Guides for Short-Term Exposures of the Public to Air Pollutants

III. Guide for Gaseous Hydrogen Fluoride

by

The Committee on Toxicology

of the

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NOTICE

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The members of the committee were selected for their individual scholarly competence and judgment with due consideration for the balance and breadth of disciplines. Responsibility for all aspects of this report rests with the committee, to whom we express our sincere appreciation.

Although the reports of our study committees are not submitted for approval to the Academy membership nor to the Council, each report is reviewed by a second group of scientists according to procedures established and monitored by the Academy's Report Review Committee. Such reviews are intended to determine, inter alia, whether the major questions and relevant points of view have been addressed and whether the reported findings, conclusions and recommendations arose from the available data and information. Distribution of the report is permitted only after satisfactory completion of this review process.

INTRODUCTION

This is one of a series of documents prepared by the Committee on Toxicology of the National Academy of Sciences-National Research Council, with the support of the Advisory Center on Toxicology, at the request of the Air Pollution Control Office of the Environmental Protection Agency. A subcommittee was appointed to prepare the document which was then endorsed by the Committee.

In preparing this document the subcommittee was guided by the principles set forth in the first document of this series: "Basis for Establishing Short-Term Inhalation Exposure Limits of the Public to Atmospheric Pollutants," (1) and the National Academy of Sciences-National Research Council report, "Fluorides," (2) now in preparation by the Committee on Biologic Effects of Atmospheric Pollutants of the Division of Medical Sciences. The latter document treats the effects of HF on vegetation and animals, and methods for the analysis for airborne concentrations. Therefore, this report gives only minimal attention to the effects of HF on vegetation and animals and no attention to analytical procedures.

Most of the authoritative work on hydrogen fluoride gas dates from the mid-1940's, although there are a few significant contributions from earlier periods. In most of the documents published prior to the late 1960's, mathematical units expressing concentrations vary. For ease of comparison, all units expressing atmospheric concentration have been converted to mg/m^3, considering HF as a monomolecular compound where 1 ppm = 0.8184 mg/m^3. In those few instances where parts by volume are more appropriate, either ppm or ppb has been used.

Some atmospheric standards for HF already exist. For instance, occupational levels recommended by the American Conference of Governmental Industrial Hygienists set the threshold limit value (TLV) for an eight-hour work day at 2 mg/m^3. By application of the ACGIH Permissible Excursion (Time-weighted) rule, a maximum atmospheric concentration for HF of 4.0 mg/m^3 is permitted, provided it is compensated by an equivalent excursion below the limit during the workday. The state of Pennsylvania has adopted an atmospheric limit for HF of 2 mg/m^3 for 15 minutes (3) and the National Academy of Sciences-National Research Council Committee on Toxicology recommended emergency exposure limits for HF of 16 mg/m^3 for ten minutes, 8 mg/m^3 for 30 minutes, and approximately 7 mg/m^3 for 60 minutes applicable only to military and space operations (4).

Experimental data and histories of exposures are reviewed in this report and recommendations for short-term exposure limits of the public are made.

Physical-Chemical Properties

At atmospheric pressure, below 19° C, hydrogen fluoride is a corrosive, fuming, nearly colorless liquid. Above 19° C it is gaseous.

HF has a monomolecular weight of 20.01, but at 1 atmosphere pressure and at a temperature below 100° C it exists as an associated molecule up to H_6F_6, with an average molecular weight of 50 to 55. Some authors (5) have considered the possibility that toxicity may be related to molecular species, but this concept has not been thoroughly explored.

Hydrogen fluoride gas has a density of 0.921 g/l at 0° C and 1 atmosphere of pressure and is very soluble in water. When anhydrous liquid HF is vaporized into the atmosphere it is an almost colorless gas cloud that forms a fog upon combination with moisture in air. This fog is an aerosol of hydrofluoric acid, which is corrosive to almost all inorganic and organic materials.

Sources of HF

Active volcanoes are the only known natural source of gaseous HF. Gaseous effluents from fumaroles and volcanoes contribute background levels of HF throughout the world. This gas has been detected at locations far removed from man-made sources (6).

Many industrial processes contribute gaseous HF to the atmosphere. The gas is an effluent of processes such as aluminum reduction, phosphate fertilizer manufacturing, petroleum refining, manufacture of fluorocarbon compounds; the making of brick, pottery, glass, and ceramics; ferro-enamel production, metal fluxing agents used in foundries and metal-fabricating plants, welding processes, and the burning of coal. Coal may contain 40 to 295 ppm of fluoride depending on its source, some of which is released as HF on burning.

