipping, or filling refrigeration equipment. As HFC-143a is a gas, it is used in sealed systems.

The Workplace Environmental Exposure Level for HFC-143a is 1000 ppm as an 8-hour time-weighted-average (1).

HFC-143a is not used in any commercial products. Therefore the general public should not be exposed to it.

3. HUMAN HEALTH HAZARDS

3.1 Effects on Human Health

3.1.1 Metabolism

Groups of 3 male Charles River CD BR rats were exposed to HFC-143a for 4-5 hours in a recirculating chamber at levels of 100, 390, 1040, 2050, 4800 and 40,000 ppm. The chamber concentrations were monitored by gas chromatography every 10 minutes. Partition coefficients between air and blood, fat, liver, and muscle were measured. At the end of the exposure, samples of urine were collected and examined for the presence of metabolites by 19F NMR. The partition coefficients were 0.66 for blood:air, 1.13 for liver:air, 1.04 for fat:air, and 0.99 for muscle:air. Analysis of the gas uptake data with a PBPK model suggested that there was measurable metabolism of HFC-143a. The estimated Vmax and Km were 4.15 ± 0.1 mg/hr-kg and 4.37 ± 0.18 mg/L, respectively. Based on a separate exposure study at 40000, exposure appears to be saturated between 20000 and 40000 ppm. Based on 19F NMR, the major metabolite in the urine was trifluoroethanol and minor metabolites identified included the glucuronide conjugate of trifluoroethanol, trifluoroacetic acid, trifluoroacetaldehyde and the urea conjugate of trifluoroacetaldehyde. These metabolites were all identified in urine from rats exposed to 40000 ppm of HFC-143a for 4-hours. No metabolites were identified in the urine from rats exposed to 4800 ppm or lower. (13)

In a second study, male Wistar rats were individually exposed by inhalation for 4 hours at levels of 5500, 10,000, 16,000, 20,000, or 30,000 ppm. The concentration of the test substance in the exposure chamber was monitored by gas chromatography. The amount of chemical retained in the body was calculated from measurements of the initial and final concentration in the chamber. A physiologically-based pharmacokinetic computer modeling program was used to calculate the kinetic parameters of absorption and metabolism of the test article. A significant decrease in liver glutathione was seen in the rats exposed to concentrations ≥ 10000 ppm. (15)

Nine occupationally exposed male volunteers were exposed to 500 ppm of HFC-143a for two hours during light physical exercise in an exposure chamber. Blood, urine, and exhaled air were collected before, during and up to two days after the exposure period. The presence of HFC-143a in the biological samples was analyzed by head-space gas chromatography. Trifluoroacetic acid and fluoride in the urine were analyzed using high performance liquid chromatography and an ion specific electrode, respectively. The study results showed a low metabolic rate and a low solubility in blood for HFC-143a. A plateau blood concentration of 1.4 µg/g was found. Two elimination phases were observed with half lives of 4 and 300 minutes. (10)

3.1.2 Acute Toxicity

Groups of 6 Charles River CR BR male rats were exposed; nose only, to concentrations of 97000 or 540000 ppm of the test article for a single 4 hour period. They were then observed for 14 days following the exposure. During the exposure, oxygen concentrations in the breathing air were maintained at 20% by the addition of supplemental oxygen to the chamber supply air. The exposure levels were monitored at half-hourly intervals using an on-line gas

chromatograph with a flame ionization detector. There was no lethality. Slight body weight loss was noted for the 97000 ppm exposure level group and a more severe body weight loss was noted for the rats in the 540000 ppm exposure level group on the day following the exposure. By the second day post exposure, body weight gain was normal. (4, 7)

