1,1,1,2-Tetrafluoroethane; CASRN 811-97-2

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR 1,1,1,2-Tetrafluoroethane

File First On-Line 08/01/1995

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	not evaluated	
Inhalation RfC (I.B.)	yes	08/01/1995
Carcinogenicity Assessment (II.)	not evaluated	

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — 1,1,1,2-Tetrafluoroethane
CASRN — 811-97-2
Primary Synonym — HFC-134a

Not available at this time.

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — 1,1,1,2-Tetrafluoroethane
CASRN — 811-97-2
Primary Synonym — HFC-134a
Last Revised — 08/01/1995

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

NOTE: ****SEE BENCHMARK CONCENTRATION IN DISCUSSION. Discussion of the benchmark dose can be found in the Discussion of Principal and Supporting Studies Section.

I.B.1. Inhalation RfC Summary

Critical Effect	Exposures*	UF	MF	RfC
Leydig cell hyperplasia **Rat Chronic Inhalation Study** **Collins et al., 1995**	Benchmark Concentration: See Conversion Factors and Assumptions and Principal and Supporting Studies	100	1	8E+1 mg/cu.m

* Conversion Factors and Assumptions: MW = 102. The Benchmark Concentration (BMC) associated with a 10% extra risk in the critical effect was determined to be 11,030 ppm =

BCM10. Assuming 25 C and 760 mmHg, BMC10 (mg/cu.m) = BMC10 (ppm) x MW/24.45 = 46,000. BMC10(ADJ) = 46,000 x 6 hours/24 hours x 5 days/7 days = 8200 mg/cu.m. The BMC10(HEC) was calculated for a gas:extrarespiratory effect assuming periodicity was attained. Because the b:a lambda values are unknown for the experimental animal species (a) and humans (h), a default value of 1.0 is used for this ratio. BMC10(HEC) = 8200 x [b:a lambda(a)/b:a lambda(h)] = 8200 mg/cu.m.

I.B.2. Principal and Supporting Studies (Inhalation RfC)

Collins, M.A., G.M. Rusch, F. Sato, P.M. Hext and R.J. Millischer. 1995. 1,1,1,2-Tetrafluoroethane repeat exposure inhalation toxicity in the rat, developmental toxicity in the rabbit, and genotoxicity in vitro and in vivo. Fund. Appl. Toxicol. 25: 271-280.

A 2-year inhalation exposure study with 1,1,1,2-tetrafluoroethane (HFC- 134a) was conducted (Collins et al., 1995; Hext and Parr-Dobrzanski, 1993), and the interim report described results through 52 weeks (Collins et al., 1995; Hext and Mould, 1991). In this study, groups of 85 Wistar-derived rats/sex were whole-body exposed to 0, 2500, 10,000, or 50,000 ppm (0, 10,400, 41,700, and 208,600 mg/cu.m) HFC-134a (99.8% pure) for 6 hours/day, 5 days/week (duration-adjusted concentrations = 1860, 7450, or 37,250 mg/cu.m, respectively). The animals were observed for clinical signs of toxicity at least once daily and several times during exposure. Body weight and food consumption were monitored weekly during the first 14 weeks and biweekly thereafter. Hematological, clinical chemistry, and urinalysis evaluations were conducted on 10 rats/sex/group at 14, 27, 52, and 104 weeks. Ten rats/sex/group were sacrificed after 1 year of exposure for gross and microscopic tissue examination of over 40 tissues, including the nose, trachea, and lung. The remaining animals were examined after 104 weeks of exposure. The HFC-134a vapor was generated by evaporating the liquid test material in a stream of metered air. The test atmosphere concentration was assayed at 1-hour intervals by gas chromatography. Mean daily chamber concentrations were found to be within 90-110% of the nominal concentrations.