In recent years there has been a change in aluminum-reduction processes, using a synthetic cryolite manufactured using HF, rather than naturally occurring cryolite. This process, as well as the production of HF itself, also contributes to atmospheric levels of the gas.

Most of the modern industrial plants whose operations can release HF to the atmosphere have scrubbers that are reported to be about 95%

efficient, whereas less modern plants are estimated to have control efficiencies as low as 80%.

Within the past decade another potential source of atmospheric HF has appeared: this is liquid-fueled rockets employing propellant oxidizers consisting of mixtures of liquid fluorine and liquid oxygen. In the event of accidental release of these liquid oxidizers to the atmosphere, which would almost certainly be attended by fire, the resultant products would include HF. HF also is a product of normal combustion of the fuel in these rocket engines.

Increased use of HF for a variety of applications similarly increases the need to transport the material from place of manufacture to place of use. Temporary storage facilities at both locations and transfer from storage containers to other containers are operational requirements. In each instance there is danger of accidental spills that could result in exposure of occupational personnel and the public.

Clinical and Pathological effects of HF

The primary effect of acute exposure to gaseous HF in concentrations above a few mg/m^3 is irritation of the skin, eyes, and respiratory passages. In addition, localized tissue damage may occur as a result of the corrosive nature of this compound at concentrations above the recommended limits.

There are several published accounts of the clinical effects in man of acute poisoning with gaseous HF. Local irritation of the mucous membranes of the eyes, nose, throat,and bronchi is reported by Sollmann (7) and Williams (8). In addition, there may be difficulty in breathing. An individual exposed to more than 10 mg/m^3 will almost immediately experience a biting or burning sensation in the nose, followed by a nasal discharge and, occasionally, a nosebleed; burning of the eyes and lacrimation also occur. Exposure to higher concentrations may lead to pulmonary edema and respiratory distress with an onset that may be delayed 12 to 24 hours. This may be accompanied by gastroenteritis, with nausea, vomiting, abdominal burning, diarrhea, and collapse. Exposure to lethal concentrations produces muscular weakness and tremors, clonic convulsions, a drop in blood pressure, and moderate cyanosis; and death may result from sudden respiratory or cardiac arrest.

There are but a few reports of pathological findings available from fatal HF poisonings. According to one report (9) the heart was dilated, the bronchial tree was acutely inflamed with a partially ulcerated mucosa, and the lungs were edematous,congested, and severely hemorrhagic.

Additional pathological studies have been done on experimental animals exposed to either lethal or nonlethal concentrations of HF (10, 11, 12). Pulmonary changes, mainly hemorrhage, edema, and congestion, are consistently seen in acute HF poisoning. The severity of the change is more or less proportional to the duration and concentration of exposure to HF. Lesions can be found in the kidney, liver, and nasal passages. Also, bone marrow obtained seven days after exposure shows an increased cellularity, but with a disproportionate increase in myeloid cells.

Toxicity Studies

Several groups of workers have studied the effects of HF gas on experimental animals. Machle and co-workers (10) conducted time-concentration studies on rabbits and guinea pigs at concentrations ranging from 24 to 8000 mg/m^3 for periods of five minutes to 41 hours. They found that exposure for five minutes or longer to concentrations greater than 1500 mg/m^3 may be lethal to rabbits and guinea pigs. Exposure to 1000 mg/m^3 for 30 minutes caused changes in the lungs, but no deaths, in either species of animals. Concentrations below 100 mg/m^3 for five hours produced lung damage, but no deaths, in rabbits or guinea pigs. The primary tissue damage was manifested by hemorrhage, edema, or congestion. At concentrations of HF above 2000 mg/m^3 areas of the cornea were eroded, there was necrosis of turbinates, necrosis of heart muscles, alveolar and interstitial hemorrhage, edema, emphysema, and, in those that survived several days following exposure, broncho-pneumonia. Also, in the animals exposed at the higher concentrations, the liver showed necrosis of the parenchyma and destruction of cytoplasm; the spleen was edematous and congested and the kidneys showed some degenerative changes in renal tubules and glomeruli.