The acute toxicity of HFC-143a was assessed by exposing two groups of 5 male and 5 female Sprague Dawley rats nose only to either 305000 or 591000 ppm of the test article for 4 hours. An additional control group was exposed to air for 4 hours. The test atmospheres were generated by dilution of the test gas with air and oxygen in such a manor that the oxygen level was maintained between 19-21%. The exposure level was determined 5 times during the 4-hour exposure. The analysis consisted of collection of a sample using a gas sampling bulb. Samples were withdrawn using a gas tight syringe and analyzed on a gas chromatograph using a thermal conductivity detector. Rats were observed for 14 days following the exposure. There was no lethality. Increased respiratory depth observed at 350,000 in one male and one female and 591,000 ppm in one female. Peripheral vasodilation was reported at 591,000 ppm in one male and four females. Aside from a slight decrease noted on the day following the exposure, body weight and body weight gain appeared normal. The slight loss on the day following the exposure was attributed to the restraint system used for the exposures. There was no adverse effect on organ weight nor were there any gross observations of treatment related abnormalities seen during the gross necropsy. (5)

To determine if HFC-143a caused sensitization of the heart to epinephrine, groups of 5-6 beagle dogs that were previously trained to accept a canvas sling restraining device were exposed to atmospheres containing 50000, 100000, 150000, 200000, 250000 and to 300000 ppm of the test article. Exposures were conducted using a single pass face mask. During the exposure, oxygen concentrations in the breathing air were maintained at 20% by the addition of supplemental oxygen to the breathing air. Electrocardiogram leads were attached to the dog and the EKG was recorded for the 17 minutes of the test. Two minutes after breathing air alone, the dog was given an injection of epinephrine just below that which could have caused a spontaneous arrhythmia. After five additional minutes of exposure to air, the dog was exposed to vapors of HFC-143a. After 5 minutes of the exposure to the HFC-143a, the dog was given a second injection of epinephrine at the same dose as the first injection. Exposure to HFC-143a was continued for an additional 5 minutes. A positive response was the development of an arrhythmia or at a minimum 5 consecutive ectopic heart beats. The threshold for this effect was 300,000 ppm. (4)

As the HFC-143a is a gas at room temperature, skin and eye irritation and dermal sensitization data are not available.

3.1.3 Repeated-Dose Toxicity

Following a 4-week inhalation toxicity study that was judged to be unreliable due to technical problems (27), groups of male Charles River CD BR rats were exposed whole body to levels of 2,000, 10,000, and 40,000 ppm, 6-hrs/day 5-days/wk for 4 weeks. An air exposed control group was also included. The exposure levels were monitored at half-hourly intervals using an on-line gas chromatograph with a flame ionization detector. There was no mortality, no effects on body weight and no adverse clinical signs. Gross pathology and histopathology were limited to the testes and epididymides. Changes related to exposure were not observed in any group. Therefore 40,000 ppm represented the No Observed Effect Level. (4, 28)

Twenty male and 20 Charles River CD BR female rats were exposed to concentrations of 0, 2000, 10000 or 40000 ppm of the test article. The exposure levels were monitored at half-hourly intervals using an on-line gas chromatograph with a flame ionization detector. On the day following the last exposure, blood and urine samples were collected and the standard serum chemistry, hematology and urinalysis evaluations were conducted on all animals. Approximately 35 tissues were collected form each animal. They were examined histopathologically only from animals in the control and high level exposure group Also at termination, samples of liver were collected from 5 rats per group for determination of ß-oxidation activity a measure of peroxisome proliferation. There were no effects on body weight and food consumption. There were no clinical signs. There were no effects on ophthalmological examinations, hematology, biochemistry and urinalysis. There were no changes in gross pathology and histology. There was no proliferation of hepatic peroxisomes. It was concluded that the NOEL for this study was 40000 ppm. (4, 17)

3.1.4 Genetic Toxicity

Several bacterial mutation assays were conducted with HFC-143a. In the first, strains TA 100 and TA 1535 were exposed for 48 hours to a level of 500,000 ppm of HFC-143a, both with and without metabolic activation. HFC-143a gave a positive response in this assay. However, as discussed below, this is the only report of a positive response with HFC-143a and the weight of evidence demonstrates that HFC-143a is not mutagenic.