There were no exposure-related effects on mortality, clinical signs, food consumption, body weight, behavior, or ocular characteristics. In males, slight concentration-related decreases were noted in hemoglobin, hematocrit, RBCs and WBCs after 14 weeks of exposure (but not after 27, 53, or 104 weeks). Slightly elevated plasma glucose was noted in males at week 14, but not in succeeding weeks, and in females at week 27 (but not weeks 52 or 104). There were no treatment-related effects on plasma cholesterol or triglycerides. In animals necropsied after intercurrent deaths (too few per group for meaningful statistical comparisons) and in the 10 animals killed after 52 weeks, there were no treatment-related effects on organ weight or histopathology. The only treatment-related effects after 104 weeks of exposure were found in the testes. A statistically significant increase in absolute and relative testes weight was found at the terminal sacrifice (n = 75, relative weights were 0.6, 0.59, 0.61, and 0.66 in the 0-, 2500-,

10,000-, and 50,000-ppm groups, respectively). In addition, there was a significant increase in the incidence of Leydig cell hyperplasia (incidence of 27, 25, 31, and 40 in the 0-, 2500-, 10,000-, and 50,000-ppm groups, respectively). The testicular effects were considered adverse. This study establishes a LOAEL of 50,000 ppm [LOAEL(HEC) = 37,250 mg/cu.m] and a NOAEL of 10,000 ppm [NOAEL(HEC) = 7450 mg/cu.m].

DERIVATION OF A BENCHMARK CONCENTRATION (BMC): An analysis of the testicular effects using the benchmark concentration (BMC) approach was performed using the data reported by Collins et al. (1995). The benchmark response for Leydig cell hyperplasia was defined as a 10% extra risk of response [extra risk = P(d)-P(0)/P(0)] = BMC10. Because of the high background, a substantial difference in the estimates of benchmark concentration occurs for extra vs. additional risk models (65,000 vs. 46,000 mg/cu.m). An extra risk model was selected as most appropriate, based on the conservative assumption of independence of mechanisms causing the background and treatment-related responses. Dose-response models for dichotomous data using a polynomial (multistage) and a Weibull form and either including a model parameter for a background intercept or not (sometimes referred to as a threshold parameter) were applied. Using any of these four model forms, an excellent model fit was obtained, and the BMC estimates were the same after rounding to two significant figures. The BMC for a 10% extra increase in Leydig cell hyperplasia is 46,000 mg/cu.m [BMC10(HEC) = 8200 mg/cu.m]. For the other endpoint of concern in this study (increase in relative testes weight), the increase was apparent only in the high-concentration group, and the BMC estimated using the polynomial model for continuous data and a benchmark response of 10% change was 107,000 mg/cu.m.

I.B.3. Uncertainty and Modifying Factors (Inhalation RfC)

UF — The uncertainty factor of 100 reflects a factor of 10 to protect sensitive individuals, 3 for interspecies extrapolation, and 3 for database deficiencies, including the lack of a chronic study in a second species and a two-generation reproductive study. A full factor of 10 was not considered necessary for database deficiencies because rats and mice have been shown to respond similarly to several hydrofluorocarbons and in vitro studies show similar metabolism of HFC-134a in human and rodent liver tissue; therefore, a chronic study in a second species is considered unlikely to result in substantial changes of the database.

MF — None

I.B.4. Additional Studies/Comments (Inhalation RfC)

A 90-day toxicity study (Hext, 1989) was performed on 5-week old male and female Wistar-derived rats, 20/sex/group, which were exposed to 0, 2000, 10,000, or 50,000 ppm (0, 8340, 41,700, or 208,600 mg/cu.m) 99.97% pure HFC- 134a for 6 hours/day, 5 days/week (duration-

adjusted concentrations = 0, 1500, 7500, or 37,500 mg/cu.m). Ten animals per group were examined at the end of a 13-week exposure, and 10 were retained after the last exposure for a 4-week observation (recovery) period. There were no clinical signs of toxicity and no significant effect on food consumption or body weight gain. There were no treatment-related effects on serum chemistry, hematology, or urinalysis parameters. A statistically significant decrease in plasma triglycerides was observed in both sexes after 13 weeks of study in the recovery group, but not in the group examined after 13-week exposures, and appears to be due to unusually high values in the control groups. A statistically significant decrease in absolute brain weights was noted in the 10,000 and 50,000-ppm females that were killed at 90 days. The brain weight changes are not considered to be toxicologically significant because the difference compared with controls is maximally 2.5% and because a reanalysis of relative brain weight data by the International Pharmaceutical Aerosol Consortium for Toxicology Testing (1990) showed that there was no statistically significant difference in either sex at any concentration. Neither absolute nor relative weights of other organs were changed. There were no significant microscopic changes attributable to exposure. Thus, the NOAEL for this study is 50,000 ppm [NOAEL(HEC) = 37,250]. A LOAEL was not identified in this study.