In another study (5), designed to bracket maximal and minimal effects, five species of animals inhaled HF concentrations of 25 mg/m^3 or 7 mg/m^3 for 166 hours in repeated, daily six-hour exposures for approximately 30 days. Exposure to the higher concentration was lethal to 100% of the rats and mice, but not lethal to guinea pigs, rabbits, and dogs. Among the surviving animals, the rabbits showed a slight loss in weight, the dogs were apparently unaffected, and the guinea pigs began to lose weight after the third week of exposure. Exposure to the low concentration did not interfere with normal weight gains in any of the animals except the rabbits. Three species, the dog, rabbit, and rat, were examined for pathological changes following exposure to both concentrations of HF. At the 25 mg/m^3 level there was moderate hemorrhage and edema of the lungs in all three species, ulceration of the scrotum in dogs and renal cortical degeneration and necrosis in the rat. At the 7 mg/m^3 level, localized hemorrhages were found in the lungs of one dog

out of five examined, and no changes were observed in the rat or rabbit.

Rosenholtz and co-workers (11) studied rats, rabbits, guinea pigs, and dogs to make possible a better estimate of the LC_{50} (the concentration calculated to be lethal to 50% of a group of experimental animals) from single exposures to high concentrations of HF for short periods of time. The LC_{50}'s were as follows:

Species	Exposure Time (minutes)	LC_{50} (mg/m^3)
Rat	5	4060
Rat	15	2200
Guinea Pig	15	3540
Rat	30	1670
Rat	60	1070

Signs of toxicity in the animals included irritation of the conjunctiva, nasal tissues, and respiratory system. The survivors ceased to show these signs about one week after the exposure. Pathological lesions were observed in the kidney and liver, the severity of which was directly related to the dosage received. The external nares and nasal vestibules were black and, at dosages causing considerable mortality, those areas showed zones of mucosal and submucosal necrosis. The skin of animals exposed to high (lethal) concentrations showed superficial subcutaneous and deep dermal zones of acute inflammation. The hair of these animals could be pulled out with ease and the skin ruptured under minimal tension. The rat was found to be the most susceptible species of those tested, which confirmed the findings of Stokinger (5). Additional evaluations were made at nonlethal concentrations of HF that approximated 50%, 25%, 12.5%, and 6% of the rat LC_{50}.

Exposure of animals to nonlethal concentrations produced similar clinical signs that decreased in intensity and duration with decreasing dosage. At 6% of the rat LC_{50} level (250 mg/m^3 in rats for 15 minutes and 80 mg/m^3 in rats for 60 minutes), clinical signs decreased to mild irritation of the eyes and nose. These signs disappeared shortly after withdrawal from the chamber. Few pathological changes were seen at sub-lethal concentrations, although the skin was not examined histopathologically. Two rabbits, one exposed at 1020 mg/m^3 for 15 minutes, and one at 700 mg/m^3 for 15 minutes, showed pulmonary changes ranging from intense intra-alveolar septal congestion to frank hemorrhage with a discrete lobular distribution.

In connection with the animal studies reported by Machle (10), some observations were made on two humans. After one minute at 100 mg/m^3, there was smarting of the skin, conjunctival and respiratory irritation, and recognition of the flat sour taste of HF. Exposure at 50 mg/m^3 was

exceedingly uncomfortable, as shown by irritation of mucous membranes, but there was no smarting of the skin. At 26 mg/m^3 irritation decreased and the ability to taste gas was delayed, but the atmosphere was uncomfortable throughout the three-minute period of exposure.

Instances of Apparent HF Injury

Even though there are numerous industrial processes that can cause HF injury to workers and closely associated nonworkers, there are few records of such injuries. However, there are several well-documented reports of injuries resulting from accidental exposures to ruptured containers of anhydrous HF (9).

Although gaseous HF undoubtedly played some role in these accidents, the major effect is associated with the liquid state. The information does, however, emphasize the extreme hazard of HF and the clinical developments encountered. In three separate events, a total of eight men were splashed with liquid HF. In one event, involving four men, one died of pulmonary edema approximately two hours after the accident. The other three survived, but suffered severe chemical burns. In the other two events, all four men died; two of them in two hours, one in four hours, and one ten hours after the accident. Consciousness remains until death, which is usually sudden, from respiratory distress and cardiac arrest. Characteristically, the respiratory tree is inflamed and the lungs moderately to severely congested.

There are other reported cases of severe poisonings and deaths among workers in superphosphate factories, fertilizer factories, among welders, garage workers, waterworks employees, and enamel-factory workers (11). In most of these cases the clinical picture in part resembles HF poisoning, but other fluoride-containing particulates and gases such as SiF_4, phosgene (from the high temperature decomposition of halogenated hydrocarbons) and a variety of fluoride dusts, also were present.