In a second study, TA 100, TA 1535, TA 97, TA 98, and e coli WP2 uvrA were exposed to 5,000, 15,000, 25,000 and 35,000 ppm both with and without metabolic activation. Again the cells were incubated for 48 hours at 37 ˚C. HFC-1434a was not active in this assay. In a third assay, TA 100, TA 1535, TA98, TA 1537, TA 1538 and e coli WP2 uvrA were exposed to levels of 100,000, 300,000, 500,000, 700,000, or 900,000 of HFC-143a for 48 hours at 37˚C both with and with out metabolic activation. Again, HFC-143a was not mutagenic. (2, 4, 14, 18)

It was reported that HFC-143a was not active when tested for mutagenicity using baby hamster kidney fibroblasts (BHY21). However the details of this study were not available. (14)

A chromosome aberration study was conducted in which human lymphocytes were exposed to 5,000, 15,000, 25,000 or 35,000 ppm for 3 hours at 37˚C both with and with out metabolic activation. Treatment was conducted by exposing the cell cultures to atmospheres containing the test article at the specified concentrations. The maximum exposure was set to be approximately 50% of the lower flammability level for the test article. HFC-143a was not active in this test. (3, 4)

Charles River CD-1 mice were exposed, whole body, to the test article, 6-hrs/day for two consecutive days at levels of 2,000, 10,000 and 40,000 ppm. Bone marrow smears were prepared approximately 24 and 48 hours after the second exposure, and 1000 polychromatic erythrocytes per animal were evaluated for the presence of micronuclei. The positive control was cyclophosphamide. The exposures were conducted on days 49 and 50 of the 13-week rat exposure study described above. Again HFC-143a was not active in this assay. (4, 22)

3.1.5 Reproductive Toxicity

HFC-143a has not been evaluated in a reproduction study. However, it has been tested in a 13 week subchronic inhalation toxicity study with exposures up to 40,000 ppm, 6-hours/day, 5 days/week and included full histopathological evaluation of the tissues from the reproductive organs (testes, epididymides, ovaries, and uterus). None of these studies reported any adverse findings in the reproductive organs. (4, 13)

3.1.6 Developmental Toxicity

Groups of 25 pregnant Charles River CD BR rats were exposed to 2,000, 10,000, or 40,000 ppm of HFC-143a 6 hours/day from day 7 through day 16 of gestation. An air control was also included. Exposure levels were monitored using a gas chromatograph with a flame ionization detector. The day of copulation was designated as Gestation Day 1 (GD 1). On GD 20, the maternal animals were killed by CO2 inhalation. The ovaries and uteri were examined for the number of corpora lutea, live young, embryofetal deaths and resorptions. The uterus of each apparently nonpregnant rat was opened and stained with ammonium sulfide. Fetuses were weighed, the sex was determined and the fetuses were examined for external malformations. Live fetuses were killed and examined for visceral malformations by the method of Staples. There were no significant findings on maternal mortality, clinical observations and gross pathology examinations. Treatment related findings were not observed in litter size, embryo-fetal loss and litter and fetal weight. Effects on the incidence of malformation were not observed at any exposure level. There was however a slight, but significant increase in the incidence of fetal visceral variations in the litters of all the exposed groups in comparison to

the control group. Since there was no evidence of any other developmental toxicity, the increased incidences were not dose-dependent and fell into the historic average of the incidence for variations of this effect, they were not considered to be biologically significant. (4, 20)

Groups of 24 pregnant New Zealand white rabbits were exposed to 0 (control), 2,000, 10,000, or 40,000 ppm of HFC 143a 6-hours/day from day 6 till day 18 of gestation. Exposure levels were monitored using a gas chromatograph with a flame ionization detector. Does were artificially inseminated and subsequently injected (iv) with 100 IU of hCG. The day of insemination was considered Gestation Day 0 (GD0). On GD 29, the maternal animals were killed by iv injection of Socumb Euthanasia solution and the internal organs examined for gross abnormalities. The ovaries and uteri were examined for the number of corpora lutea, live young, embryofetal deaths and resorptions. The uterus of each apparently nonpregnant rabbit was opened and stained with ammonium sulfide. Fetuses were weighed, the sex was determined and the fetuses were examined for external malformations. Live fetuses were killed and examined for viseral malformations by the method of Staples. One female in the 2000 ppm group aborted on gestation day 17. No other findings were observed in adult females within the scheduled period, thus the abortion was judged spontaneous. There was no indication of developmental toxicity at any exposure level. Fetal malformation and variation incidences were similar among all the groups. (4, 12)