A 14-day study (Silber, 1979a), in which 10 male rats/group were exposed to air or HFC-134a at a target concentration of 100,000 ppm (417,000 mg/cu.m), 6 hours/day, 5 days/week (duration-adjusted concentrations = 74,500 mg/cu.m), showed no compound-related changes in survival, body weight, hematology, blood chemistry, gross necropsy, or histopathology. The only clinical sign reported was an increased breathing rate during exposure. Focal interstitial pneumonitis was observed to about the same extent in control as in exposed rats. Increased urinary fluoride showed that the administered HFC-134 was absorbed, and metabolized to a small extent.

In a 28-day study (Riley et al., 1979) 16 rats/sex/group were exposed to target concentrations of 1000, 10,000, or 50,000 ppm (4170, 41,700, and 208,600 mg/cu.m) HFC-134a for 6 hours/day, 5 days/week (duration-adjusted concentrations = 745, 7450, or 37,250 mg/cu.m, respectively). No compound- related effects were observed on clinical (including ophthalmic) signs, except for lethargy on day 17 in males exposed to 50,000 ppm; food consumption; body weight gain; hematology; or urine composition. High-concentration (50,000- ppm) males showed mild interstitial pneumonitis (3/16 rats); slightly statistically significant increases in alanine transaminase (12%); and absolute kidney and liver weights, 11 and 21%, respectively. Mid-concentration (10,000 ppm) males showed a statistically significant (8%) increase in absolute liver weight. Relative liver weights were increased marginally in a concentration-related manner. Absolute, but not relative testicular weights of high-dose rats were decreased by 2%. None of these organ weight changes were associated with histopathology. It should be noted that none of these changes were observed in the 90-day or chronic studies.

In rats, short-term (single or repeated) exposures to relatively high concentrations of HFC-134a result in reversible CNS depression. The 4-hour lethal concentration in the rat is about 567,000 ppm (Silber, 1979b). The estimated LC50 concentration for the 30-minute exposure was reported as 750,000 ppm in rats (Silber, 1979b). High exposures cause signs of pronounced CNS depression, such as lethargy, unresponsiveness, incoordination, respiratory distress, and depression, and convulsions.

At all except very high doses, CNS depression is fully reversible, with no clinical sequelae being noted after repeated exposures and continued observation. Thus, rats exposed for 6 hours to 100,000 ppm HFC-134a showed decreased responsiveness to sound; at 300,000 ppm, they did not respond to sound and showed severe tremors and incoordination, but completely recovered by 1 hour postexposure (Lu, l981). No CNS effects were observed in several other studies (Riley et al., 1979; Hext, 1989; Hext and Mould, 1991) in which rats were exposed to 50,000 ppm HFC-134a. The cardiac sensitization potential of HFC-134a was studied in dogs by Hardy et al. (1991). The 50% effective concentration for epinephrine-induced cardiac sensitization for a 10-minute exposure was between 160,000 and 320,000 ppm, and positive responses were observed at 80,000 ppm.

In a developmental study, pregnant rats (11 controls and 6/dose group) were exposed for 6 hours/day on gestation days 6-15 to 30,000, 100,000, or 300,000 ppm HFC-134a (125,200, 417,200, or 1,252,000 mg/cu.m, respectively) (Lu, 1981). Rats exposed to 100,000 ppm HFC-134a showed decreased response to sound based on observation of the animals response to tapping on the side of the chamber. Rats exposed to 300,000 ppm HFC-134a showed no response to sound, severe tremors, and incoordination that were completely reversed 1 hour postexposure. Compared with the control, 30,000-, and 100,000-ppm rats, the 300,000-ppm animals ate less and gained significantly less body weight. These differences were not present by gestation day 21 (6 days postexposure). Statistically significant fetal effects of exposure at 300,000 ppm included reduced mean fetal weights (2.4 vs. 3.7 g in controls), increased average percent malformed fetuses/litter (5.4 vs. 1.5; not statistically significant), and average percent fetuses with variations/litter (93.4 vs. 44.8). This study establishes maternal and fetal NOAELs of 100,000 ppm [NOAEL(HEC) = 417,200 mg/cu.m] and LOAELs of 300,000 ppm [LOAEL(HEC) = 1,252,000 mg/cu.m].