Species Susceptibility to HF Poisoning

Plants are more susceptible to damage from gaseous HF than are animals, although among plant species there is a wide variation in tolerance. Some plants, gladiolus for example, may be severely damaged by airborne concentrations as low as 3 $\mu g/m^3$ (leading to accumulations of 20 ppm in plant tissues), whereas camellia can tolerate tissue accumulation to as much as 1500 ppm. There is a complex and poorly understood relationship between airborne concentrations and levels that accumulate in plants. Grasses and forage crops vary throughout a wide range in accumulation and tolerance. HF is absorbed systemically through the

stems and leaves of plants. The absorbed fluorides migrate to the margins of the leaves where early evidence of damage appears as yellowing or browning of tissues.

Cattle, sheep, and goats that feed on fluoride-contaminated forage may sicken and die from chronic fluoride poisoning, fluorosis, a not uncommon occurrence in areas where effluents containing fluorides affect grazing lands. Animals with a total dietary intake of fluoride greater than 300 ppm per day are likely to develop fluorosis. A generally accepted guide (14) for fluoride concentration in animal forage is based on an annual average of no more than 40 ppm of fluoride by dry weight.

Inhaled HF, even in heavily contaminated areas, contributes so small a fraction of the total intake by grazing animals that it cannot be considered an important factor in either chronic or acute systemic poisoning.

Atmospheric Concentrations of HF

National surveillance programs conducted since the mid-1960's reveal atmospheric levels of fluoride ranging from 0.5 to 1.89 $\mu g/m^3$ in urban areas and 0.05 to 0.16 $\mu g/m^3$ in nonurban areas (15). Huffstutler (16), reporting on fluoride concentrations within a 7-1/2 mile radius in an area of Florida where a number of fertilizer plants are operated, found a four-hour sample peak of 100 μg fluoride/m^3 one year, and a maximum four-hour sample average of 2.7 μg fluoride/m^3 in another year. Similarly, he reports a 24-hour sample peak of 68 μg fluoride/m^3, and a maximum 24-hour sample average of 4.3 μg fluoride/m^3. These studies have been carried out over a period of seven years and the 1969 data show a marked decrease both in peak levels and in sample period averages as a result of improved scrubbing procedures.

As with other gaseous contaminants, the greatest hazard is downwind from the effluent stacks. Workers in the immediate environment of operations involving HF may not be as readily or as heavily exposed as nonworkers some several hundred yards downwind. Beyond two or three miles downwind, mixing and dilution with air reduces HF below detectable or background levels.

Guides for Short-Term and Emergency Exposures of the Public to HF

The basis for setting limits for short-term exposure of the public and for emergency exposure of the public to air pollutants is detailed in the first document of this series, "Basis for Establishing Guides for Short-Term Exposures of the Public to Air Pollutants" (1)

Experimental data and occupational experience indicate that man is susceptible to irritation and possible injury from gaseous HF. At 10 mg/m^3 the mucosa are irritated; at 26 mg/m^3 for three minutes he is uncomfortable and able to taste the gas; at 50 mg/m3 the severity of the irritation increases; and at 100 mg/m^3 a stinging sensation of the skin is added and other irritations are so severe as to make exposure for more than one minute intolerable. Since the primary irritant action of HF is on the mucosa, particularly the respiratory mucosa, it is important to recognize the more sensitive segments of the population, e.g., the asthmatics and bronchitics, as the limiting factors.

The following recommendations are time-weighted averages, which are considered not to present any health hazard. It should be recognized, however, that excursions above these averages are likely to produce objectionable odors and, possibly, minimal irritation.

Short-Term Public Limits (STPL's)

The limits for short-term exposure of the public to air pollutants are established in view of the possibility of repeated events in the same locality. These events, such as intentional release of HF to the atmosphere, are assumed to be controllable with respect to concentration and duration of release so that the limit is not exceeded. No adverse health effects, however transient, are anticipated at the limits set forth below:

	STPL's
10-30 and 60 min	3 mg/m^3 (4 ppm) (frequency limit 1 hr daily)
5 hr/day, 3-4 days/mo	1 mg/m^3 (1 ppm)

Public Emergency Limits (PEL's)

Public emergency limits represent values necessary to cope with an accidental, unpredictable, or uncontrollable event. The PEL assumes that some temporary discomfort may accrue to the public but that any effect resulting from the exposure is reversible and without residual damage.

	PEL's	
10 min	8 mg/m^3	(10 ppm)
30 min and 60 min	4 mg/m^3	(5 ppm)

Research Needs

The data from industrial experience and laboratory experimentation are insufficient to permit a relaxation in efforts to determine the toxic hazards of atmospheric hydrogen fluoride. It is hoped that this guide will serve as a stimulus for occupational health authorities and researchers to investigate further the effects of HF on the health and welfare of the public and on the environment so that greater confidence can be placed in the recommended limits.

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