3.1.7 Experience with Human Exposure

Nine male volunteers were exposed to 500 ppm HFC-143a for 2 hours for a human toxicokinetic study. The electrocardiogram of the exposed volunteers was monitored during and until 20 hours after exposure. Irritative and central nervous system symptoms were rated in a questionnaire prior to, during and after exposure. The authors stated that analyses suggested no increase in symptoms ratings during or after exposure. (10)

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3.2 Initial Assessment for Human Health

HFC-143a has a very low mammalian toxicity. A 4-hour exposure of 591,000 ppm did not result in lethality. It did not cause any significant signs of toxicity in a 13 week inhalation toxicity study with exposures up to 40,000 ppm. It also did not cause any developmental effects in either rats or rabbits with exposures during gestation up to 40,000 ppm. Based on 19F NMR, the major metabolite in the urine was trifluoroethanol and minor metabolites identified included the glucuronide conjugate of trifluoroethanol, trifluoroacetic acid, trifluoroacetaldehyde and the urea conjugate of trifluoroacetaldehyde. These metabolites were all identified in urine from rats exposed to 40000 ppm of HFC-143a for 4-hours. No metabolites were identified in the urine from rats exposed to 4800 ppm or lower. Volunteers exposed to 500 ppm for 2-hours did not show or report any adverse effects. The current occupational exposure guideline (WEEL) is 1000 ppm.

4. HAZARDS TO THE ENVIRONMENT

4.1 Aquatic Effects

Since HFC-143a will reside in the atmosphere as indicated by a high vapor pressure and limited water solubility, only a few aquatic toxicity tests have been conducted. An acute flow-through toxicity study in the freshwater fish *Oncorhyncus mykiss* estimated the 96-hour LC_{50} to be > 40 mg/l, the highest concentration evaluated. (32) A static acute toxicity study in the freshwater invertebrate *Daphnia magna* estimated the 48-hour EC_{50} to be 300 mg/l. estimates for the toxicity of HFC-143a have been calculated with the ECOSAR program in the EPIWIN model. For fish, the 96-hr LC_{50} was estimated to be 109 mg/L; for daphnids, the 48-hr EC_{50} was estimated to be 115 mg/L; and for green algae, the 96-hr EC_{50} was estimated to be 71 mg/L. (16)

4.2 Terrestrial Effects

No data were found on terrestrial effects.

4.3 Initial Assessment for the Environment

Due to the physicochemical properties of HFC-143a, its use in closed systems, and its predicted partitioning to air, environmental concentrations in the aquatic environment are expected to be low. (26)

5. CONCLUSIONS AND RECOMMENDATIONS

5.1 Human Health

In acute and repeated-exposure inhalation toxicity studies, HFC-143a has been shown to have a low toxicity and low rate of metabolism. In two teratology studies, it was not embryo- or fetotoxic nor did it produce teratogenicity. Also, evaluation of the reproductive organs from a subchronic inhalation exposure study did not reveal any effects on these organs. While showing a positive response in an older Ames assay, HFC-143a was not mutagenic in a series of subsequent *in vitro* or *in vivo* genotoxicity studies.

Because HFC-143a has no applications in consumer products, consumer exposure is not expected. The occupational exposure limit of 1000 ppm has been in place for over 10 years.

5.2 Environment

HFC-143a is used in closed systems. It also has a water solubility of 761 mg/L and a log K_{ow} of 1.74. It is predicted to degrade in the atmosphere with a half-life of approximately 9,600 days based on reaction with hydroxyl radicals. As HFC-143a does not contain chlorine or bromine, it has no potential for stratospheric ozone depletion. Its high vapor pressure, low log K_{ow} and low water solubility imply that it will not be an environmental hazard in the aquatic environment. Finally, releases to the environment are expected to be low.

5.3 Recommendations

Human Health: The chemical is currently of low priority for further work. HFC-143a has not shown signs of acute toxicity even at a level of 591,000 ppm. Due to its flammability, repeat exposure studies were conducted at levels of 40,000 ppm and lower. There has been no evidence for toxicity in these studies.

Environment: The chemical is currently of low priority for further work. Modeling has predicted that it will partition almost exclusively into the air and its low log Pow suggests that it will not bioaccumulate.

6 REFERENCES

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