In a developmental study (Hodge et al., 1980), rats (29-30/group), exposed for 6 hours/day on gestation days 6-15 to target concentrations of 1000, 10,000, or 50,000 ppm HFC-134a (4170, 41,700, or 208,600 mg/cu.m, respectively) showed no significant clinical signs of toxicity, and reproductive parameters were not impaired, including numbers of implantations, resorptions, corpora lutea, postimplantation loss, and live fetuses. Mean fetal weight of the 50,000-ppm rats was slightly but significantly reduced ($p<0.01$), although litter weight and gravid uterine weight were not affected. Skeletal development of these fetuses was retarded slightly, as indicated by

increased incidence of unossified or partially ossified bones. The maternal NOAEL in this study is 50,000 ppm [NOAEL(HEC) = 208,600 mg/cu.m], and the fetal NOAEL and LOAEL are 10,000 ppm [NOAEL(HEC) = 41,700 mg/cu.m] and 50,000 ppm [LOAEL(HEC) = 208,600 mg/cu.m], respectively.

In a developmental study reported by Wickramaratne (1989a), New Zealand rabbits (28/group) were exposed for 6 hours/day on gestation days 7-19 to target levels of 0, 2500, 10,000, or 40,000 ppm HFC-134a (0, 10,400, 41,700, 166,900 mg/cu.m). All dams survived, and there were no compound-related clinical observations. Dams exposed to 10,000 or 40,000 ppm HFC-134a gained significantly less weight than controls between 7 and 19 days (during the exposure), but body weights at gestation day 30 were not different from controls (97% of control weight in both groups). The weight gains during days 7-19 of gestation were 260, 222, 190, and 162 g in the control and 2500-, 10,000-, and 50,000-ppm groups, respectively. Historical control data were available on weight gain in rabbits used as controls in nine studies done at the same laboratory during a 2.5-year period (seven studies before and two studies after the HCFC-134a study). The mean weight gains averaged 224 g with a standard deviation of 42 g, and there was no trend over time. Because the weight changes in the control and 10,000-ppm groups in this study were within one standard deviation of the historical mean and were within the range of historical control values, it is not clear whether the effect in the 10,000- ppm group is treatment related; therefore, the 10,000-ppm exposure is considered a NOAEL. There were no exposure-related effects on fertility rate, the number and survival of implants or fetuses, or external fetal defects. This finding does not corroborate the reported effect on preimplantation loss reported in the preliminary study (Wickramaratne, 1989b). Litter and fetal weight were not affected by exposure. Exposure to 10,000 or 40,000 ppm caused statistically significant increased incidences of nonossified seventh lumbar transverse processes. Increased incidence of partially ossified pelvic pubes also was seen in the groups exposed to 10,000 or 40,000 ppm, but was statistically significant only in the 10,000-ppm group. These minor skeletal defects were not significantly different in statistical comparison of the litter incidence. None of the approximately 75 other skeletal variations reported were significantly different, and the total incidence of minor variations was not affected by exposure. The fetal defects noted above are not considered to be biologically significant. This study defines a NOAEL of 40,000 ppm for fetal and developmental effects. For the maternal weight effect, a LOAEL of 40,000 ppm [LOAEL(HEC) = 166,900 mg/cu.m] and a NOAEL of 10,000 ppm [NOAEL(HEC) = 41,700 mg/cu.m] are identified. Benchmark concentration analysis was not performed for the maternal weight effect because the LOAEL is much higher than the LOAEL in the principal study and because of the uncertainty in the reliability of the reported weights in the control and the two lower concentration groups.

In a preliminary rabbit developmental study (Wickramaratne, 1989b), 10 animals/group, were exposed to 5000, 20,000, and 50,000 ppm (21,000, 84,000, and 210,000 mg/cu.m, respectively) HFC-134a for 6 hours/day on days 7-19 of gestation. In the group exposed to 50,000 ppm HFC-

134a, one animal died and one aborted. These dams showed statistically significant increased preimplantation loss (26% vs. 6% in unexposed controls) with a resulting reduced number of fetuses, reduced litter and gravid uterine weight, and increased fetal weight. These effects were not corroborated in the larger study (Wickramaratne, 1989a), which used larger groups of animals. Compared with controls, the number of live fetuses from 50,000-ppm dams (45 vs. 58 in controls) and litter weight (244.0 g vs. 369.3 g) were decreased; fetal weight (43.4 g vs. 38.2 g) was increased. In the group exposed to 50,000 ppm, there was a slight loss of body weight during days 7-10, compared with weight gains of 40-50 g in the other groups. Weight gains during gestation were not different in any exposed groups. This transient reduction in weight gain also was seen in the principal study in all exposed groups and in the controls, and the reduced weight gain in the group exposed to 50,000 ppm persisted throughout gestation (Wickramaratne, 1989a). No external abnormalities were seen.

The pharmacokinetics and metabolism of HFC-134a were studied by Ellis et al. (1991) in Wistar rats that inhaled 10,000 ppm of radiolabeled compound for 1 hour. Urine, feces, and exhaled carbon dioxide were collected and analyzed for up to 5 days (urine and feces) or 48 hours (carbon dioxide). Very little of the compound was absorbed; total radioactivity recovered in the expired air, urine, and feces accounted for approximately 1% of the total inhaled dose. Of this 1%, approximately two-thirds was recovered as unchanged HFC- 134a in the exhaled breath. The compound is only minimally metabolized (0.37% of the inhaled radioactivity was recovered in urine and feces as metabolites). Carbon dioxide was the major metabolite, and trifluoroacetic acid was the only metabolite detected in urine. There was no evidence of tissue accumulation 5 days after exposure.

I.B.5. Confidence in the Inhalation RfC

Study — High
Database — Medium
RfC — Medium

Confidence in the principal study was rated high because it was well conducted, was a chronic-duration study, and used a large number of animals. The database is given a medium level of confidence because there are no reproductive toxicity studies. The developmental toxicity following inhalation exposure has been studied adequately. A medium confidence in the RfC follows.

I.B.6. EPA Documentation and Review of the Inhalation RfC

Source Document — This assessment is not presented in any existing U.S. EPA document.

This assessment was peer reviewed by external scientists. This review was completed on 05/22/1995. Their comments have been carefully evaluated and considered in the revision and finalization of this IRIS summary. A record of these comments is included in the IRIS documentation files.

Other EPA Documentation — None

Agency Work Group Review — 09/23/1992, 09/24/1995, 05/11/1995

Verification Date — 05/11/1995

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfC for 1,1,1,2-Tetrafluoroethane conducted in August 2003 identified one or more significant new studies. IRIS users may request the references for those studies from the IRIS Hotline at hotline.iris@epa.gov or 202-566-1676.

I.B.7. EPA Contacts (Inhalation RfC)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — 1,1,1,2-Tetrafluoroethane
CASRN — 811-97-2
Primary Synonym — HFC-134a

This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — 1,1,1,2-Tetrafluoroethane
CASRN — 811-97-2
Primary Synonym — HFC-134a

VI.A. Oral RfD References

None

VI.B. Inhalation RfC References

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Wickramaratne, G.A. 1989b. HFC 134a: Embryotoxicity inhalation study in the rabbit. ICI Central Toxicology Laboratory, Alderley Park, Macclesfield, Cheshire, UK. Report No. CTL/P/2380.

VI.C. Carcinogenicity Assessment References

None

VII. Revision History

Substance Name — 1,1,1,2-Tetrafluoroethane
CASRN — 811-97-2
Primary Synonym — HFC-134a

Date	Section	Description
08/01/1995	I.B.	Inhalation RfC summary on-line
10/28/2003	I.B.6	Screening-Level Literature Review Findings message has been added.

VIII. Synonyms

Substance Name — 1,1,1,2-Tetrafluoroethane
CASRN — 811-97-2
Primary Synonym — HFC-134a
Last Revised — 10/01/1992

- 811-97-2
- Ethane, 1,1,1,2-tetrafluoro-
- Norflurane
- Norflurano [INN-Spanish]
- Norfluranum [INN-Latin]
- R 134a
- 1,1,1,2-Tetrafluoroethane